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The dental management and prosthodontic reconstruction of patients with amelogenesis imperfecta: A narrative review

Christina I. Wang [a,*], Naif Sinada [a], Todd R. Schoenbaum [b]

[a] Private Practice, 3394 N Futrall Drive, Fayetteville, AR 72703, United States
[b] Restorative Sciences, Dental College of Georgia, Augusta University, United States

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ABSTRACT

Amelogenesis imperfecta (AI) is a rare genetic condition that affects normal enamel formation of both the primary and permanent dentition. Patients with AI not only present with restorative challenges, but also suffer from extreme hypersensitivity, compromised esthetics, and poor self-esteem. The functional, social, and psychological impairment often leads to an overall lower quality of life. Extensive dental treatment is often required at a young age, therefor early diagnosis, appropriate preventative strategies, and proper dental management is essential for successful treatment outcomes throughout all phases of life. This review presents the dental management and prosthetic reconstruction of patients with amelogenesis imperfecta.

1. Introduction

Amelogenesis imperfecta (AI) is a rare genetic disorder affecting normal enamel formation of both the primary and permanent dentition [1]. Disruption in any of the three developmental stages of normal enamel formation [1–3] causes a diverse phenotypic clinical presentation [1] reflecting changes in the microstructure, mineral composition, and therefore quality and/or quantity of the enamel [4,5]. The prevalence of AI in the United States is 1 in 14,000 [6] but varies depending on the country or population being studied [1]. AI can be found in isolation or associated with other syndromes and conditions [7]. Various gene mutations have been identified [8,9] and can be inherited via: autosomal dominant, autosomal recessive, X-linked [10], or by sporadic inheritance with no familial history [11]. Although some genetic mutations have been identified, genetic testing has shown only a 25 % success rate in identifying the genetic cause of isolated AI [9,12], however, recently Bloch-Zupan et al. had a 60 % diagnostic rate [7]. Typically, AI diagnosis is more often based on clinical and radiographic findings [13,14]. Clinical expression of AI, however, is extremely variable and can make identification difficult. Some cases of AI are very subtle, and may appear relatively normal to the untrained eye, while other cases may have extensive break-down of all the enamel [11,14,15].

2. Background

2.1. Classification

Classification of AI can be broken down into 4 main types: hypoplastic (type 1), hypomaturation (type 2), hypocalcified (type 3), and hypomaturation-hypoplastic with taurodontism (type 4), which can be further subdivided into 15 different subtypes [15]. Identification and distinction is difficult as the main types of AI clinically overlap, with phenotypic variation even among family members [1]. Characteristics of multiple AI types may also coexist in the same patient or same tooth [16]. The incidence of hypoplastic (type 1) AI has been found most frequently at 61.2 %, followed by hypomatured (type 2) AI at 32.2 %, and hypocalcified (type 3) and hypomaturation-hypoplastic with taurodontism (type 4) AI show a combined rate of 3.2 % [17].

Hypoplastic (type 1) AI (Figs. 1–7) occurs when there is a secretory defect in the enamel matrix formation [1]. This results in a quantitative alteration which can be localized, generalized, or in extreme cases a complete absence of the enamel [1,14]. The surface texture is often rough with pitting or larger defects, while the color of the teeth can appear normal or anywhere from yellow to orange or brown [14]. Radiographically, the reduction in enamel thickness can often be noted (Fig. 2b), and the enamel still contrasts normally from the dentin [13,15].

With hypomaturation (type 2) AI (Figs. 8–10), the removal of the

* Corresponding author.
E-mail address: christinawangdmd@gmail.com (C.I. Wang).

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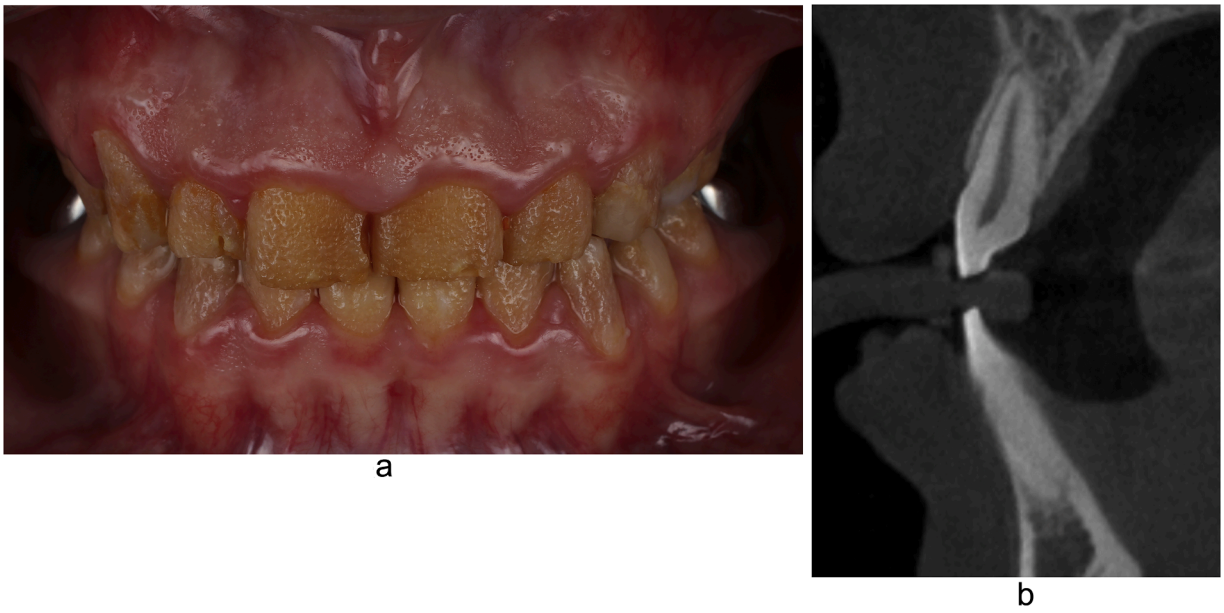

