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RESEARCH ARTICLE

Evaluation of the effect of smoking on periodontal disease and its response to non-surgical treatment: a randomized controlled trial

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Compliance with ethical standards: The protocol was approved by the Ethical Committee of the King Khalid University. After being informed on the purpose of the study, the patients signed informed consent forms. Research was conducted according to the principles outlined in the Declaration of Helsinki on experimentation involving human subjects.

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ABSTRACT

This prospective clinical study was designed to evaluate clinically the effect of smoking on periodontal disease and its response to non-surgical treatment. Fifty patients fulfilling the inclusion and exclusion criteria were divided into Group 1 with 25 smokers, and Group 2 with 25 non-smokers (control). At baseline all the subjects received periodontal examination. Supragingival and subgingival scaling using ultrasonic tips and root planing using curettes were performed at the initial visit. The following clinical parameters were selected for the evaluation at baseline, 1 month, 2 months and 3 months: Probing Depth (PD), Gingival Recession (GR), Plaque Index (PI), Gingival Index (GI) and Gingival Crevicular Fluid (GCF) volume measurement. The results showed that: 1. smokers had significantly greater PD, GR and PI as compared to controls, 2. smokers had significantly lesser GI scores than controls indicating suppressed inflammation, 3. smokers had lesser GCF scores than non-smokers indicating suppressed inflammation. In conclusion, due to deleterious effects of tobacco on periodontal tissues, it seems important that tobacco cessation therapy should be emphasized to smokers and tobacco chewers requiring such treatment.

Keywords: Periodontal disease; Non-surgical therapy; Smoker; Non-smoker; Response to therapy.

INTRODUCTION

Periodontitis is defined as an inflammatory disease of the supporting tissues of the teeth caused by specific microorganisms, resulting in progressive destruction of the periodontal ligament and alveolar bone with pocket formation, recession, or both. Environmental, acquired, and genetic risk factors modify the expression of periodontal diseases and may therefore affect the onset or progression of periodontitis [1]. Some of the known risk factors are diabetes, tobacco use, pathogenic bacteria, and microbial tooth deposits.

Among the environmental risk factors, tobacco smoking has been found to be associated with an increased prevalence and severity of periodontal disease [2, 3]. There is mounting evidence that cigarette smoking is an important risk factor for destructive forms of periodontal diseases [4]. Studies suggest that cigarette smoking may be causally associated with periodontitis and also may contribute to a less favorable response to periodontal treatment.

In India, prevalence of tobacco consumption is very high; especially bidis in rural areas and cigarette smoking in urban population. Cigarette smoke contains nicotine, cotinine, acrolein, acetaldehyde which have detrimental effects on periodontium. Cotinine is the principle metabolite of nicotine and as such provides a valuable quantitative measure of smoking status. Patient's cotinine

levels have recently been shown to correlate directly with outcomes of progressive periodontal breakdown [5].

Non-surgical mechanical periodontal therapy, including oral hygiene instruction, scaling and root planing, is an effective treatment modality for periodontal disease [6, 7].

This prospective clinical study was designed to evaluate clinically the effect of smoking on periodontal disease and its response to non-surgical treatment.

MATERIAL AND METHODS

Sixty three patients with chronic periodontitis who visited the Department of Periodontology and Implantology of institute were screened for the study. Thirteen patients could not be enrolled in the study as they decided to quit the tobacco habit. Fifty patients fulfilling the inclusion and exclusion criteria were grouped as follows (Fig. 1): Group 1 with 25 smokers (mean age: 35.32 years), and Group 2 with 25 non-smokers - controls (mean age: 37.40 years). All subjects in groups 1 and 2 were males.

This study was approved by the Ethical Committee of the Institution. All participants gave their written informed consent to the study protocol, which had been approved by the Ethical Committee of the College under MUHS University. Inclusion criteria for patients selection was chronic periodontitis, history of tobacco consumption for a minimum duration of 3 years (except in controls), patients who did not discontinue tobacco consumption in spite being counselled and advised to stop the habit were only include, at least four teeth with pocket depth 3-5 mm and/or attachment loss of 1-3 mm, systemically healthy.

Patients were excluded if they; have aggressive periodontitis, require surgical periodontal therapy, periodontal therapy or antibiotics in previous 3 months, medication with drugs affecting periodontal tissues, pregnant or lactating mothers.

At baseline all the subjects received periodontal examination. As per the protocol, patients were counselled and motivated to quit tobacco consumption. Those patients who did not quit the tobacco consumption habit were included in the study and clinical parameters were recorded.

Supragingival and subgingival scaling using ultrasonic tips and root planing using curettes was performed at the initial visit. Routine oral hygiene instructions were given and were reinforced at every visit. No chemical plaque control agents were advised to the patients. Subsequently periodontal clinical parameters were evaluated at baseline, 1 month, 2 months and 3 months.

