

Amelogenesis Imperfecta: Case Study

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Clinical Relevance

This article describes the diagnosis and treatment of a patient with amelogenesis imperfecta from mixed dentition to permanent dentition. This study aims to provide a treatment plan that can be used as a source for other amelogenesis imperfecta cases.

SUMMARY

Amelogenesis imperfecta (AI) refers to a group of rare genetic disorders that involve tooth development and that are passed down through families as a dominant trait. This condition is characterized by abnormal enamel formation caused by gene mutations that alter the quality and/or quantity of enamel. This dental problem can impact both primary and permanent dentition, varies among affected individuals, and results in esthetic and functional problems. This condition caused the patient in the current case report to have a lack of confidence when speaking.

The treatment for amelogenesis imperfecta depends on the severity of the problem and age of the patient. It is crucial to plan a proper remedy, which requires collaboration among dental specialties to execute comprehensive

dental treatment in order to provide a long-term solution with adequate esthetics.

The current clinical study presents a patient affected by AI that was diagnosed when the patient was a child. The interdisciplinary treatment continued throughout his childhood and into adult life. The initial treatment consisted of resin composite veneers and stainless-steel crowns to restore the defective tooth structure. The malocclusion of the patient was corrected using a fixed orthodontic appliance that was placed when he had an entire permanent dentition. The treatment plan was eventually intended to include all ceramic crowns and veneers.

INTRODUCTION

Amelogenesis imperfecta (AI) is defined as a group of hereditary developmental defects of the dental enamel affecting both primary and permanent dentition.¹ AI may be inherited in an X-linked manner or by autosomal dominant, autosomal recessive, or sporadic inheritance patterns.^{2,3} The most common type of AI occurs as a result of the autosomal dominant form of transmittance.⁴ This anomaly exists independent of any related systemic diseases.⁴⁻⁶

There are various classification systems proposed for the different types of AI. Generally, AI is classified into three types—hypoplastic, hypominer-

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alization, and hypomaturation^{4,7-10}—on the basis of clinical and radiographic findings. The hypoplastic type is defined as a deficiency in the deposition of organic matrix. Therefore, the enamel is reduced in quantity but is mineralized properly, has normal radiopacity, and can be observed as thin, malformed, lacking contact but appearing hard and shiny. The hypomineralization type of AI presents with a deficiency in the mineralization of the formed matrix. The enamel has normal thickness but has a low degree of mineralization. Thus, there is no contrast in the radiopacity between the enamel and the dentin. The enamel is soft, friable, and easy to remove from the dentin. In hypomaturation, there are abnormalities in the final stage of the mineralization process because the enamel crystals remain immature and there is no contrast of radiopacity between the enamel and dentin. The hypomaturational type differs from the hypocalcification type in that the enamel is harder and presents with a mottled opaque white to yellow-brown or red-brown color. However, the enamel is softer than normal and tends to chip from the underlying dentin.

The estimated prevalence of AI is reported to vary between approximately 1:16,000 and 1:700, depending on the population studied and the diagnostic criteria used.¹¹ Extrinsic disorders, chronological disorders, and localized disorders of tooth formation and metabolic disturbance affecting enamel formation should be considered in the differential diagnosis for AI.³

The primary clinical problems of AI include poor dental esthetics, dental sensitivity, and loss of vertical dimension.¹² AI also has been associated with impacted teeth and anomalies in tooth eruption, congenitally missing teeth, pulp calcification, root and coronal resorption, hypercementosis, root malformation, and taurodontism.¹³⁻¹⁷ Moreover, the AI patient has an increased probability for having dental caries and plaque accumulation. The common malocclusion frequently associated with AI is anterior open bite. It has been suggested that craniofacial skeletal and enamel formation may share a common ectomesenchymal origin. The incidence of anterior open bite in AI patients varies from 24% to 60%.¹³ Thus, restorations are important not only because of esthetic and functional concerns but also because there may be a positive psychological impact for the patient.

Preventive treatment for an AI patient is impossible because AI is genetic. However, there are many options proposed for the treatment of AI-affected teeth, including simple microabrasion,¹⁸ composite

veneers,¹⁹ porcelain laminate veneers, onlays,^{10,20-22} gold or stainless-steel crowns,^{20,23-25} metal ceramic crowns,^{10,20,21,23,26} and all-ceramic crowns.^{10,20} The treatment planning for patients with AI is related to many factors, including the age of the patient, the type and severity of the disorder, intraoral conditions, and the socioeconomic status of the patient.²¹ This clinical report describes the sequence of treatment by interdisciplinary approach to manage a case of AI from childhood to adulthood.

Case Report

A healthy Thai boy, 10 years of age, presented to the Faculty of Dentistry, Chulalongkorn University, with his parents. His major concerns were the appearance of his teeth and sensitivity to cold. His parents mentioned that his mother and grandfather also suffered from the same problem. A specialist in the Department of Oral Medicine examined the patient's disorder with clinical and radiographic examinations and diagnosed the patient with AI (hypoplastic type).

A comprehensive treatment plan was formed at the outset and was divided into three sessions. During preadolescence, restorations were performed to restore the defective tooth structure. Oral hygiene was also reinforced to reduce the risk of caries. In the early permanent dentition, the subject's malocclusion was treated orthodontically using fixed appliances. As the patient's growth ceased, permanent restorations were performed to improve both esthetics and function. The following provides the details of the comprehensive treatment plan.

At the first session, the patient was referred to the pediatric and operative departments for initial restorations to protect the remaining tooth structure against further wear and sensitivity by using resin composite veneers for the anterior and premolar teeth and stainless-steel crowns for the second deciduous molars and first permanent molars. Furthermore, occlusal sealants were performed on the premolar and molar teeth. Oral hygiene instructions were provided. The patient presented to the clinic every six months for an oral hygiene recall and application of 1.23% acidulated phosphate fluoride.

After getting an entire permanent dentition, the subject was referred to an orthodontist at the age of 14 to treat his underlying malocclusion. An intraoral examination revealed an anterior open bite. A posterior open bite and posterior cross bite were found at the right premolar and molar region, respectively. The upper dental midline was centered,