Fig. 1. A 16-year old patient with hypoplastic (type 1) amelogenesis imperfecta.
(a) Intra-oral frontal view. Generalized pitting with an orange/brown appearance and shortened clinical crown heights noted. (b) CBCT slice of #8 exhibiting a generalized decreased thickness of enamel.

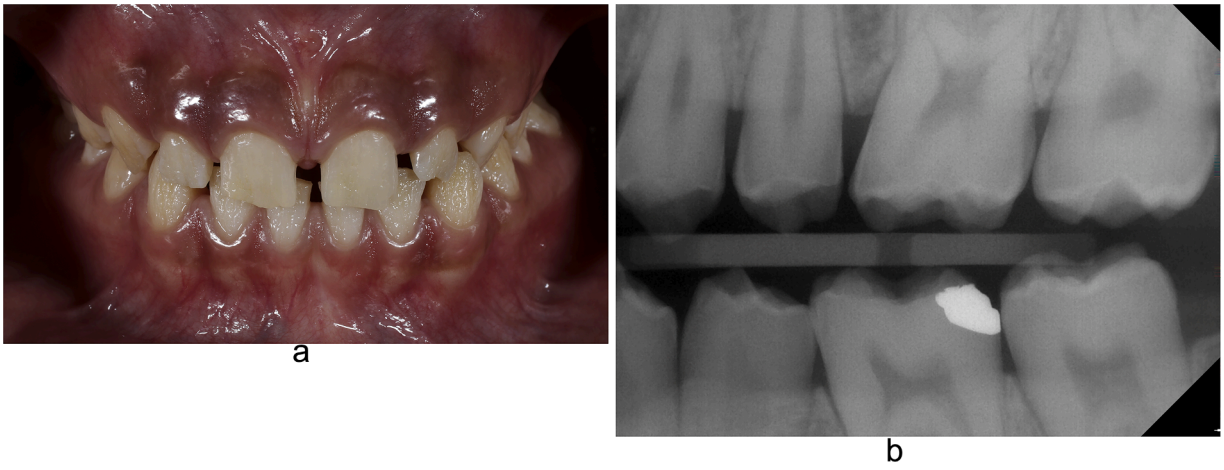

Fig. 2. A 17-year old patient with hypoplastic (type 1) amelogenesis imperfecta.
(a) Intra-oral frontal view. Generalized pitting and diastemas noted. (b) Radiograph exhibiting a generalized decrease in enamel thickness.

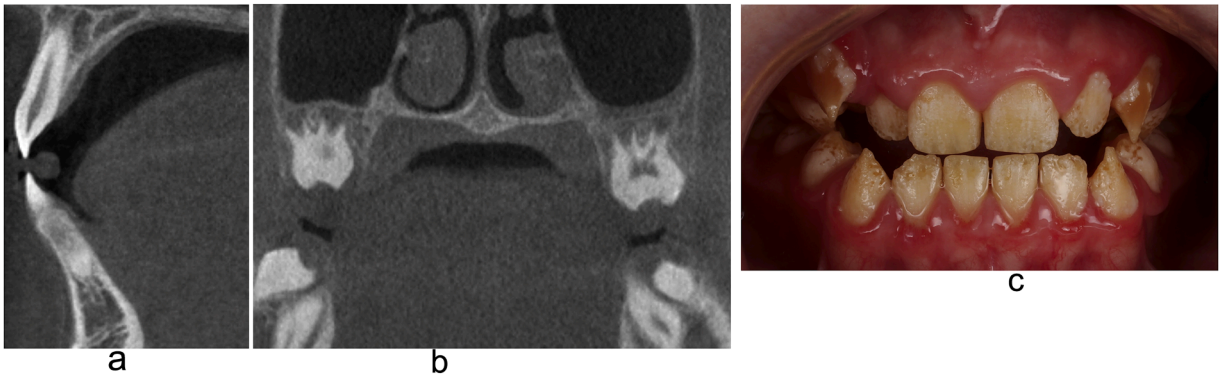

Fig. 3. A 14-year old patient with hypoplastic (type 1) amelogenesis imperfecta.
(a) Intra-oral view. Pitting, rough surface texture, and areas of exposed dentin noted. (b) CBCT slice of #8 exhibiting a generalized decrease in enamel thickness. (c) CBCT slice of unerupted #1,16 with a decrease in enamel thickness, confirming type 1 AI.