Probing depth (PD) was examined with UNC-15 probe. Probing was carried out on mesial, distal, midfacial and midoral aspects of each tooth. The deepest pocket was recorded as the “probing depth” for that tooth. Gingival recession (GR) was measured with UNC-15 probe as the distance from cemento-enamel junction (CEJ) to the gingival margin. Indices: full mouth Plaque Index (PI) and Gingival Index (GI) [5] were recorded. Gingival Crevicular Fluid (GCF) measurement was in 4 sites with deepest pocket depths were selected for sample collection. Each GCF sample was collected with sterile absorbent paper points. The paper points were consecutively inserted into the pocket until mild resistance was felt. They were then transferred to the chairside located digital pocket scale (MH-Series, ACE^{TM}) for volume determination.

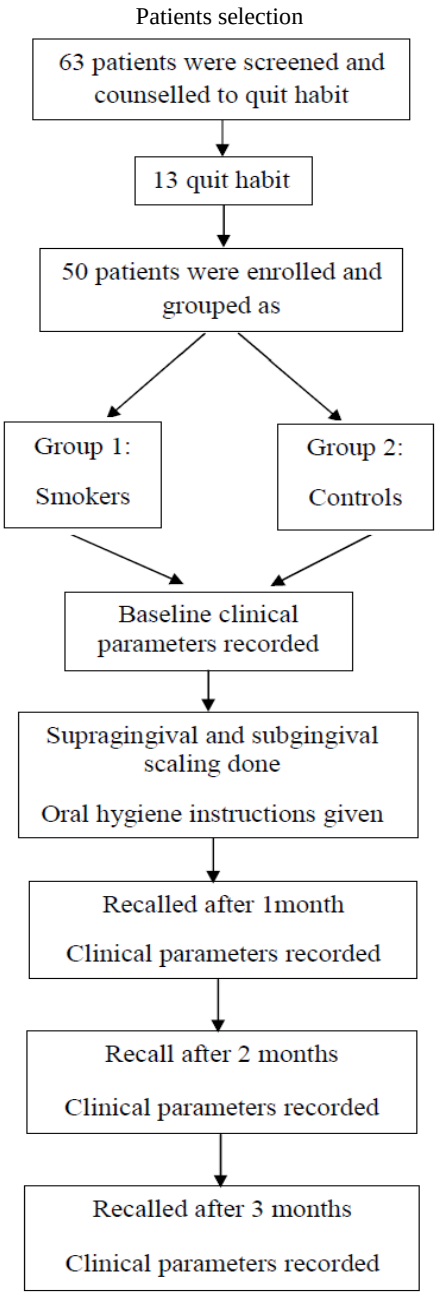

Figure 1. Flow chart of study design.

RESULTS

All the clinical parameters recorded were subjected to the following statistical analysis. For intragroup variation paired "t" test was performed. For comparison between the two groups for intergroup variation two samples, independent "t" test was performed.

The result showed that: smokers had signi-ficantly greater PD (Table 1), GR (Table 2) and significantly higher PI as compared to controls (Table 3). Reduction in PI in 3 months was lesser in smokers compared to controls, indicating poorer response to therapy (Table 3). Smoker had lesser GI scores than controls indicating suppressed inflam-mation. Reduction in GI in 3 months was also less indicating poorer response to therapy (Table 4). Smoker had lesser GCF scores than controls indicating suppressed inflammation. Reduction in GCF in 3 months was also less indicating poorer response to therapy (Table 5).

DISCUSSION

The aim of the present study was to evaluate clinically the effect of smoking on periodontal disease and its response to non-surgical treatment using clinical parameters like gingival index (GI), gingival crevicular fluid (GCF) measurement, probing depth (PD), gingival recession (GR) and plaque index (PI).

To analyse the effect of the treatment in all groups over a study period of 3 months at the regular intervals from baseline paired "t" test was applied for all the parameters. The test was applied at 24 degrees of freedom and at 95% confidence interval. Intergroup comparisons were made using independent "t" test and at 48 degrees of freedom and at 95% confidence interval.

The mean GI of smokers (G1) at baseline was 1.15 ± 0.26 and for controls (G2) was 1.78 ± 0.43 showing a mean difference of -0.62 ± 0.17 which was statistically significant. There was significantly less gingival inflammation in smokers, which is in agreement with the earlier studies [8, 9]. Statistically significant differences were observed at 1, 2 and 3 months between both groups with smoker group showing lesser reductions than control group indicating less favourable response to therapy.

The mean PI of smokers (G1) at baseline was 1.71 ± 0.43 and for controls (G2) was 1.37 ± 0.31 showing a mean difference of 0.33 ± 0.12 which was statistically significant. The finding of higher PI in smokers is in agreement with other studies [10-12].