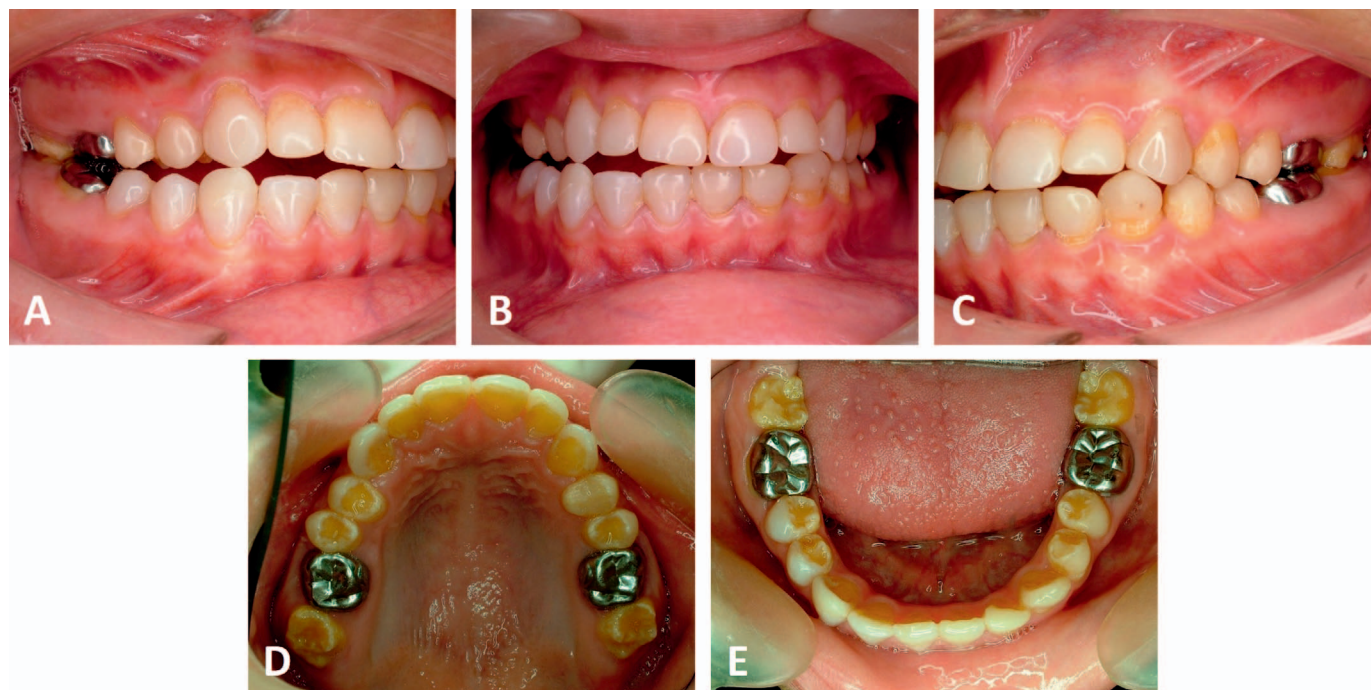


Figure 1. Intraoral views before orthodontic treatment. (A) Right-side view. (B) Frontal view. (C) Left-side view. (D) Occlusal view of the maxilla. (E) Occlusal view of the mandible.

but the lower dental midline was shifted to the right by approximately 2 mm. The molar relationship was class I. The canine relationship was class II on the right side and class I on the left side (Figure 1). Space analysis indicated a 2-mm space deficiency in the upper arch. The extraoral examination revealed a convex profile with lower lip retrusion (Figure 2). Cephalometric analysis indicated a class I skeletal relationship.

The treatment objectives were to obtain normal occlusion, normal tooth intercuspation, and normal overbite and overjet and to improve the soft tissue profile. The patient was treated using fixed orthodontic appliances. The posterior cross bite was corrected with maxillary expansion using a Quadhelix appliance (Figure 3), combined with buccal crown torque of the maxillary posterior teeth and lingual crown torque of the mandibular posterior teeth. The open bite was corrected using intermaxillary elastics (Figure 4). The total orthodontic treatment time was six years and eight months.

After completion of orthodontic treatment, the patient was recommended to wear retainers for six months to maintain the occlusion. Subsequently, the patient's overall dental condition and expectations were evaluated. At this point in the treatment, the patient wished to have good dental esthetics

and function. At 22 years of age, a clinical and radiographic reevaluation revealed that the interpupillary line and commissural line were parallel. The facial midline was centered. The patient's profile was slightly convex (Figure 5). The lower dental midline was shifted to the right by 2 mm when compared to the upper dental midline. Both overjet and overbite were 1 mm. The canine relationship was class II on the right and class I on the left. The occlusal scheme presented as canine protected occlusion. A clinical exam revealed that some composite veneers were defective. The lower-left second molar presented with an MO amalgam filling. In general, the enamel was hypoplastic and partially broken down. Exposed dentin could be observed (Figure 6). The overall root length and shape for the dentition was normal. The pulp chambers of all teeth were normal (Figure 7). The subject presented plaque-induced gingivitis. No active carious lesions were found.

The proportions of the upper anterior teeth were not symmetrical (Figure 8A). Therefore, the proper width of the teeth was calculated by using the repeated ratio or continuous proportion—the repeated proportion of the central-incisor-to-lateral-incisor width and lateral-incisor-to-canine width (as seen in a frontal view). The normal value is about 0.66 to



Figure 2. Extraoral views before orthodontic treatment. (A) Frontal view. (B) Lateral view.

0.78 (Figure 8B).^{27,28} Because of asymmetry of the gingival level, crown lengthening was proposed. However, the patient denied any periodontal surgery.

Due to the patient's concern, full-mouth rehabilitation was planned. The treatment was initiated on the posterior teeth by performing all ceramic crowns on the first permanent molars to provide good



Figure 3. Quadhelix appliance. (A) 2 months after insertion. (B) 9 months after insertion.

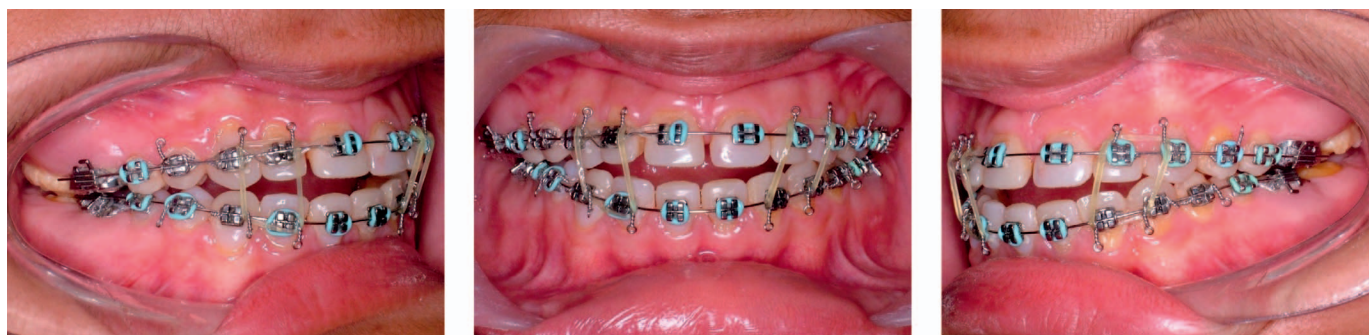


Figure 4. Intermaxillary elastics. (A) Right-side view. (B) Frontal view. (C) Left-side view.

posterior support before restoration of the other teeth. Stainless-steel crowns were removed from teeth #3, #14, #19, and #30, and all ceramic crown preparations were performed. For the upper first molars, a 3/4 zirconia core design (Lava) was chosen. For the lower first molars, an all-zirconia crown (Lava) was selected. Shade selection of all crowns was B2 Vita Classic. All crowns were temporarily fixed with Temp bond NE until the patient felt comfortable and reported no occlusal problems. A self-adhesive resin cement (Rely X Unicem, 3M

ESPE, St Paul, MN, USA) was used for permanent fixation.