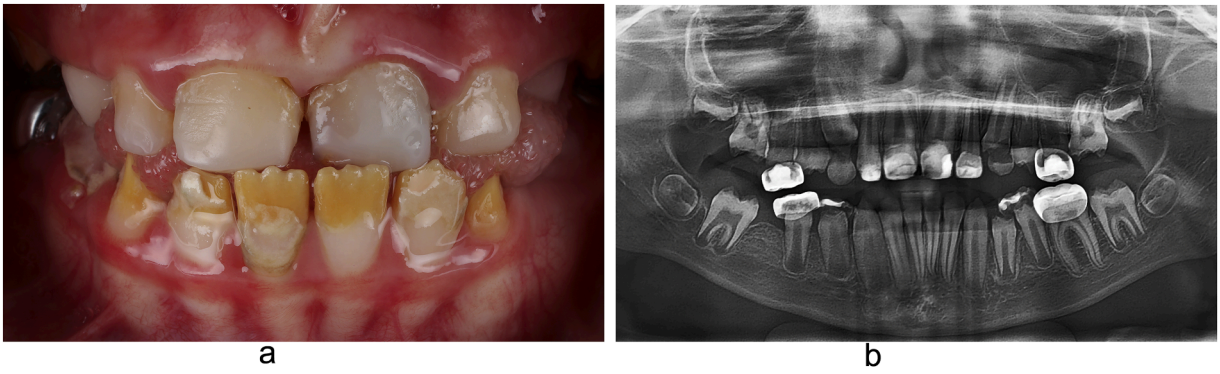

Fig. 4. An 11-year old patient with hypoplastic (type 1) amelogenesis imperfecta.
(a) Intra-oral view. Dentin cores exposed in the mandible with little enamel remaining and crossbite in the anterior noted. Without a clinical and radiographic examination, the phenotypic presentation here could be interpreted as type 3 AI. (b) Panoramic radiograph exhibiting a generalized decrease in enamel thickness on both erupted and unerupted teeth, confirming type 1 AI.

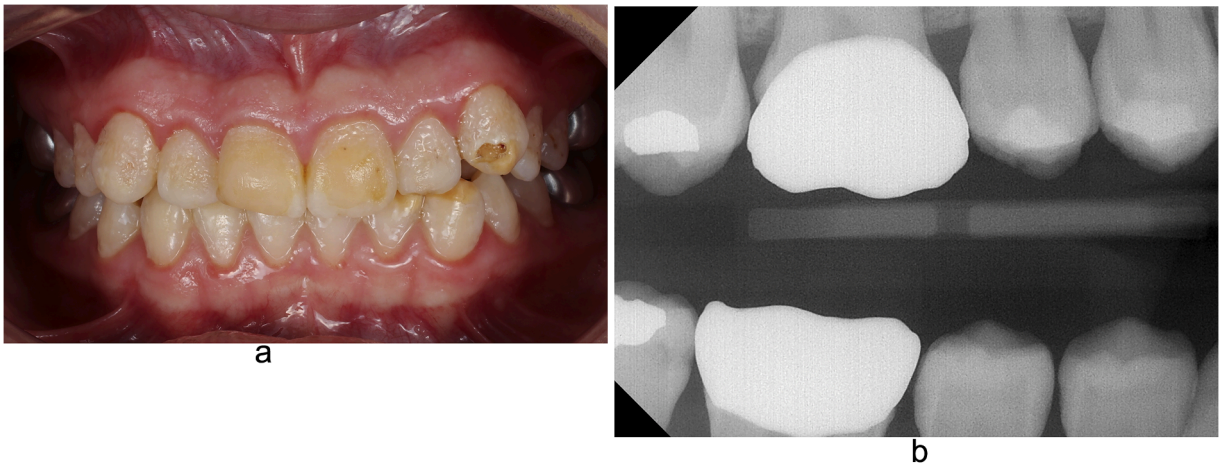

Fig. 5. A 16-year old patient with hypoplastic (type 1) amelogenesis imperfecta.
(a) Intra-oral view. Areas of rough surface texture and pitting noted, with some areas of smooth surface texture noted in the anterior mandible. (b) Radiograph exhibiting a generalized normal thickness of enamel, however a quantitative loss of enamel from pitting as seen on #4,5 is noted.

extracellular protein has a defect, which causes a higher matrix retention and decreased mineral deposition [1,2]. Unlike the hypoplastic quantitative alternation, hypomature enamel results in a qualitative defect. The enamel thickness is normal (Fig. 10b), but is usually slightly softer and tends to flake or chip off easily [1,13]. Visually, the enamel lacks translucency and has an opaque mottled appearance with white to brown discoloration, sometimes referred to as snow-capped teeth and mistaken for fluorosis [1,16,18]. Radiographically, the enamel may have a similar radiodensity to the dentin [15].

Hypocalcified (type 3) AI (Figs. 11–12) is caused by a defect in the initial nucleation of enamel crystallites [1], resulting in a decreased calcified matrix [14]. This results in an extreme qualitative alteration of the enamel. Upon eruption the enamel thickness is normal (Fig. 12b), but due to insufficient mineral content the enamel is extremely soft and wears away very rapidly [1] (Fig. 12c). These patients will often only have dentin cores remaining before adulthood (Fig. 11a) and severe wear [1,15]. Radiographically, there is less contrast between the enamel and dentin [13,15] (Fig. 11b). When little enamel remains on the teeth, it can be hard to clinically distinguish between type 1 (Fig. 4a) and 3 (Fig. 11a) AI. Tooth buds or unerupted teeth like third molars may give insight as to whether a normal thickness of enamel was present upon eruption. If the enamel thickness of a tooth bud or unerupted teeth is normal, then it is likely hypocalcified (type 3) AI (Fig. 11b), whereas if the enamel thickness is reduced, then it is likely hypoplastic (type 1) AI (Fig. 3c).

