Amelogenesis imperfecta and anterior open bite: Etiological, classification, clinical and management interrelationships

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ABSTRACT

Although amelogenesis imperfecta is not a common dental pathological condition, its etiological, classification, clinical and management aspects have been addressed extensively in the scientific literature. Of special clinical consideration is the frequent co-existence of amelogenesis imperfecta with the anterior open bite. This paper provides an updated review on amelogenesis imperfecta as well as anterior open bite, in general, and documents the association of these two separate entities, in particular. Diagnosis and treatment of amelogenesis imperfecta patients presenting also with anterior open bite require a lengthy, comprehensive and multidisciplinary approach, which should aim to successfully address all dental, occlusal, developmental, skeletal and soft tissue problems associated with these two serious clinical conditions.

Key words: Amelogenesis imperfecta, anterior open bite, multidisciplinary dental treatment

INTRODUCTION

Although amelogenesis imperfecta (AI) is not a common dental pathological condition (its prevalence ranges from 1 in 2000 up to 1 in 18,000 cases),[1-3] its etiological, classification, clinical and management aspects have been addressed extensively in the scientific literature. For the clinician, of great importance is the cause and effect relationship of abnormal dentoskeletal characteristics in AI patients, especially in open bite cases. This paper aims to provide an updated review on AI and anterior open bite (AOB), in general, and to document the association of these two separate entities in many patients.

ETIOLOGY OF AMELOGENESIS IMPERFECTA

AI pertains to a group of developmental tooth abnormalities (also referred as hereditary dysplasia),[4] which affect the genome of the individual and regard at least one of the stages of enamel formation.[5] AI is, in general, a hereditary disorder with clinical impact on both deciduous and permanent teeth.[6-11] AI was first described in 1890, but the substantial separation from dentinogenesis imperfecta was not made until 1938, when AI was described as an autonomous entity.[7,12]

Regarding AI's etiology, numerous studies have reported a variety in its inheritance pattern, including either autosomal, X-linked, dominant or recessive models.[13] Sundell and Valentin[14] shed light on how each specific form of AI is inherited, unraveling the indistinct aspects. More specifically, it was mentioned that enamel hypoplasia is inherited predominantly in a sex-linked, incomplete, dominant trait, whereas enamel hypomineralization in an autosomally dominant manner.[15,16] Conversely, AI's inheritance, in general, is quoted to be mainly autosomal dominant, without excluding recessive X-linked or sporadic inheritance.[7,17] Variations in gene expressions lead to numerous gene defects, which consecutively alter the phenotype.[4,18-21] More specifically, the ENAM mutation c. 1258_1259insAG is implicated for the occurrence of hypoplastic phenotype in cases of homozygosity.[22] ENAM mutations are transmitted in an autosomal-dominant manner and are expressed with the hypoplastic type of AI. In cases of hypoplastic AI, abnormal tooth eruption and coronal resorption appear, but it has yet to be proved if the abnormal function of the enamel epithelium and ameloblasts cause these

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Access this article online

Quick Response Code:

Website: www.jorthodsci.org

DOI: 10.4103/2278-0203.127547

two problems. Abnormal tooth eruption is irrelevant to ENAM mutations and enamelin appears to have no impact on tooth eruption.[22] Elsewhere, it is presumed that responsible is the gene codifying amelogenin (the most abundant protein of enamel) and more specifically differentiations in degradation and resorption are the ones that lead to the occurrence of hypomineralized enamel.[23,24] Conclusively, albeit all the progress that has already been achieved, there are still inadequate and vague aspects that are to be enlightened, in order to completely acknowledge the physiopathology of this entity.[23] It is important to point out the correlation of AI with two rather rare syndromes named Jalili syndrome and enamel dysplasia with hamartomatous atypical follicular hyperplasia (EDHFH) syndrome. Jalili syndrome refers to the co-existence of cone rod dystrophy (CRD) and AI, due to a mutation of the CNNM4, which is a metal carrier. A variety of symptoms including visual deficiency, abnormal dentition, photophobia, nystagmus increasing under photopic conditions can also be presented with AI. It may be fully demonstrated either in the infancy or in the childhood.[25] The second syndrome is exclusively reported in black South Africans. Hamartomatous atypical follicular hyperplasia with features similar to central odontogenic fibroma in multiple impacted teeth and also a generalized enamel dysplasia with features of hypoplastic AI are nearly always present. Other conditions often mentioned are open-bite malocclusion, gingival overgrowth, hypodontia, pulpal calcifications and aberrant root formation of the unerupted teeth.[26]

A recent report identified a FAM83H mutation in two of six unrelated families with autosomal dominant hypocalcified AI and found limited phenotypic variation of the enamel in these patients.[27]