The anterior and premolar area was restored with ceramic and resin composite veneers. The teeth (#5-#12 and #22-#27) were prepared for ceramic veneers using a chamfer margin and incisal bevel preparation (Figure 9). According to minimally invasive treatment guidelines, tooth preparation was performed using a labial silicone index made from a diagnostic wax-up as a guide. The final impression



Figure 5. Extraoral views before final restorations. (A) Frontal view. (B) Lateral view.

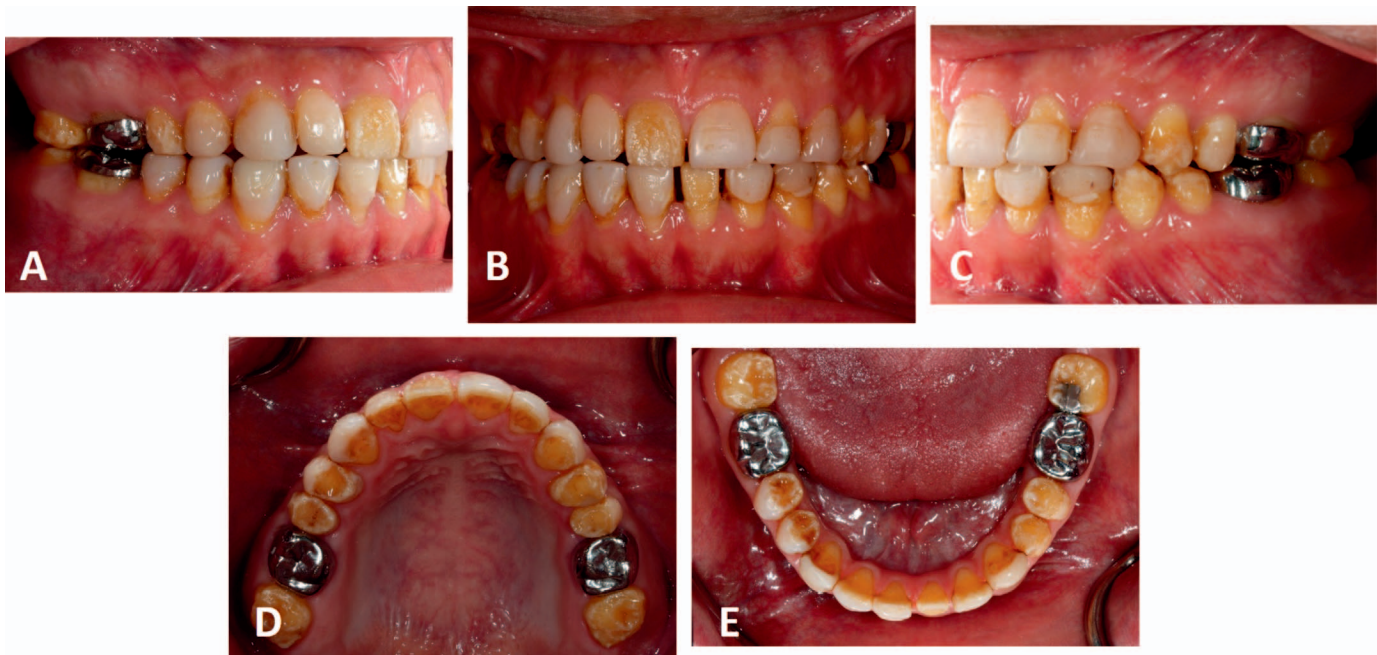


Figure 6. Intraoral views before oral rehabilitation. (A) Right-side view. (B) Frontal view. (C) Left-side view. (D) Occlusal view of the maxilla. (E) Occlusal view of the mandible.

was taken using polyvinyl siloxane (Flexitime, Heraeus Kulzer, South Bend, IN, USA). Bite registration and facebow transfer were performed. A palatal silicone index was used to fabricate temporary veneers using resin composite (Premise, Kerr Corp, Orange, CA, USA). Final working casts were mounted on a semiadjustable articulator (Artex CR, AmannGirrbach, Vorarlberg, Austria) with a customized anterior guide table. Ceramic veneers were

made from pressed ceramic (IPS Empress Esthetic, Ivoclar/ Vivadent, Liechtenstein) with a layering of veneering ceramic to mimic the natural enamel (Figure 10).

Shade selection was B1 Vita Classic. After the ceramic veneers were tried in, the appearance of #9-#11 was more yellowish than the other teeth. This appearance was caused by the underlying dentin color of those particular teeth. Therefore, white

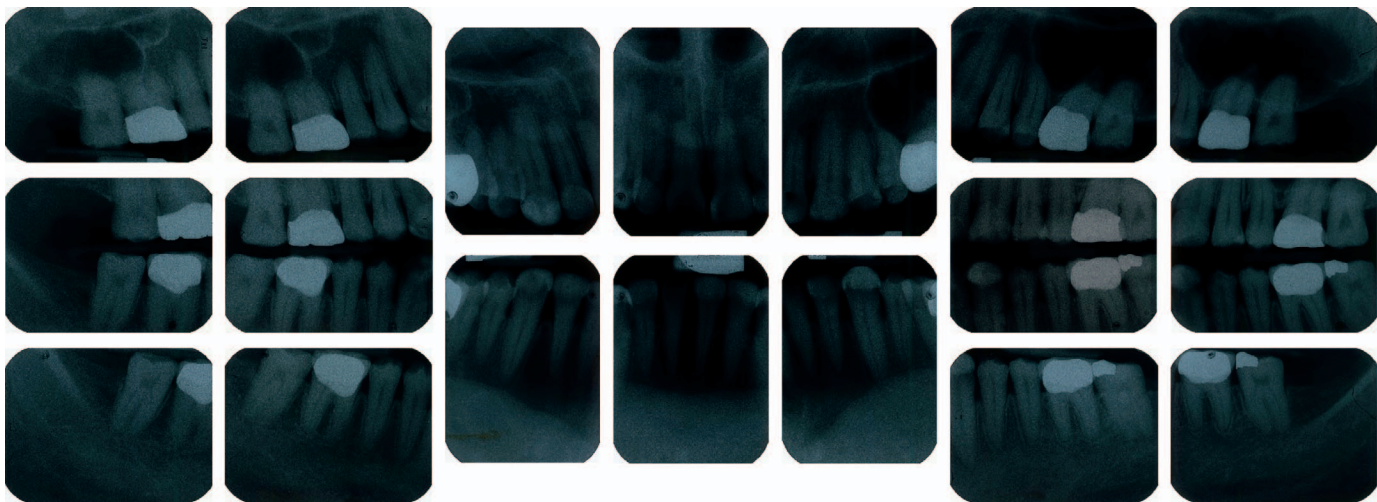


Figure 7. Full-mouth periapical radiograph.