Hypomaturation-hypoplastic with taurodontism (type 4) AI (Figs. 13 and 18a) shows areas of hypoplastic enamel with areas of hypomatured enamel that is mottled with a white, yellow, or brown appearance [15, 19–21]. Enlarged pulp chambers may be observed on some of the teeth and the molars will have a taurodontic shape [15]. Radiographically, the enamel may have a similar or slightly increased radiodensity than the dentin [15].

2.2. *Dental/craniofacial anomalies*

Patients with AI can sometimes exhibit various abnormalities such as delayed eruption, impacted teeth, follicular cysts, taurodontism, congenitally missing teeth, pulp stones, enlarged pump chambers, crown resorption, and tooth agenesis [22]. Craniofacial anomalies may also occur more frequently in patients with AI [1], such as a more vertical craniofacial growth pattern, which leads to an increased intermaxillary angle, decreased overbite, and retrognathic mandible with a Class II skeletal pattern [23]. Malocclusion, alterations in vertical dimension [24], and a higher frequency of anterior open bite has also been observed [23–29]. Skeletal morphology may also requiring multidisciplinary care requiring orthodontic intervention and orthognathic surgery [2,30,31].

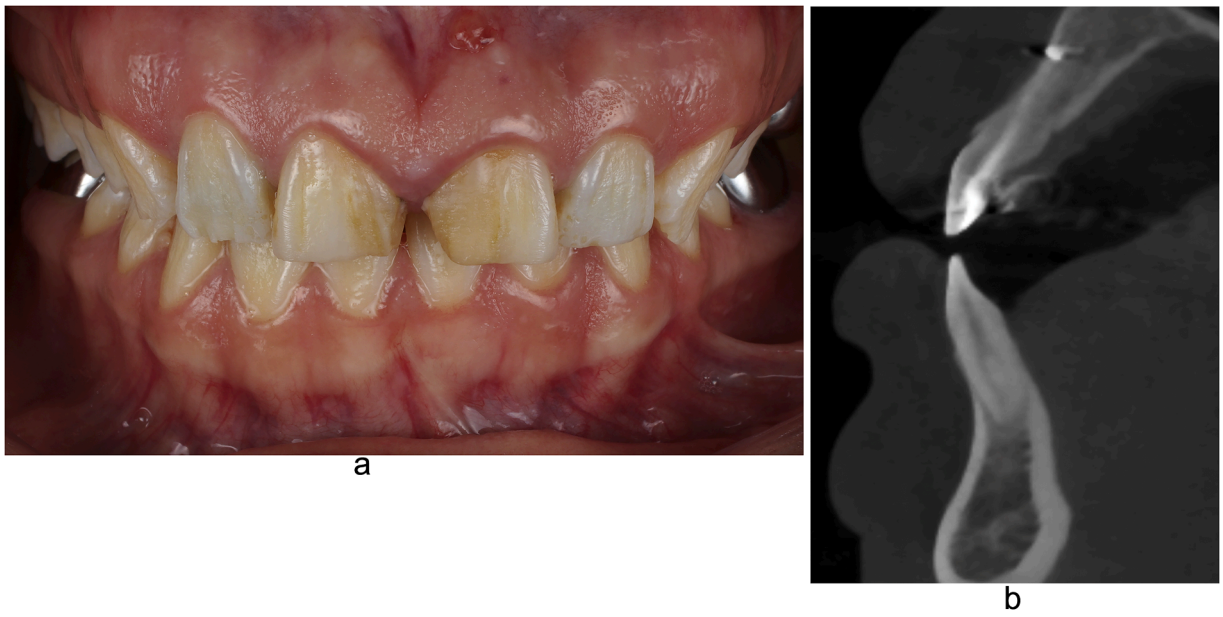

Fig. 6. A 23-year old patient with hypoplastic (type 1) amelogenesis imperfecta. Genetic testing revealed a pathogenic variant in the *ENAM* gene, confirming autosomal dominant amelogenesis imperfecta type 1B
(a) Intra-oral view. Irregular surface texture, linear depressions, and localized areas of pitting observed. (b) CBCT slice of the anterior teeth exhibiting a generalized decrease in enamel thickness.