CLASSIFICATION OF AMELOGENESIS IMPERFECTA

AI can be generally and roughly divided into three categories; hypoplastic (enamel is thin and stained, but normally calcified), hypocalcified (soft enamel that can be removed without difficulty) and hypomature (enamel is of normal thickness, but of reduced hardness {harder than the hypocalcified form} and its color varies from yellow/brown to red/brown).[2,13,22,28] Classification is mainly based on various criteria, not only the clinical appearance of the enamel (as the one mentioned above), but also pattern of inheritance, phenotypical abnormalities and molecular disorders, biochemical analysis of the enamel, etc.[29-31] The number of AI subtypes mentioned in the majority of the reports is 12.[32-39] However, 14 categories of AI have also been mentioned.[40]

CLINICAL MANIFESTATIONS OF AMELOGENESIS IMPERFECTA

A variety of symptoms can be presented with AI. The most substantial findings comprise extensive loss of tooth tissue, tooth sensitivity, excessive attrition leading to short clinical crowns, spacing in the anterior region of the dentition, normal or tight proximal contacts in the posterior region, and a general enamel caries resistance.[7,15] Reduced enamel thickness combined with normal hardness and radio-opacity in the case of hypoplastic AI has been described, whereas in the case of hypomature or hypocalcified AI enamel is of normal thickness, but softer and of reduced radio-opacity.[3] The diversity of manifestations is thoroughly delineated. Abnormal tooth eruption, morbid root and coronal resorption, congenitally missing teeth, malocclusions, AOB, pulpal calcification, dentin dysplasias, hypercementosis, root malformations and taurodontism have been ordinarily reported.[3,13,14,31,41,42] It should not be omitted from quoting the surface irregularities and the crown discoloration (mainly of yellowish brown shade).[15] Histological analysis could not prove the existence of prismatic architecture in enamel, whereas clinically and histologically the dentin was not malformed or pitted.[3,43]

MANAGEMENT OF AMELOGENESIS IMPERFECTA

An AI individual should be treated by a range of different specialists like pediatric dentists, orthodontists, maxillofacial surgeons and restorative dentists and not necessarily only in that order. The management is often complex, takes a significant amount of time (more commonly from childhood to early adulthood), but its positive psychological effect on a wounded self-esteem is priceless, thus replacing the counseling therapies that could be otherwise needed in addition to the dental approach.[6,44-47] As enamel of an individual with AI is deprived of the normal prismatic structure, many questions arise about the efficacy of bonding-based restorative options. Many AI cases have been treated in the past successfully enough with acid etch bonding methods, implying that only the total absence of enamel layer leads with certainty to the failure of rehabilitation. The minimum standard is the existence of a thin, even non-prismatic layer of enamel.[22] The age of the patient is a significant factor in order to decide which treatment path will be followed. Direct composite restorations are strongly recommended for children and adolescents with AI, as they can be easily adjusted according to the dento-alveolar development and they are minimally invasive. Indirect restorations represent a more preferable solution for adults, where an overall extensive treatment might be required.[6,48-51] As the field of micromechanical adhesions is increasingly advanced and the genotypical determination of each specific type of AI is not far away, it is anticipated that in the future the appropriate treatment will be chosen upon the gene-based diagnosis, leading to the best-achieved outcome.[20,22] As AI is characterized by a clinical diversity, a generic approach suitable in all cases of AI is to remove the discolored tooth substance as well as the defective tooth tissue and to cover-up (masking) the defects. To achieve the best feasible, the tooth substance should be reduced as less as possible result.[52]

ANTERIOR OPEN BITE IN GENERAL

AOB is the failure of the incisors to overlap and is a vertical problem that requires orthodontic treatment for its correction. AOB can arise from a number of causes such as inherited patterns, defects in embryologic development, trauma or functional influences. Specific genetic syndromes or congenital defects involving the jaws are rare, as are malocclusions caused primarily by trauma. Altered soft tissue function and sucking habits have traditionally been associated with vertical growth problems, especially AOB.[53] Lack of eruption of the upper incisors can possibly cause AOB, but rarely is the main reason. Instead, there is frequently an excessive eruption of posterior teeth, which in combination with the eruption of anterior teeth in a normal amount, inevitably results in AOB. This excessive eruption of posterior teeth results to a compensatory downward and backward rotation of the mandible. On the other hand, in other patterns of growth, where the mandible rotates downward and backward, space is created into which the posterior teeth can erupt, allowing excessive posterior dentoalveolar development.[54] Moreover, as the AOB is much more common in Blacks than Whites, whereas deep bite is much more common in the later group, it seems reasonable that this reflects a different inherent facial morphology rather than environmental influences.[55,56]

Skeletal characteristics associated with AOB include increased anterior face height, steep mandibular plane, excessive eruption of posterior teeth, maxillary constriction and high mandibular angle.[8,10,43,57-61] AOB can be found in all different malocclusions.