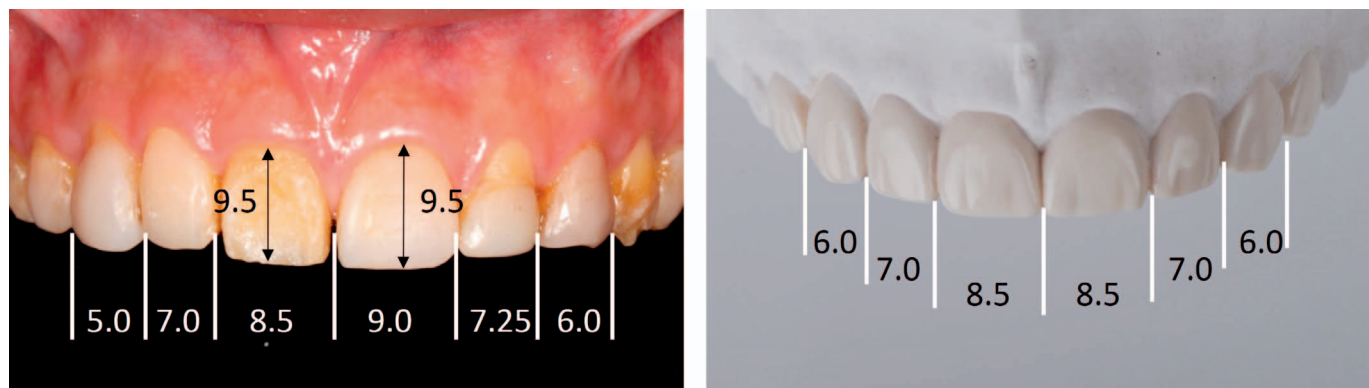


Figure 8. (A) An asymmetry of the width of upper anterior teeth. (B) A diagnostic wax-up of upper anterior teeth using the repeated ratio for calculating the proper width.

opaque, white, and white shades of the light-curing resin cement (NX3, Kerr) were chosen, respectively.

The clear shade of the light-curing resin cement was chosen for the remaining teeth (Figure 11). Every procedure was carried out following the manufacturer's instructions. The patient was instructed on the care of his restorations and was provided with an adequate oral home care regimen.

Direct resin composite veneers were performed on the upper second premolars and all lower premolars (#4, #13, #20, #21, #28, and #29). All of the teeth were etched with 37.5% phosphoric acid (Kerr Gel Etchant, Kerr). OptiBond FL (Kerr) bonding agent was then applied. Resin composite (Premise: opaque B1 and body B2, Kerr) was chosen as the restorative material.

Any exposed dentin (and the MO amalgam on #18) was restored with resin composite (Premise: body

B3, Kerr). After all treatment had been carried out, the patient was satisfied with the outcome for both esthetics and function (Figure 12). Upper and lower Hawley retainers were made to maintain the tooth alignment (Figure 13). Furthermore, upper and lower trays for GC Tooth Mousse were made to help prevent dental caries. The patient was instructed to wear these trays at least three minutes after brushing, following the manufacturer's recommendations (Figure 14). The patient was recalled at postoperative intervals of one week, one month, three months, six months, and one year.

DISCUSSION

The patient in the current case report had been treated since he was 10 years old. The skeletal growth had not completed yet at that age; therefore, the proper transitional treatment consisted of resin

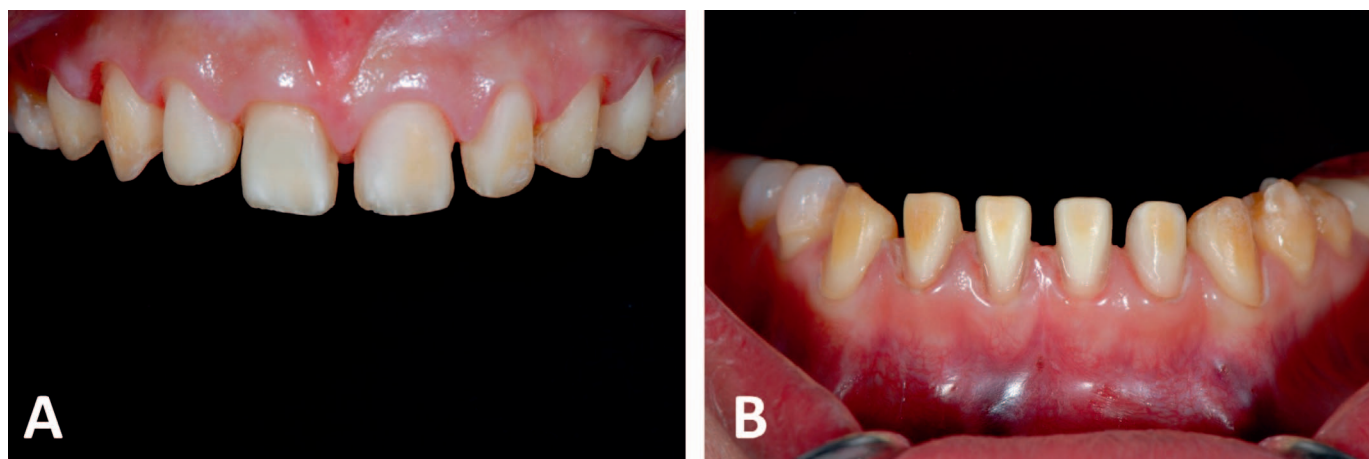


Figure 9. Preparation for ceramic veneers. (A) 5-12. (B) 22-27.