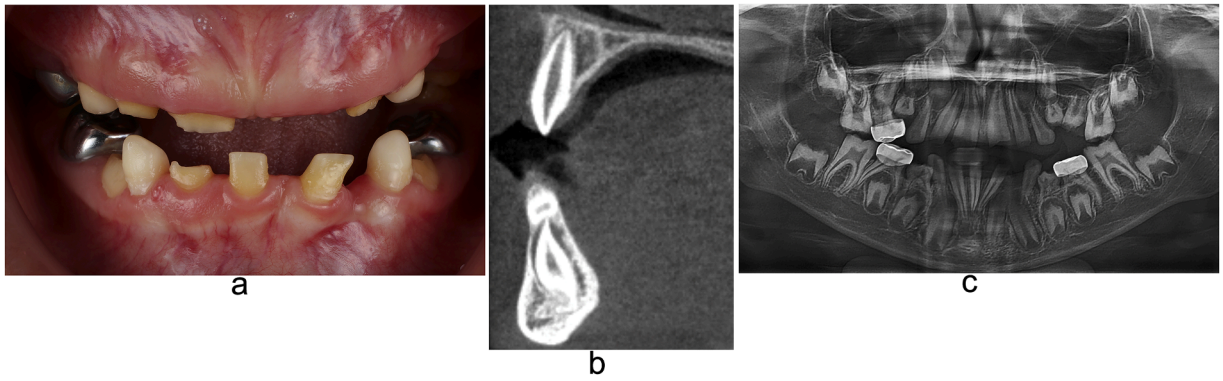

Fig. 7. An 11-year old patient with hypoplastic (type 1) amelogenesis imperfecta. Genetic testing revealed a pathogenic variant in the *FAM20A* gene, confirming autosomal recessive amelogenesis imperfecta type 1 G (Enamel-renal syndrome).
(a) Intra-oral view. Gingival hyperplasia, anterior open bite, and delayed tooth eruption noted. (b) CBCT slice of #9 exhibiting a generalized decrease in enamel thickness. (c) Panoramic radiograph exhibiting a generalized decrease in enamel thickness on both erupted and unerupted teeth, confirming type 1 AI.

2.3. Syndromic amelogenesis imperfecta

Although AI can be seen in isolation, it can also be found in syndromes [7]. Syndromic manifestations and symptoms may help with not only an overall clinical diagnosis, but also a genetic diagnosis that further improves the patients overall well-being and management [7]. Clinical signs within the head and neck area, immune deficits, skeletal defects, cardiovascular defects, neurological issues, genitourinary defects, and abnormal development of other structures of ectodermal origin such as the hair, skin and nails have been associated with syndromic AI [7].

Clinical signs associated with the head such as micrognathia and retrognathia are seen in Loeys-Dietz syndrome 2, while cone-rod dystrophy is seen in Jalili syndrome [7]. Heimler syndrome displays sensorineaural hearing loss [7] while gingival hyperplasia is a hallmark of Enamel-renal syndrome (Fig. 7a) [7]. Symptoms of the skin such as localized cutaneous deposits of superficial fat are seen with Focal dermal hypoplasia [7], while common features of Tricho-dento-osseous (TDO) syndrome (Fig. 13) are taurodontism, hypoplastic AI (Fig. 18a), abnormal bone density, curly hair, and dysplastic nails [7]. Genetic counseling for patients with AI is strongly recommended, especially when other dental and non-dental features found in syndromic AI are seen. Furthermore, patients with syndromic AI require an interdisciplinary team of not only dentists, but physicians as well [7].

2.4. Clinical implications

Although AI affects the enamel, non-enamel manifestations can also be seen. Patients with AI often experience mild to extreme tooth sensitivity depending on the severity of the AI [20]. Sensitivity is observed not only during oral hygiene habits, but also during mastication and drinking [14]. With an inability to properly clean the mouth, gingival hyperplasia, inflammation, or periodontal disease may also be found [22]. Excessive calculus formation has also been reported, which may be due in part to a rough enamel surface, altered salivary composition and flow, and poor oral hygiene secondary to hypersensitivity [22,32].

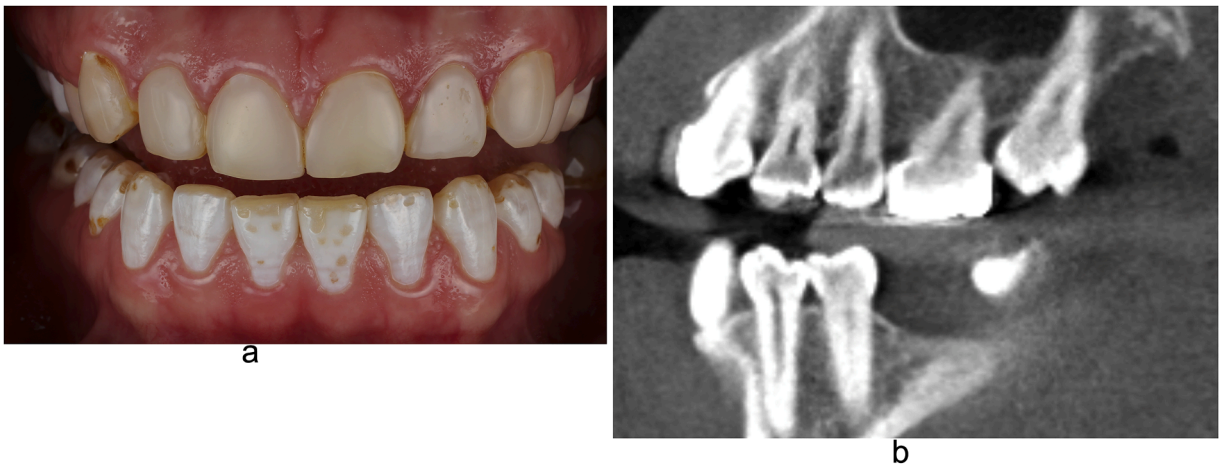

Fig. 8. A 23-year old patient with hypomaturation (type 2) amelogenesis imperfecta.
(a) Intra-oral view. White opaque appearance with a lack of translucency noted on the unrestored mandibular teeth. Incisal wear and loss of defective enamel due to a qualitative alteration of the enamel observed on the incisal and occlusal surfaces. Spotting and staining observed on the facial surface in round areas of exposed dentin. (b) CBCT slice exhibiting a normal thickness of the enamel.