The mean PD of smokers (G1) at baseline was 4.84 ± 0.59 mm and for controls (G2) was 4.38 ± 0.37 mm showing a mean difference of 0.46 ± 0.22 mm which was statistically significant. Similar findings were reported in other studies [13, 14]. The results indicate lesser reductions in mean PD following non-surgical periodontal therapy in smokers compared to controls indicating less favourable response.

The mean GR of smokers (G1) at baseline was 2.36 ± 0.75 mm and for controls (G2) was 1.60 ± 0.84 mm showing a mean difference of 0.76 ± 0.11 mm which was statistically significant. Non-surgical periodontal therapy improved GR values in both smokers and controls.

Table 1. Comparative changes in mean Probing Depth (PD): G1 vs G2.

Time Interval	G1: Smoker	G2: Control	1 vs 2			
	Mean PD ± SD (mm)	Mean PD ± SD (mm)	Mean Difference (mm)	t value	P value	Significance
Baseline	4.84 ± 0.59	4.38 ± 0.37	0.46 ± 0.22	3.268 7.602	0.002	Significant
1 Month	4.53 ± 0.51	3.64 ± 0.27	0.89 ± 0.24	7.602	0.000	Significant
2 Months	4.24 ± 0.52	3.33 ± 0.30	0.91 ± 0.22	7.551	0.000	Significant
3 Months	4.11 ± 0.57	3.00 ± 0.77	1.10 ± 0.20	5.731	0.000	Significant

Table 2. Comparative changes in Gingival Recession (GR): G1 vs G2.

Time Interval	G1: Smoker	G2: Control	1 vs 2			
	Mean GR ± SD (mm)	Mean GR ± SD (mm)	Mean Difference (mm)	t value	P value	Significance
Baseline	2.36 ± 0.75	1.60 ± 0.84	0.76 ± 0.11	3.310	0.001	Significant
1 Month	2.11 ± 0.69	1.24 ± 0.66	0.87 ± 0.03	1.541	0.000	Significant
2 Months	2.15 ± 0.61	1.02 ± 0.64	1.12 ± 0.03	6.330	0.000	Significant
3 Months	2.03 ± 0.57	1.01 ± 0.59	1.02 ± 0.02	6.168	0.000	Significant

Table 3. Comparative changes in Plaque Index (PI): G1 vs G3.

Time Interval	G1: Smoker	G3: Control	1 vs 3			
	Mean PI ± SD	Mean PI ± SD	Mean Difference	t value	P value	Significance
Baseline	1.71 ± 0.43	1.37 ± 0.31	0.33 ± 0.12	3.102 8.364	0.003	Significant
1 Month	1.09 ± 0.25	0.55 ± 0.20	0.54 ± 0.05	8.364	0.000	Significant
2 Months	1.10 ± 0.28	0.69 ± 0.25	0.41 ± 0.03	5.346	0.000	Significant
3 Months	1.22 ± 0.28	0.86 ± 0.27	0.36 ± 0.01	4.558	0.000	Significant

Table 4. Comparative changes in Gingival Index (GI): G1 vs G2.

Time Interval	G1: Smoker	G2: Control	1 vs 2			
	Mean GI ± SD	Mean GI ± SD	Mean Difference	t value	P value	Significance
Baseline	1.15 ± 0.26	1.78 ± 0.43	-0.62 ± 0.17	-6.118 4.199	0.000	Significant
1 Month	0.90 ± 0.25	0.58 ± 0.28	0.32 ± 0.03	4.199	0.020	Significant
2 Months	1.00 ± 0.32	0.79 ± 0.29	0.21 ± 0.03	2.409	0.000	Significant
3 Months	0.98 ± 0.22	0.74 ± 0.19	0.24 ± 0.03	4.081	0.000	Significant

Table 5. Comparative changes in gingival crevicular fluid (GCF): G1 vs G2.

Time Interval	G1: Smoker	G2: Control	1 vs 2			
	Mean GCF ± SD (g)	Mean GCF ± SD (g)	Mean Difference	t value	P value	Significance
Baseline	0.0400 ± 0.006	0.0524 ± 0.008	-0.0124 ± 0.002	5.894	0.000	Significant
1 Month	0.0404 ± 0.003	0.0324 ± 0.005	0.0080 ± 0.002	6.351	0.000	Significant
2 Months	0.0416 ± 0.004	0.0344 ± 0.005	0.0072 ± 0.001	4.796	0.000	Significant
3 Months	0.0408 ± 0.002	0.0336 ± 0.004	0.0072 ± 0.002	6.397	0.000	Significant

In the study, statistically significant differences were seen in GR at 3 months from baseline but not at 1 and 2 months. There was lesser reduction for smokers compared to control group indicating poorer response to therapy in smokers.

The mean GCF of smokers (G1) at baseline was 0.0400 ± 0.006 g and for controls (G2) was 0.0524 ± 0.008 g showing a mean difference of 0.0124 ± 0.002 g which was statistically significant. The lower GCF volume found in smokers in the study compared to non-smokers is in accordance with previous studies [15-17]. Statistically significant differences were observed between both groups with control group showing reductions in GCF while smoker group showing increase in GCF at 1, 2 and 3 months from baseline.