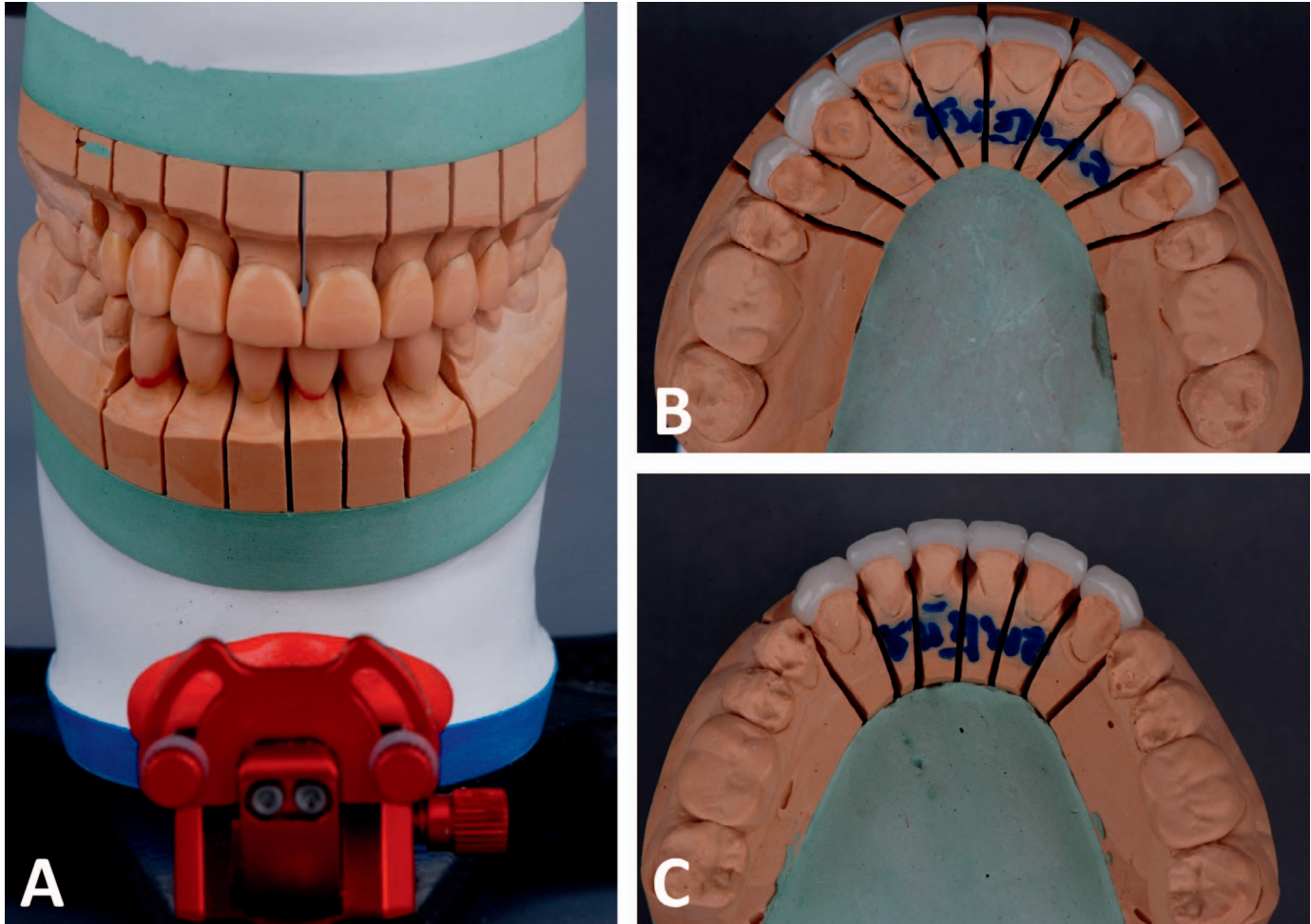


Figure 10. (A) Waxing for ceramic veneers. (B) Ceramic veneers 5-12. (C) Ceramic veneers 22-27.

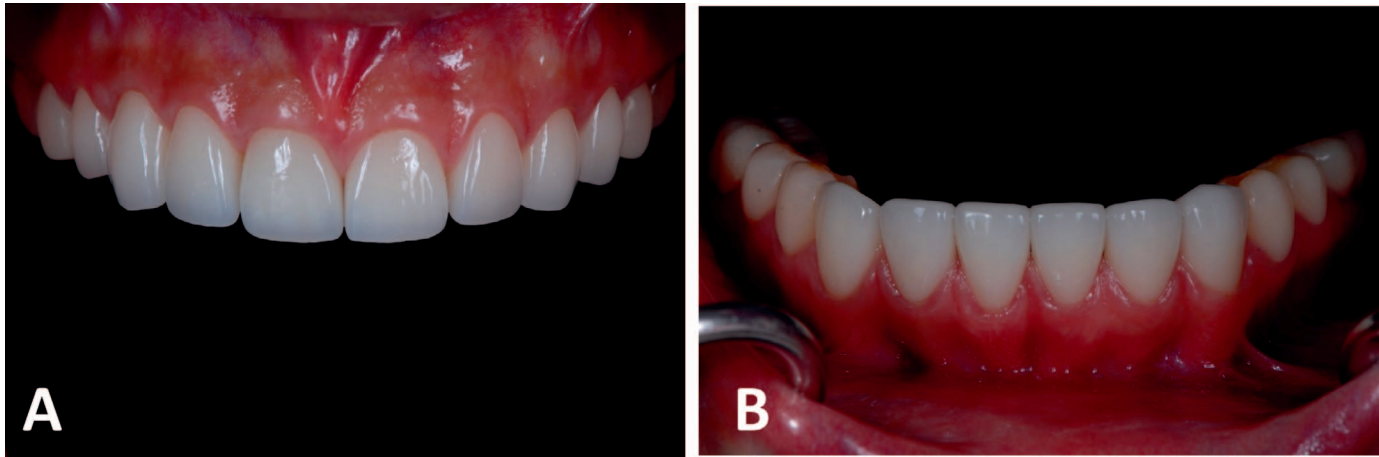


Figure 11. Ceramic veneers after cementation. (A) 5-12. (B) 22-27.