It appears that problems related to both esthetics and oral function can produce a significant need for orthodontic treatment. The management of AOB is a difficult issue and the results are not always predictable. If the AOB is diagnosed before growth spurt, the growth modification treatment may be successful.[62] On the other hand, if AOB is diagnosed after growth spurt and treated with orthodontic extrusion of the anterior teeth, relapse cannot be avoided.[63,64] In this case, orthognathic surgery by means of Le Fort I intrusion osteotomy, in most of the cases, is believed to be the effective treatment.[9,65]

ANTERIOR OPEN BITE IN ASSOCIATION WITH AMELOGENESIS IMPERFECTA

AOB and AI separately do not occur often in the general population. However, clinical studies indicate that AOB is more commonly observed in patients with AI than in the general population.[66-69] Regarding the etiology of this co-existence a discussion arose regarding whether this common factor is genetic, skeletal or local, with most of the opinions advocating that this factor is of genetic origin. It has been found that in patients with AI, who also carry ENAM or AMGX mutations, AOB is more often observed.[68,70-73] Patients with generalized hypoplastic AI, AOB and Class II malocclusion carry a homozygoys ENAM g13185-13186 insAG mutation, whereas patients with the heterozygous type of this mutation have only localized hypoplastic AI with AOB[73] or without AOB.[71] The vertical craniofacial growth is determined by a variety of genes. It is possible that an unidentified gene influences the gene associated with this growth (e.g. ENAM and AMGX).[31] Many have claimed that the association between AOB and AI is due to a genetically determined anomaly of craniofacial development affecting alveolar growth.[28,73,74] It has also been suggested that disturbances of the enamel epithilium can cause defects in the eruptive mechanism thus resulting in AOB.[75] It has been noted that the pleiotropic action of AI genes affects the development of the craniofacial complex.[76,77] Studies have proved that deficiencies in formation, migration and the proceeding development of neural crest cells (enamel and skeleton of craniofacial complex may share a common neural crest origin) may lead to a number of congenital anomalies of craniofacial complex.[78,79] Subjects with AOB and AI present the same lateral cephalometric radiographic and clinical characteristics (posterior maxillary vertical hyperplasia, high palatal vault, accentuated maxillary but reversed mandibular curve of Spee, omega-shaped maxillary dental arch and transverse discrepancy between maxillary and mandibular dental arches) with AOB but non-AI subjects. All these indicate that AOB in AI subjects is of skeletal origin.[80] Clinical investigations have shown that an anomaly of vertical jaw relationship in combination with increased lower anterior facial height has been observed in many AI patients.[28]

Consequently, vertical dysgnathia is the main reason why AOB is so frequently observed in patients with AI. Vertical dysgnathia increases the anterior maxillomandibular distance leading to AOB. At the same time, the increased lower anterior facial height also results in incompetent lips. The tongue, thus, may not behave normally either at rest position or during swallowing and as a result an anterior oral seal can be produced. This tends to cause or retain AOB, which in these patients cannot be attributed to other potential local factors.[28] Based on the above-mentioned statements, it can be said that the growth of dentoalveolar complex may be inhibited by the tongue. It is highly unlikely that this could modify the morphology of craniofacial complex to the extent that it has been found from the lateral cephalometric radiographic analyses.[28] It is claimed that the increased sensitivity of teeth either to cold or to hot predisposes to a tongue interposition, which acts as a mechanical obstacle to the vertical alveolar growth.[29,37] Contrary to the above, the association between AOB and teeth hypersensitivity has been questioned.[28] Most of the studies which investigated the prevalence of AOB in subjects with AI concluded that the prevalence varies between 24% and 60%.[28,29] Vertical dysgnathia in AI individuals was found more often in women than in men, but without any statistically significant difference. AOB most frequently occurred in the hypocalcification type of AI, less in the hypoplastic type, and it was totally absent in the hypomaturation type.[28]