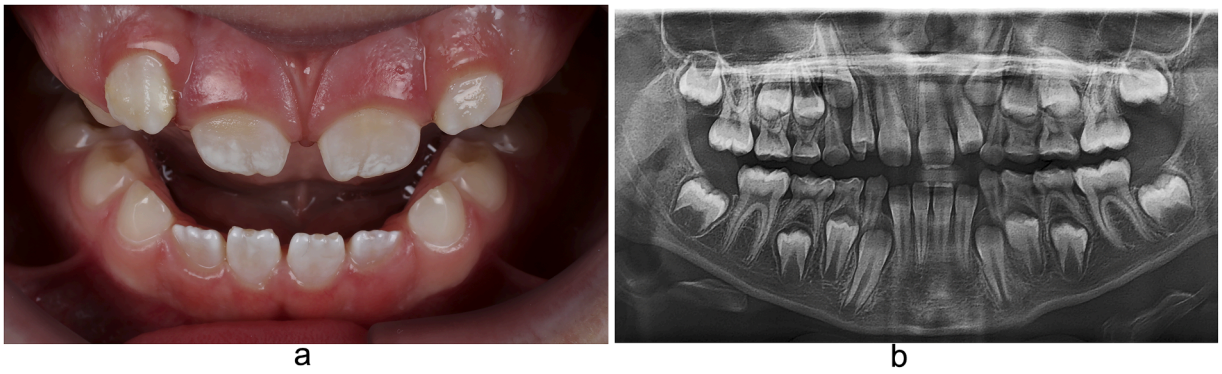

Fig. 9. A 8-year old patient with hypomaturation (type 2) amelogenesis imperfecta.
(a) Intra-oral view. White opaque appearance with a lack of translucency on the partially erupted permanent teeth noted. Due to a qualitative alteration of the enamel, areas of the defective enamel have worn or been abraded away. (b) Panoramic radiograph exhibiting a normal thickness of enamel on the erupted and non-erupted adult dentition. Thin enamel noted on the primary teeth from abrasion of the defective enamel over time.

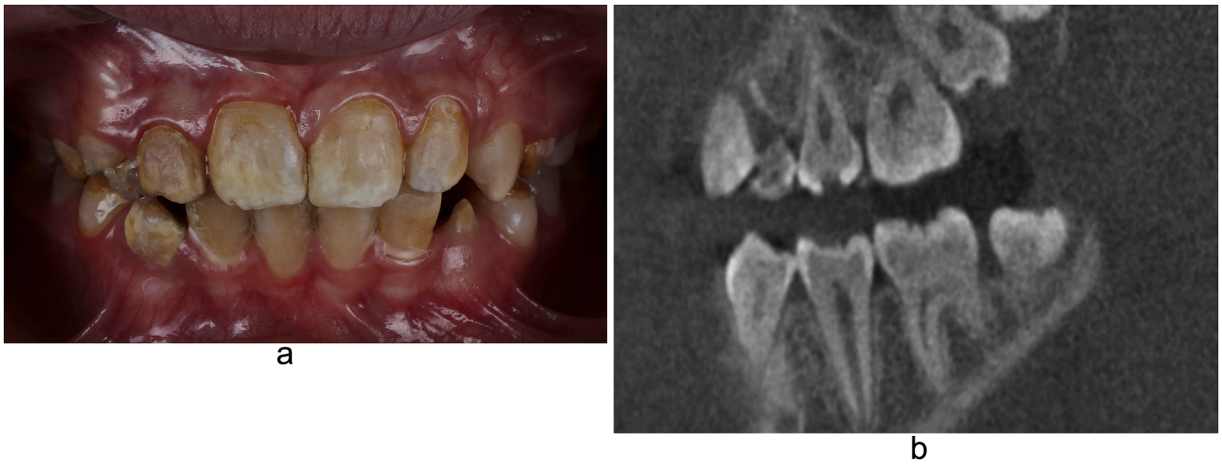

Fig. 10. A 15-year old patient with hypomaturation (type 2) amelogenesis imperfecta.
(a) Intra-oral view. Generalized white, yellow, brown opaque appearance of the teeth with a loss of translucency. Exposed dentin in the posterior where defective enamel has worn or broken off due to a qualitative alteration in the enamel. (b) CBCT slice exhibiting a normal thickness of enamel.