CONCLUSIONS

Tobacco consumption affects the severity of periodontal disease but can mask the expression of gingival inflammation. Tobacco consumption also hampers the response of periodontal tissues to non-surgical treatment and continues to mask the expression of gingival inflammation in addition it significantly lower gingival crevicular fluid flow, as compared to controls. Due to deleterious effects of tobacco on periodontal tissues, it seems important that tobacco cessation therapy should be emphasized to smokers and tobacco chewers requiring such treatment.

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CASE REPORT

An interesting co-occurrence of amelogenesis imperfecta and osteogenesis imperfecta: a case report

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ABSTRACT

Amelogenesis imperfecta is an inherited disorder which the appearance and the structure of enamel is altered. Osteogenesis imperfecta is a genetic condition that type I collagen is affected. A 37 year old osteogenesis imperfecta patient has referred to Dentomaxillofacial Department with esthetic complaints about her teeth. Her dental status was diagnosed as hypoplastic type of amelogenesis imperfecta; according to the appearance of teeth and also dentin tissue along with detection of very thin or absence of enamel in panoramic radiography is exposed. Patient was referred to prosthodontist. A new occlusal dimension was determined for the patient and after adaptation of masticatory system, fixed prosthetics were performed. This case report highlights the rare incidence of amelogenesis imperfecta in osteogenesis imperfecta patient and the treatment for present dental condition.

Keywords: Amelogenesis imperfecta; Dental enamel hypoplasia; Dental prosthesis; Osteogenesis imperfecta.

INTRODUCTION

Amelogenesis imperfecta (AI) is a genetic-based disorder which structure and appearance of enamel affected in various ways. AI affects both deciduous and permanent dentitions [1]. The prevalence is between 1:700 and 1:14000. The reported prevalence for Turkey is 43:10.000 [1, 2]. The genes which may be responsible for AI development are ENAM, AMELX, MMP-20, KLK4, and DLX3 [1, 3]. There are various classifications for AI but based on clinical display the simplest classification of AI has 4 subtypes as hypoplastic, hypocalcified, hypomature, hypomature/hypoplastic with taurodontism respectively [3, 4].

Osteogenesis imperfecta (OI) is a heritable connective tissue disorder which affects type I collagen [3, 5, 6]. Heritable ways are including autosomal dominant and recessive patterns, furthermore sporadic cases may occur [3]. As tendons, ligaments, skin sclera, teeth, middle and inner ear also contain type I collagen, these structures may also be deficient. Type I dentinogenesis imperfecta (DI) is a condition which is associated with OI [5]. There is no exact knowledge about the incidence of OI but the reported occurrence is between 1:5000 and 1:20.000 [5, 6]. There isn't any race or gender predominance [6].

In all probabilities, the highest prevalence for AI and OI, which are seen together, is 86:1000000 for Turkish population. The aim of this case report is to present the

first known case of AI in association with OI and treatment approach to the patient.

CASE PRESENTATION

A 37 year old female patient was referred to Istanbul University, Faculty of Dentistry, Oral and Maxillofacial Radiology Department about esthetic concerns. Patient's concern was the teeth in both deciduous and permanent dentitions were discolored, sensitive and had tendency to wear off. Patient's background revealed that she was diagnosed as OI, keratoglobus and mild hearing loss. Patient's elder brother had the same clinical history, both dentally and skeletally.

Clinical examination showed that she had yellow-brown discoloration in all teeth and blue sclera (Figure 1). Patient's oral hygiene was moderate with no gingival bleeding. The teeth were smaller in size with adequate crown lengths and polidiastema was detected. Posterior teeth had severe attrition, manifesting normal exposed dentine (Figures 2 and 3). Radiological examination showed that enamel was completely absent or thin and 48, 38 numbered teeth were impacted. Also external resorption was observed on the crown of impacted 38 numbered tooth.

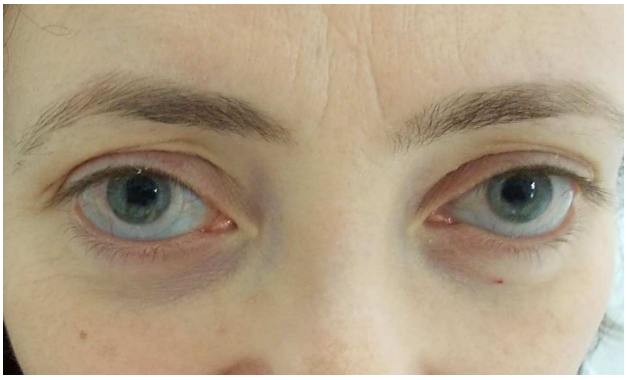

Figure 1. Patient's blue sclera.