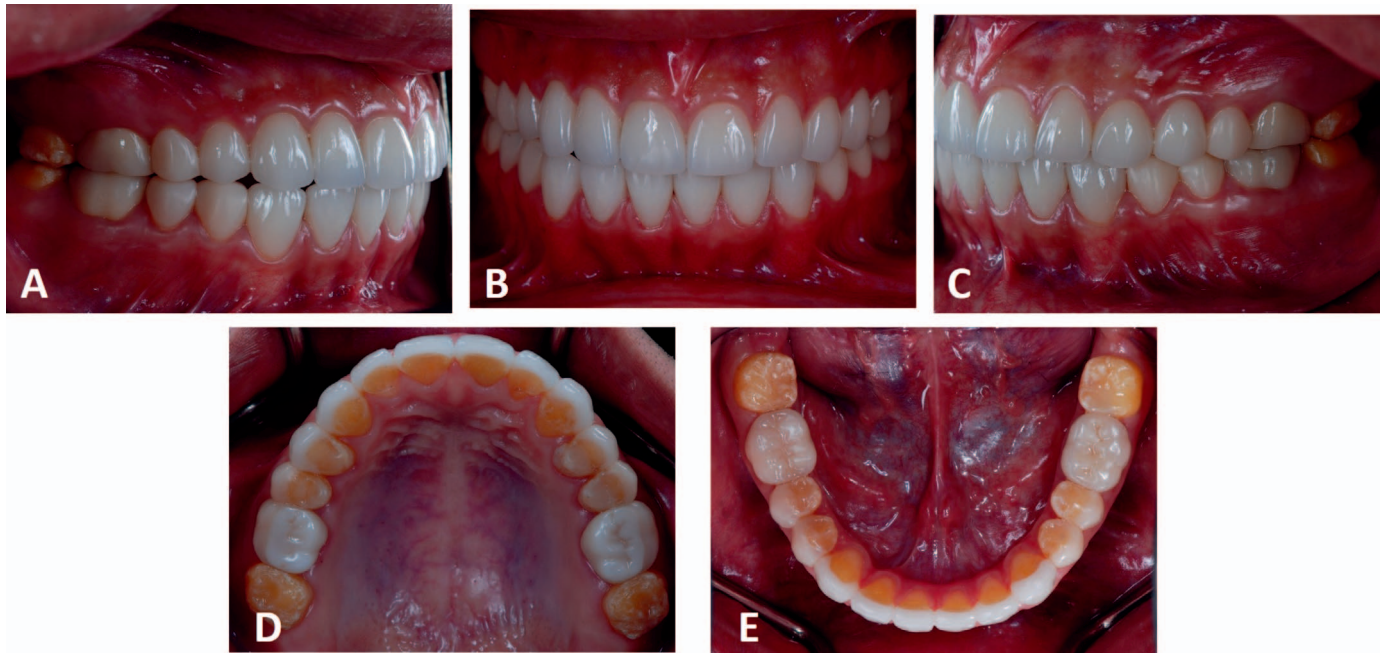


Figure 12. Final restorations six months after insertion (ceramic veneers at 5-12 and 22-27; resin composite veneers at 4, 13, 20, 21, 28, and 29; and all ceramic crowns at 3, 14, 19, and 30). (A) Right-side view. (B) Frontal view. (C) Left-side view. (D) Occlusal view of the maxilla. (E) Occlusal view of the mandible.

composite veneers and stainless-steel crowns. This treatment relieved tooth sensitivity and improved esthetics. Additionally, the resin composite veneer is considered the best way to preserve tooth structure since it requires a minimal preparation.^{12,29,30} However, there has been some evidence that for AI patients, especially those with poorly mineralized and friable enamel, the outcome of bonding between a resin composite and enamel is not as efficient as it is with normal enamel.³¹ Because of tremendous advances in the field of esthetic dentistry, especially in bonding to dentin, it is currently possible to restore function and esthetics to an acceptable level for areas with poor bonding substrates, as found in patients with AI.^{32,33}

With regard to the final restorations, the stainless-steel crowns were converted to all-ceramic crowns. It has been found that there was no significant difference in the five-year success rates between single unit zirconium oxide-based ceramic crowns and single-unit porcelain-fused-to-metal crowns.³⁴ Although all ceramic crowns seem to deliver a better biocompatible material and esthetics, they are prone to fracture, particularly for the veneering layer. Therefore, 3/4 zirconia cores and all zirconia crowns were selected for use with this patient. Additionally, zirconia cores require only 0.5 mm of occlusal

reduction, reducing the amount of enamel preparation. For the incisors and first premolars, ceramic veneers and resin composite veneers were chosen due to the minimal loss of labial enamel and no loss of lingual enamel. Because the patient had a limited budget, ceramic veneers were used only in the anterior zone. Crown restoration, which is a relatively invasive technique, is not justified in this case because the lingual surface of the teeth was intact. Moreover, veneer restorations have been proven to achieve a success rate of 80%-100% for patient satisfaction.^{35,36}

The case study of Vailati and Belser³⁷ suggested another idea for the treatment of generalized initial dental erosion. Those authors provided minimally invasive treatment using a thin occlusal ceramic onlay that required only 0.5-0.6 mm of enamel reduction and a palatal veneer for maxillary anterior teeth (IPS e. max Press, Impulse 01, Ivoclar Vivadent). Not only was the patient in that study satisfied with his outcome, but the process used in that study requires minimal preparation. However, that method demands much technical expertise for both the clinician and the dental technician.

Because of an irregularity of the enamel surface for patients affected with AI and the subsequent higher risk of developing dental caries, customized

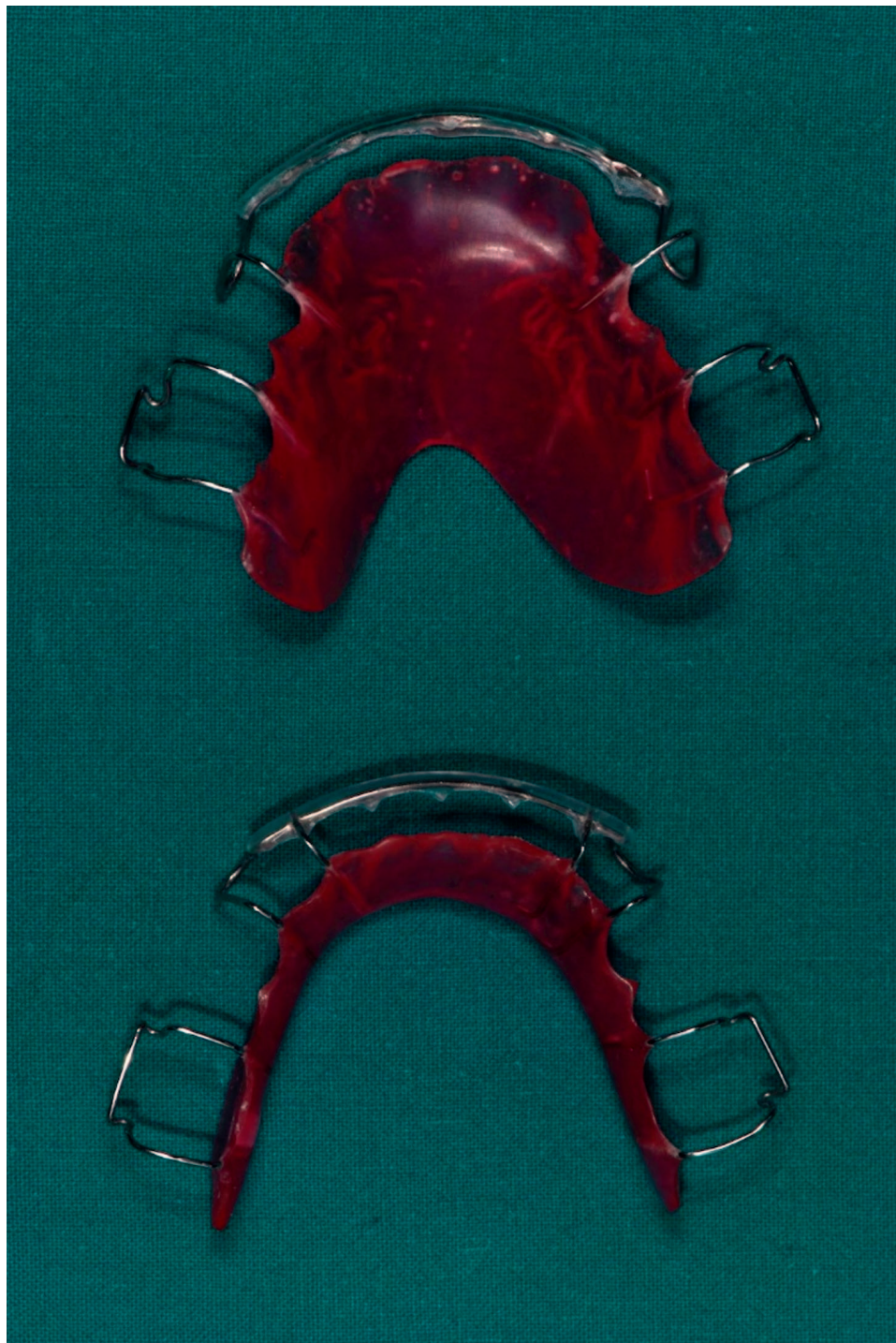


Figure 13. Hawley retainers.