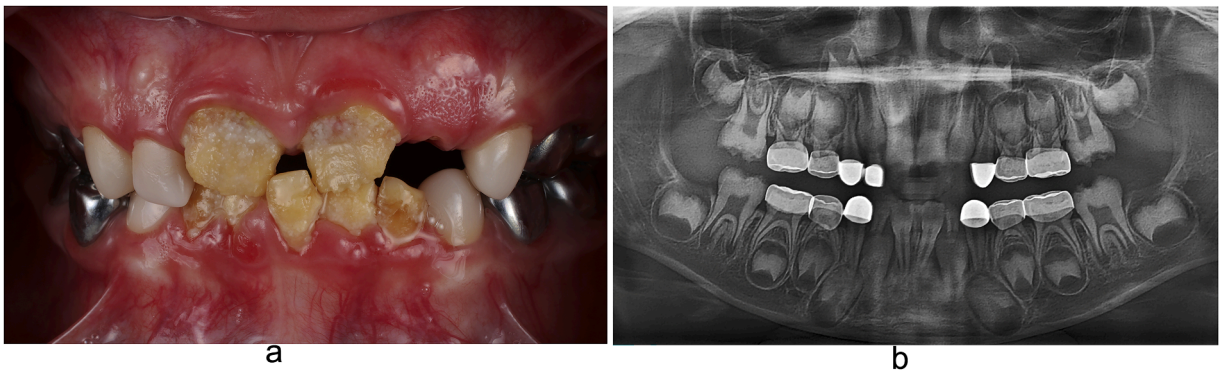

Fig. 11. An 8-year old patient with hypocalcified (type 3) amelogenesis imperfecta. Genetic testing revealed a pathogenic variant in the *FAM83H* gene, confirming autosomal dominant amelogenesis imperfecta type IIIA.
(a) Intra-oral view. Exposed dentin cores with minimal enamel remaining on the unrestored teeth is noted. Defective enamel is highly susceptible to post-eruptive breakdown due to a significant qualitative alteration of the enamel. Without a clinical and radiographic examination, the phenotypic presentation here could be interpreted as type 1 AI. (b) Panoramic radiograph exhibiting a normal thickness of enamel on the toothbuds, confirming type 3 AI. A decreased contrast and difficulty differentiating the dentin-enamel junction is noted.

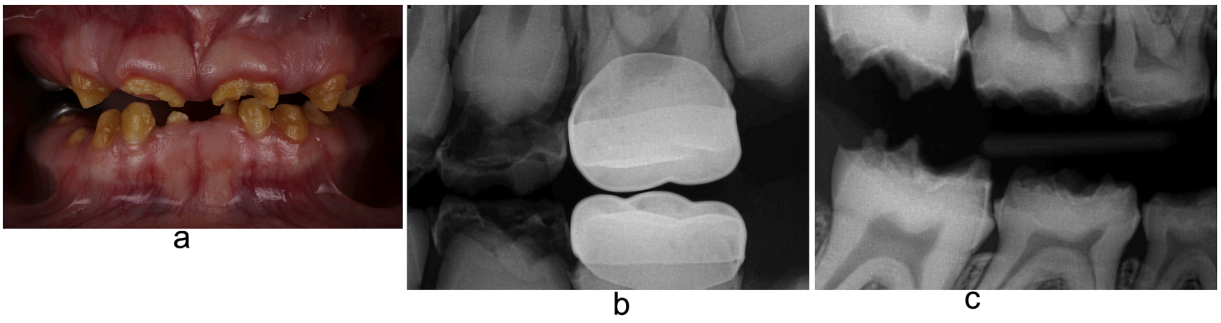

Fig. 12. A 14-year old patient with hypocalcified (type 3) amelogenesis imperfecta.
(a) Intra-oral view. Severe wear with minimal enamel remaining and gneralized exposed dentin. (b) Radiograph exhibiting a normal thickness of enamel on the adult teeth prior to eruption. (c) Radiograph exhibiting minimal enamel remaining and severe breakdown and loss of the defective enamel on both the primary and adult first molars.