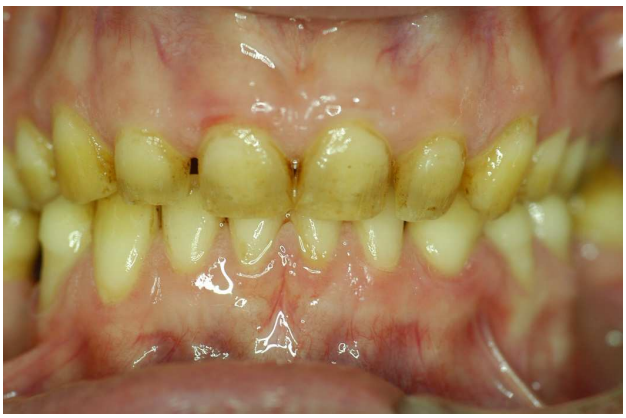

Figure 2. Patient's intraoral picture presenting polidiastema and small discolored anterior teeth.

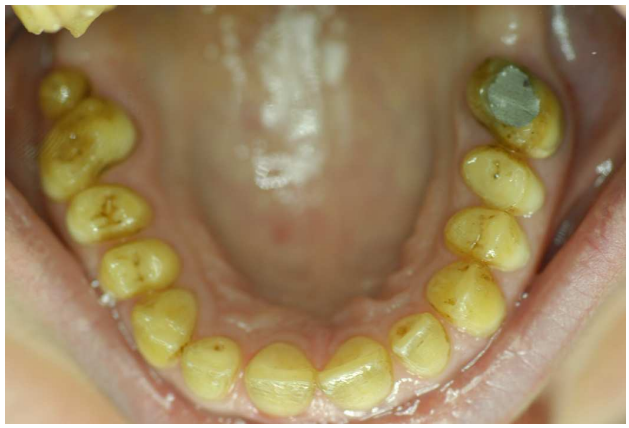

Figure 3. Patient's intraoral picture showing severe attrition on posterior teeth.

The pulp chambers of the teeth were normal except 17, 26, 37, and 47 numbered teeth which had elongated pulp chambers to teeth apices as taurodontism (Figure 4). Depending on the clinical and radiographic findings, her dental condition was diagnosed as a hypoplastic type of AI. The patient was referred to Medical Biology Department for determination of exact genetic typing of AI. However, patient had declined the required genetic tests and the diagnosis was based only on radiological and clinical appearance of teeth.

First of all, the patient was treated with scaling, root planning is done as a step of initial periodontal therapy and instructions of oral hygiene were given. After the calculus and stains were cleared, prosthetic treatment was planned. Fixed metal reinforced porcelain crowns and bridge restorations were recommended for posterior teeth. In order to fulfill the esthetic concerns of patient, zirconia based all-ceramic crown restorations were planned for anterior teeth. Before the procedure, the occlusal vertical height was examined and found insufficient.

Occlusal splint therapy was planned to evaluate patient's response to the increased vertical dimension. The maxillary and mandibular impressions were made with irreversible hydrocolloid, and diagnostic casts were mounted in a semi adjustable articulator. The mandibular cast was used for fabrication of occlusal splint. The splint was adjusted by an articulation paper of 8 μm thick to provide equal intensity stops on all teeth in centric relation and to achieve functional neuromuscular stability (Figures 5A and 5B).

The freeway space which is the difference in vertical dimensions between the positions at rest and in occlusion of mandible was measured. The vertical dimension was increased gradually at predetermined amount of 3 mm with addition of a self-cured acrylic resin to occlusal surface of the splint (Figures 6A-C). The temporo-mandibular joint and masticatory muscles were examined

on the first week and in every 2 weeks for 6 months with careful palpation of all structures in terms of the adaptation to changes in vertical dimension.

After the adaptation of masticatory muscles to the new vertical dimension, the splint was divided bucco-lingually into two parts to preserve the same vertical height due to teeth preparations. The sectioned splint was placed unilaterally on one side of the mandibular arch to preserve the determined vertical dimension, the maxillary and mandibular teeth on other side of the arch were prepared in a standardized manner for all-ceramic and metal-ceramic fixed partial denture restorations (Figure 7). An occlusal record was made using a pattern resin material on the prepared side of the arch while the sectioned splint was on the unprepared side of the mandibular arch (Figure 8). Then the teeth preparations on other side of the arch were completed while the pattern resin occlusal record material was on the prepared teeth (Figure 9). Impressions were made with a polyvinyl siloxane impression material, and the records were transferred on the semi-adjustable articulator. The all-ceramic restorations were fabricated using the zirconium-oxide–based material with CAD/CAM system for the anterior teeth, and were luted with resin cement. The metal-ceramic restorations were fabricated from a chromium-cobalt alloy, using the traditional lost-wax technique for the posterior teeth and were luted with zinc polycarboxylate. After oral hygiene instructions, the patient was recalled after first week, first month, and third month for examinations. No aesthetic or functional problems were seen (Figure 10).