trays (both upper and lower trays) and GC Tooth Mousse were presented to the patient. The GC Tooth Mousse was selected because it is a water-based cream containing RECALDENT (CPP-ACP: casein phosphopeptide–amorphous calcium phosphate) and its component, CPP-ACP, has been proven to promote the remineralization process.^{38,39}

CONCLUSION

As AI disorders noticeably affect the primary dentition, it is crucial to obtain a proficient diagnosis and effective treatment plan. An interdisciplinary approach should be used to treat the patient starting at the first stage, as not only is the tooth structure preserved but the esthetics and functional approach

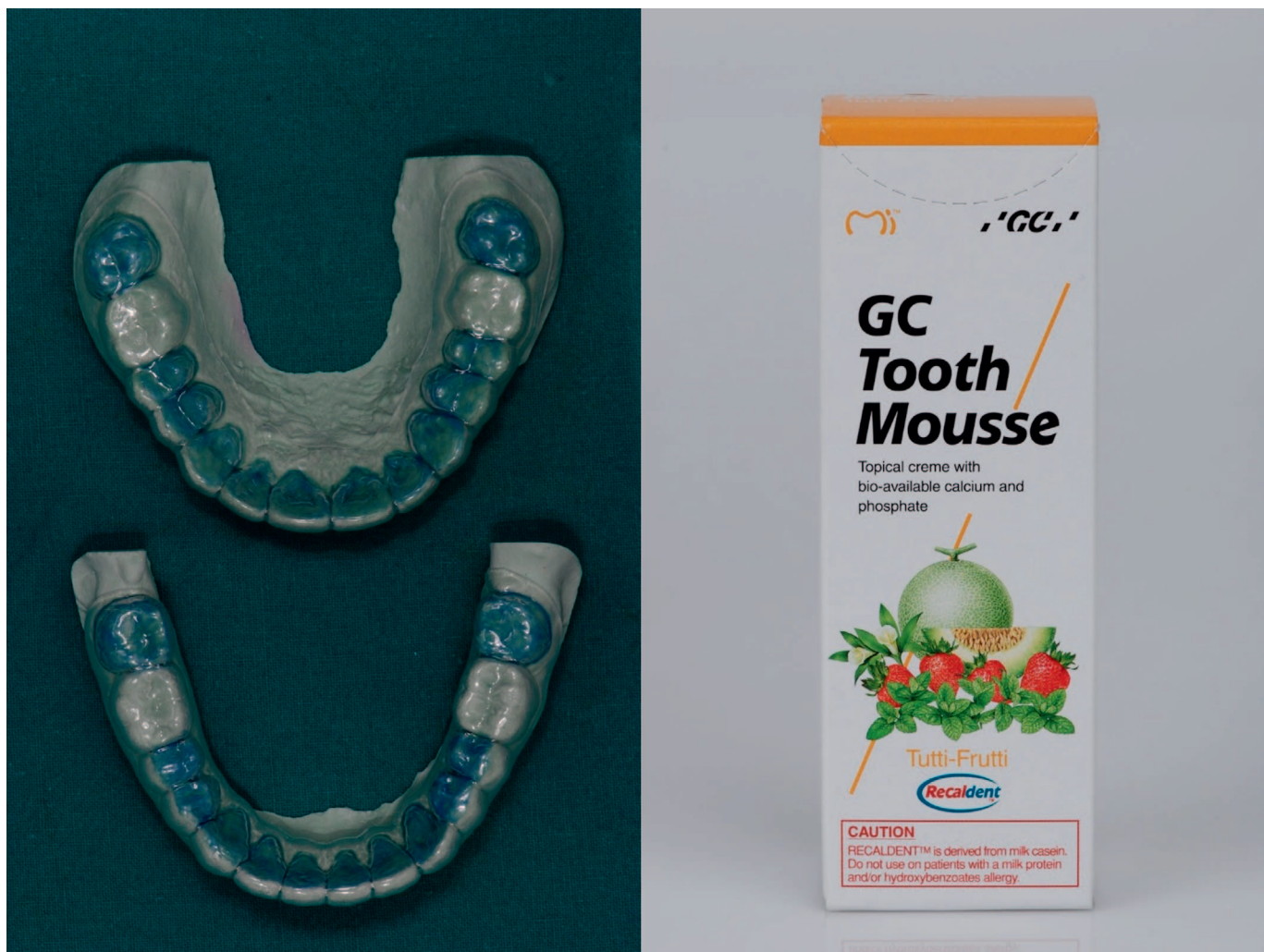


Figure 14. Upper and lower trays with GC Tooth Mousse.

are feasible as well. This case study demonstrates a thorough treatment plan that could be applied to other cases as guidance for an AI patient from the period of mixed dentition.

Conflict of Interest

The authors have no proprietary, financial, or other personal interest of any nature or kind in any product, service, and/or company that is presented in this article.

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REFERENCES

1. Coley-Smith A, & Brown CJ (1996) Case report: Radical management of an adolescent with amelogenesis imperfecta *Dental Update* **23**(10) 434-435.
2. American Academy of Pediatric Dentistry Council on Clinical Affairs (2008-2009) Guideline on oral health care/dental management of heritable dental development anomalies *Pediatric Dentistry* **30**(Supplement 7) 196-201.
3. Crawford PJM, Aldred M, & Bloch-Zupan (2007) Amelogenesis imperfecta *Orphanet Journal of Rare Diseases* **2** 17.
4. Bsoul SA, Flint DJ, Terezhalmay GT, & Moore WS (2004) Amelogenesis imperfecta *Quintessence International* **35**(4) 338-339.
5. Aldred MJ, Savarirayan R, & Crawford PJM (2003) Amelogenesis imperfecta: A classification and catalogue for the 21st century *Oral Disease* **9**(1) 19-23.