MANAGEMENT OF AMELOGENESIS IMPERFECTA COMBINED WITH ANTERIOR OPEN BITE

Orthodontic correction of AOB is considered to be very difficult due to the high relapse rate observed in many cases.[80,81] Inevitably, surgical treatment of patients with AOB combined with AI is one of the challenging alternative therapeutic modalities in orthognathic surgery.[15] Treatment of such patients is a challenge for any clinician and a multidisciplinary approach by a team of orthodontist, pediatric dentist, maxillofacial surgeon and prosthodontist is usually necessary. Rehabilitation of such patients is best carried out in a specialized unit of a dental hospital.[2] Many factors should be taken into account such as the age of the patient, the quality and quantity of enamel, the periodontal condition and the skeletal developmental status.[2,17] It is of vital importance to inform parents about the situation, the prognosis and the cost of the treatment.[6] At the first stage of the treatment tooth sensibility should be reduced and attrition should be prevented. Patients should be informed about oral hygiene and all measures of prophylaxis should be taken into consideration.[17] The pre-surgical orthodontic treatment should aim at the correction of the transversal relation with rapid maxillary expansion or surgically assisted rapid maxillary expansion. Alternatively, a three-piece Le Fort 1 osteotomy will be used during the operation. In cases of vertical dentoalveolar discrepancies, orthodontic alignment is difficult because of the enamel conditions and the height of the crowns. In this case, a multi-segment Le Fort 1 intrusion osteotomy is the choice of treatment[82-84] combined with bilateral sagittal split osteotomy or genioplasty, where required.[84,85] As a result, rehabilitation of the maxillary hyperplasia, the accentuated curve of Spee and the omega-shaped maxillary dental arch can be achieved in a one-stage surgical procedure with limited or no pre-surgical orthodontics.[15] The risk of periodontal complications and damage to roots are some of the drawbacks of multi-segment Le Fort 1 osteotomy, which should be taken into account. Vascular impairment may also lead to aseptic necrosis of teeth, loss of teeth or even loss of major maxillary dentoalveolar segments.[86] Since a multi-segment Le Fort 1 osteotomy is often followed by a high rate of relapse, rigid internal fixation with micro-screws or mini-plates offers better transverse skeletal stability[80,84] and more adaptive condylar capacity when applied.[87] It is suggested to use silicone putty indices during the surgery to maintain control of the occlusion as well as a staged replacement of the previous restoration in order to make the intercuspal and retruded contact position coincident.[2] A full cover occlusal splint can be utilized, when the height of the crown allows, for maintaining the segments in the desired position on the arch bar.[15] It is highly significant that the surgeon is informed about the planned prosthetic rehabilitation. The reduced height of crowns in these patients may require a larger than normal freeway space and the vertical position of the maxilla should be determined by the relationship between the upper lip and the incisal edge of the maxillary incisors.[15,88]

Regarding the quality of dentin, a thicker peritubular dentin is found in hypocalcified AI and dentin tubuli are partly or completely sclerotic. All the other types of AI have the same or similar characteristics.[89] Glossiness, discoloration or abrasiveness can be the clinical signs of sclerotic dentin.[17] The bonding strength of sclerotic dentin is less effective compared to that of healthy dentin.[90-92] Self-etching primers or short etching with phosphoric acid (total etch) are recommended according to some studies.[92] A bracket or button may be bonded to the maxillary teeth to support the arch bar giving a solution to the difficulty of applying the ligatures around the teeth due to the dentin conditions, tight contact surfaces between abraded short premolars or molars, and the conical shape of teeth in the anterior region. The arch bar in the mandible can be fixed with 4 or 6 circummandibular wires.[15]

After surgery, when facial harmony as well as vertical and sagittal occlusal relationships are improved, orthodontic refinement should follow.[17] During the settling period, stabilization with an occlusal splint or with temporary restorations can be useful to the patients who are getting used to the new habitual resting position. Final stabilization of the occlusion and articulation with permanent fixed crowns in centric relation is performed 1 year after the surgery.[15] The prosthodontic treatment of such teeth includes either application of adhesive material or fillings,[93] especially in transitional treatment of adolescent patient[94] or use of gold or porcelain-fused-to-metal crowns fixed with conventional cements (e.g. zinc-phosphate cements).[17] Although the loss of tooth substance is greater in this case due to shoulder preparation, metal crowns are considered to have high precision and long-term results.[17]

It is highly significant that a strict recall system and a good oral hygiene protocol are applied.[17] Due to the young age of the patients, parents should be well informed about the prolonged period of the multidisciplinary treatment. Studies have shown that biological and technical failures in rehabilitation of AI patients are no higher than those with no birth defects and conventional fixed reconstruction.[95] Complications of fixed reconstructions tend to appear after 10 years of function or later.[96] Other complications may include loss of transverse stability (60% of patients), but this can be prevented by rigid internal fixation or progressive condylar adaptation (13%).[84]

CONCLUSIONS

AOB is more commonly observed in patients with AI than in the general population implying the presence of a common factor associated with these two conditions.

Diagnosis and treatment of AI patients with AOB require a lengthy, comprehensive and multidisciplinary approach, which should aim to successfully address all dental, occlusal, developmental, skeletal and soft tissue problems associated with these two serious clinical conditions.

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How to cite this article: Alachioti XS, Dimopoulou E, Vlasakidou A, Athanasiou AE. Amelogenesis imperfecta and anterior open bite: Etiological, classification, clinical and management interrelationships. J Orthodont Sci 2014;3:1-6.

Source of Support: Nil, **Conflict of Interest:** None declared.