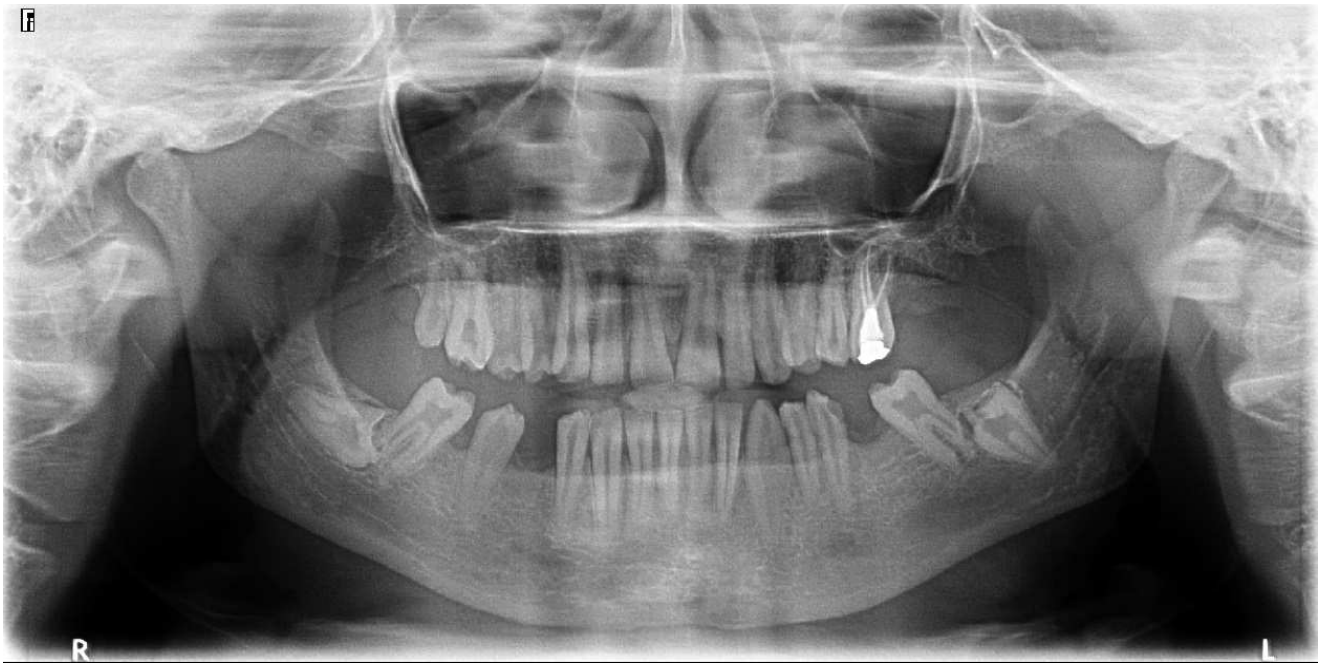

Figure 4. Patient's panoramic radiography.

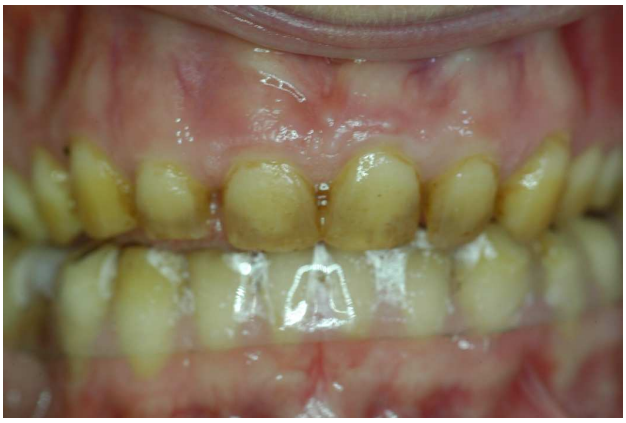

Figure 5A. Patient is wearing occlusal splint on mandibular teeth.

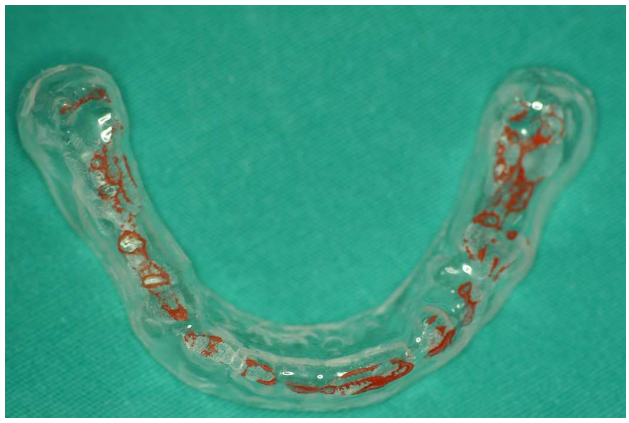

Figure 5B. Adjustment of the splint by an articulation paper to provide equal intensity stops.