6. Neville BW, Damm DD, Allen CM, & Bouquot JE (2002) *Oral and Maxillofacial Pathology* 2nd ed. Elsevier, Philadelphia 89-94.
7. Soares CJ, Fonseca RB, Martins LR, & Giannini M (2002) Esthetic rehabilitation of anterior teeth affected by enamel hypoplasia: A case report *Journal of Esthetics and Restorative Dentistry* **14**(6) 340-348.
8. Witkop CJ Jr (1988) Amelogenesis imperfecta, dentinogenesis imperfecta and dentin dysplasia revisited: problems in classification *Journal of Oral Pathology* **17**(9-10) 547-553.
9. Akin H, Tasveren S, & Yeler DY (2007) Interdisciplinary approach to treating a patient with amelogenesis imperfecta: A clinical report *Journal of Esthetics and Restorative Dentistry* **19**(3) 131-135.
10. Nel JC, Pretorius JA, Weber A, & Marais JT (1997) Restoring function and esthetics in a patient with amelogenesis imperfecta *International Journal of Periodontics and Restorative Dentistry* **17**(5) 478-483.
11. Sundell S, & Koch G (1985) Hereditary amelogenesis imperfecta I. Epidemiology and clinical classification in a Swedish child population *Swedish Dental Journal* **9**(4) 157-169.
12. Robinson FG, & Haubenreich JE (2006) Oral rehabilitation of a young adult with hypoplastic amelogenesis imperfecta: A clinical report *Journal of Prosthetic Dentistry* **95**(1) 10-13.
13. Rowley R, Hill FJ, & Winter GB (1982) An investigation of the association between anterior openbite and amelogenesis imperfecta *American Journal of Orthodontics* **81**(3) 229-235.
14. Petters E, Cohen M, & Altini M (1992) Rough hypoplastic amelogenesis imperfecta with follicular hyperplasia *Oral Surgery, Oral Medicine and Oral Pathology* **74**(1) 87-92.
15. Aldred MJ, & Crawford PJM (1988) Variable expression in amelogenesis imperfecta with taurodontism *Journal of Oral Pathology & Medicine* **17**(7) 327-333.
16. Collins MA, Mauriello SM, & Tyndall DA (1999) Dental anomalies associated with amelogenesis imperfecta: A radiographic assessment *Oral Surgery, Oral Medicine and Oral Pathology* **88**(3) 358-364.
17. Encias RP, Garcia-Espona I, & Rodriguez de Mondela JMN (2001) Amelogenesis imperfecta: Diagnosis and resolution of a case with hypoplasia and hypocalcification of enamel, dental agenesis, and skeletal open bite *Quintessence International* **32**(3) 183-189.
18. Ashkenazi M, & Sarnat H (2000) Microabrasion of teeth with discoloration resembling hypomaturation enamel defects: Four-year follow up *Journal of Clinical Pediatric Dentistry* **25**(1) 29-34.
19. Sabatini C, & Guzman-Armstrong S (2009) A conservative treatment for amelogenesis imperfecta with direct resin composite restorations: A case report *Journal of Esthetics and Restorative Dentistry* **21**(3) 161-170.
20. Yip HK, & Smales RJ (2003) Oral rehabilitation of young adults with amelogenesis imperfecta *International Journal of Prosthodontics* **16**(4) 345-349.
21. Sari T, & Usumez A (2003) Restoring function and esthetics in a patient with amelogenesis imperfecta: A clinical report *Journal of Prosthetic Dentistry* **90**(6) 522-525.
22. Gemalmaz D, Isik F, Keles A, & Kukrer D (2003) Use of adhesively inserted full-ceramic restorations in the conservative treatment of amelogenesis imperfecta: A case report *Journal of Adhesive Dentistry* **5**(3) 235-242.
23. Bouvier D, Duprez JP, Pirel C, & Vincent B (1999) Amelogenesis imperfecta-prosthetic rehabilitation: A clinical report *Journal of Prosthetic Dentistry* **82**(2) 130-131.
24. Quinonez R, Hoover R, & Wright JT (2000) Transitional anterior esthetic restorations for patients with enamel defects *Pediatric Dentistry* **22**(1) 65-67.
25. Rosenblum SH (1999) Restorative and orthodontic treatment of an adolescent patient with amelogenesis imperfecta *Pediatric Dentistry* **21**(4) 289-292.
26. Lumley PJ, & Rollings AJ (1993) Amelogenesis imperfecta: A method of reconstruction *Dental Update* **20**(6) 252-255.
27. Lombardi RE (1973) The principle of visual perception and their clinical application to denture esthetics *Journal of Prosthetic Dentistry* **29**(4) 358-382.
28. Rosenstiel SF, Ward DH, & Rashid RG (2000) Dentists' preferences of anterior tooth proportion—A web-based study *Journal of Prosthodontics* **9**(3) 123-136.
29. Ozturk N, Sari Z, & Ozturk B (2004) An interdisciplinary approach for restoring function and esthetics in a patient with amelogenesis imperfecta and malocclusion: A clinical report *Journal of Prosthetic Dentistry* **92**(2) 112-115.
30. Turkun LS (2005) Conservative restoration with resin composites of a case of amelogenesis imperfecta *International Dental Journal* **55**(1) 38-41.
31. Venezie RD, Vadiakas G, Christensen JR, & Wright JT (1994) Enamel pretreatment with sodium hypochlorite to enhance bonding in hypocalcified amelogenesis imperfecta: Case report and SEM analysis *Pediatric Dentistry* **16**(6) 433-436.
32. Nakabayashi N, & Pashley DH (1998) *Hybridization of Dental Hard Tissues* Quintessence Publishing, Chicago 37-39.
33. Yoshida Y, van Meerbeek B, & Nakayama Y (2001) Adhesion to and decalcification of hydroxyapatite by carboxylic acids *Journal of Dental Research* **80**(6) 1565-1569.
34. Vigolo P, & Mutinelli S (2012) Evaluation of zirconium-oxide-based ceramic single-unit posterior fixed dental prostheses (FDPs) generated with two CAD/CAM systems compared to porcelain-fused-to-metal single-unit posterior FDPs: A 5-year clinical prospective study *Journal of Prosthodontics* **21**(4) 265-269.
35. Meijering AC, Creughers NH, Roeters FJ, & Mulder J (1998) Survival of three types of veneer restorations in a clinical trial: 2.5-year interim evaluation *Journal of Dentistry* **26**(7) 563-568.
36. Rucker LM, Richter W, MacEntee M, & Richardson A (1990) Porcelain and resin veneers clinically evaluated: 2

- year results *Journal of the American Dental Association* **121**(5) 594-596.
37. Vailati F, Bruguera A, & Belser UC (2012) Minimally invasive treatment of initial dental erosion using pressed lithium disilicate glass-ceramic restorations: Case report *Quintessence Dental Technology* **35** 65-78.
38. Cochrane NJ, Cai F, & HuqNew NL (2010) Approaches to enhanced remineralization of tooth enamel *Journal of Dental Research* **89**(11) 1187-1197.
39. Sudjalim TR, Woods MG, & Manton DJ (2006) Prevention of white spot lesions in orthodontic practice: A contemporary review *Australian Dental Journal* **51**(4) 284-289.