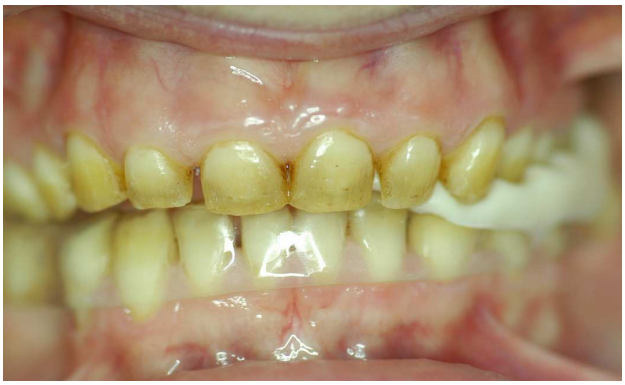

Figure 6A. The addition of self-cured acrylic resin to left occlusal surface of the splint.

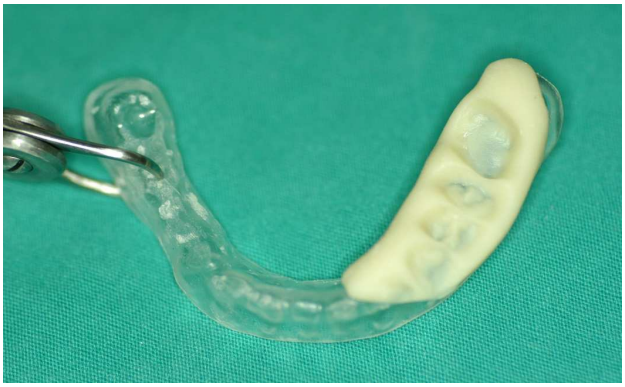

Figure 6B. The comparision of thicknesses of the prepared splint on left and right sides.

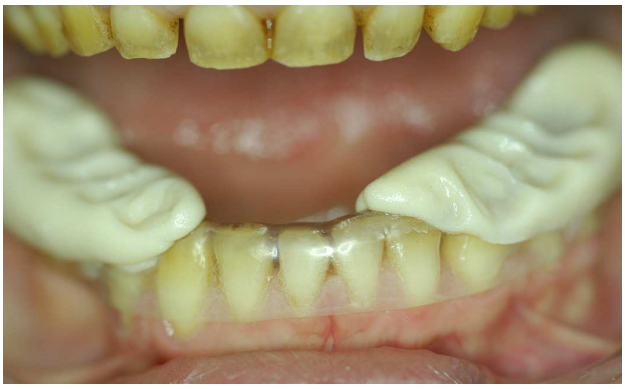

Figure 6C. Application of the splint in patient's mouth.

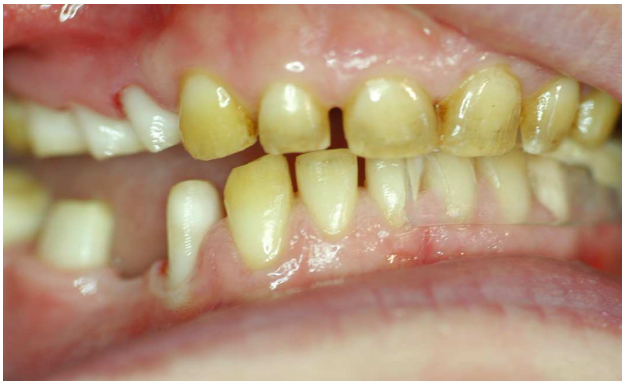

Figure 7. Preparation of teeth while the sectioned splint was placed on the other side.

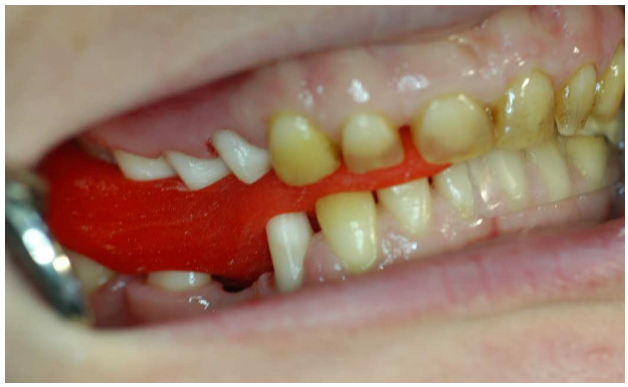

Figure 8. An occlusal record was made using a pattern resin material on the prepared side.

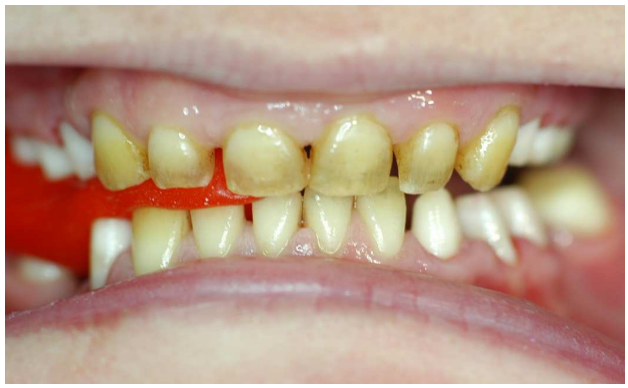

Figure 9. The completion of teeth preparations while the pattern resin occlusal record material was placed on the teeth which were initially prepared.

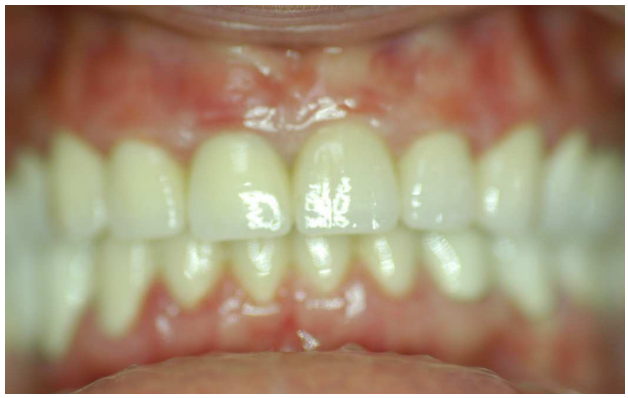

Figure 10. Patient's final dental status.

DISCUSSION

Abnormal collagen type I is not only present at bones of AI patients, also in other structures like skin, sclera, tendons and teeth etc. This situation leads to defects at these structures. DI type I is one of the abnormalities which may be seen with OI. The rate of OI with DI type I is %28 to %73 and this association is most frequent at OI type III and IV [7]. Besides that there is no report of OI and AI seen in the same patient.

OI patients are likely to have bleeding problems and increased risk of bone fractures because of the deficient collagen synthesis. OI patients might be treated with bisphosphonates and bisphosphonate-related osteonecrosis of jaws (BRONJ); risk for surgical, periodontal or endodontic therapies should be taken into consideration. OI patients may also have valvular defects [5]. Our patient hasn't undergone bisphosphonate therapy nor had valvular defect.

AI has clinically 4 different types as hypoplastic, hypomature, hypocalcified and hypoplastic/hypomature with taurodontism. Enamel mineralization and radiopacity is normal in hypoplastic type, but thickness of enamel is reduced. Pits and grooves may be observed [1, 8]. Enamel matrix synthesis, as well as the thickness of enamel, is normal in hypomature type but crystal structures are immature. Clinically tooth crown has mottled appearance; hardness and radiopacity of enamel is reduced. Radiopacity of enamel is close to dentin [1, 3, 8]. In hypocalcified type the thickness of enamel is normal but enamel calcification is defected, leading easily worn off enamel once the tooth erupted. Radiopacity is less than the dentin related with the hypomineralization [1, 8, 9]. Hypoplastic/hypomature with taurodontism type has the characteristics of both hypoplastic and hypomature types, along with apically enlarged pulp chambers [1]. Dental anomalies that can be seen along with AI are; crown and root resorption, taurodontism, impacted teeth, dens in dente, root malformation, pulp stones, agenesis and skeletally anterior open bite [4, 8]. In our case crown resorption, taurodontism, impacted teeth were observed.

Due to attrition/chipping of enamel AI patients have exposed dentine, increased sensitivity and tendency for caries. Patients also have reduced vertical occlusal dimension and difficulties during mastication [10]. Most of the patients with AI have edematous and hyperemic gingival [8]. Our patient had increased sensitivity and reduced vertical occlusal dimension that is required splint therapy to increase the occlusal dimension. Patient's oral hygiene was moderate. AI has autosomal dominant, autosomal recessive and X-linked genetic traits but sporadic cases may also occur [2, 4].

Treatment of AI is important for esthetics, function and psychological reasons. Due to the AI affects both deciduous and permanent dentitions; treatment outcome should meet the psychological, functional and aesthetic expectations during adolescence and adulthood [10]. The cases of AI are treated with various techniques, such as; overdentures, fixed full mouth crown restorations, bridges, onlays/inlays, implant therapy, adhesive restorative techniques according to patient's age, socio-economic status and severity of disorder [4, 10]. When patient's socio-economic status considered, implant therapy was ruled out and fixed metal reinforced porcelain crown restorations for posterior regions and for esthetics. On the other hand ceramic crown restorations in the anterior region were the treatment of choice.

CONCLUSION

Co-occurrence of AI and OI is a rare situation. Malocclusion is observed in OI patients and due to accordance of AI in these circumstances dental treatment should be planned with caution. Local and systemic conditions should be taken into consideration for dental treatment.

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