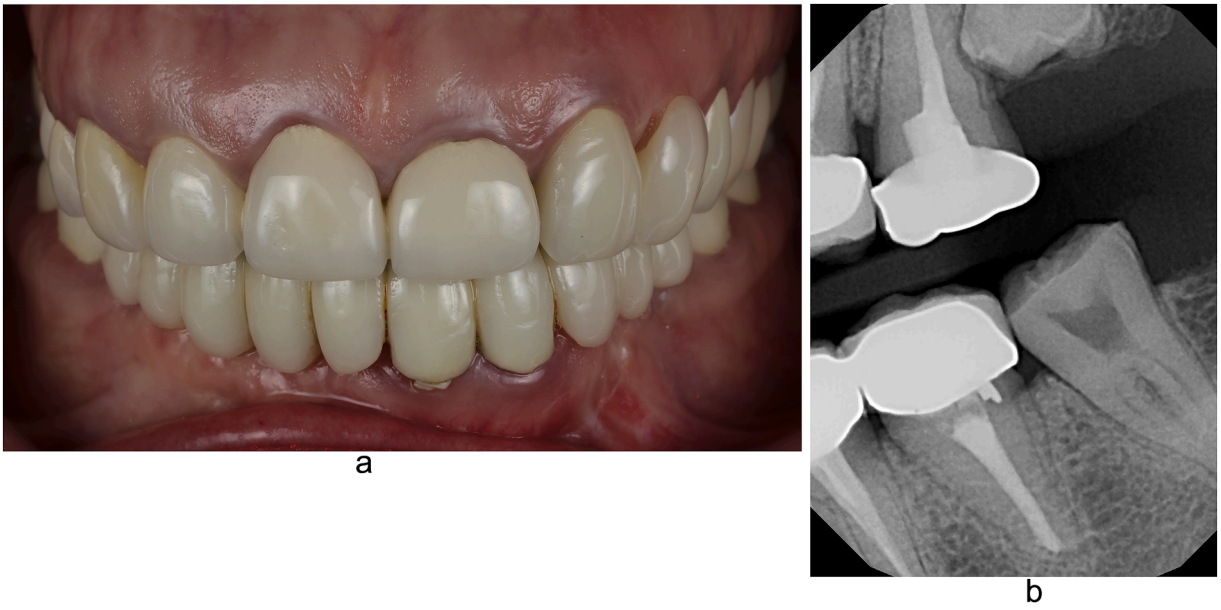

Fig. 13. A 53-year old patient with hypomaturation-hypoplastic with taurodontism (type 4) amelogenesis imperfecta. Genetic testing revealed a pathogenic variant in the *DLX3* gene, confirming autosomal dominant amelogenesis imperfecta type IV and autosomal dominant tricho-dento-osseous (TDO) syndrome.
(a) Intra-oral view. Previously completed full mouth rehabilitation with failing restorations noted. (b) Radiograph exhibiting a decrease in enamel thickness on unerupted #16.

Smaller crown sizes, shortened clinical crowns, loss of proximal contacts, malocclusion, attrition, and subsequent loss of vertical dimension of occlusion (VDO) has also been observed [22,29].

Compromised esthetics also often occurs in patients with AI. The clinical phenotypic presentation of AI varies greatly, as the teeth may look completely normal, or have anywhere from a mild to severe discoloration and breakdown of the dentition [11,14,15]. Comprehensive restorative care is often overlooked or not provided for younger patients with AI, which often leads to further structural loss of the teeth [33] and subsequently a heightened dental anxiety that may affect patients for the rest of their lives [11].

2.5. Psychosocial impact/OHRQOL

Patients with AI often experience an overall lower quality of life with significant physical, functional, psychological, and social impairments [34]. Increased sensitivity and visible disfigurement of the teeth greatly affects psychological health and presents many social challenges [34]. Social anxiety, poor self-esteem, and teasing can be especially challenging for younger patients with AI [35]. Childhood and adolescence is an impressionable, emotionally sensitive time for most children and as children grow older they become more aware of appearances, as do their peers. In a retrospective study, Lindunger et al. reported that half of patients with AI wished that they had their prosthetic reconstruction done before the age of 16 [36]. This highlights the importance of early diagnosis and intervention as it may improve the psychosocial well-being of patients with AI, especially during the adolescent age [33].

Parents and family members of patients with AI also face challenges [37]. Parents experience higher stress levels, guilt and shame for passing on a hereditary disorder to their child, fear of insufficient pain management, and fear of their child being bullied [37]. Parents may also share the same frustrations as a patient with AI, reporting a lack of knowledge, proper diagnosis, and inability to meet unique treatment needs [37]. Along with psychological distress, comes the added treatment financial burden [37,38], along with other costs such as traveling, and absence from work or school in order to attend appointments [37,38].

3. Dental management

3.1. General goals and considerations

The needs of a patient with AI will vary from the average dental patient. It is important to evaluate the initial and present needs of the patient while not overlooking and planning for an overall comprehensive plan that considers future treatment possibilities and outcomes [11]. Establishing good rapport with both children and parents/caregivers at a young age helps to create positive initial experiences and ensure the patient is not deterred from pursuing dental treatment for the remainder of their life [11,39]. In addition, earlier and timely intervention is imperative to address the sensitivity and poor esthetics experienced to try and spare the patient from potential psychosocial consequences, poor self-perception, and disfigurement [11,39].

General treatment goals are preservation of tooth structure, to maintain tooth vitality, decrease pain and sensitivity, and improve esthetics [33]. Treatment needs will vary depending on the stage of dental development, condition/breakdown of the existing dentition, skeletal growth, psychological well-being, patient maturity, readiness, and compliance [1]. The goals and needs of both the patient/parents must be balanced in order to devise appropriate treatment planning throughout different phases of life [1,40].

3.2. Phases of treatment

Establishment of comprehensive dental team as early as possible is imperative for long term management and treatment. At minimum, this dental team should include a pediatric dentist, orthodontist, and prosthodontist. Whether in the primary, mixed, or permanent dentition, patients with AI often require more extensive treatment coupled with heightened dental anxiety, which may require sedation or general anesthesia for their treatment needs [11].

During the primary dentition, treatment goals are to reduce sensitivity and pain, preventive care, establish favorable conditions for permanent tooth eruption and skeletal growth [33], and minimizing negative psychosocial consequences [11]. Routine periodic examinations and hygiene visits will help establish a baseline of needs and identify specific treatment needs as teeth continue to erupt [11]. Although difficult at this age, encouraging meticulous oral hygiene is imperative. Stainless steel crowns or glass ionomer restorations may be needed on the primary molars in order to prevent caries and further attrition of the enamel [33,39]. While in the anterior, direct composite resin, prefabricated crowns, or veneered crowns may improve sensitivity and esthetics [11,33,39].

In the mixed dentition, treatment goals are to maintain tooth vitality, preserve tooth integrity, decrease sensitivity, and improve esthetics [33]. Upon eruption of the permanent first molars and anterior teeth, orthodontic and prosthodontic evaluation is imperative, even though definitive rehabilitation will not ensue until eruption of the permanent dentition is complete [33]. Stainless steel crowns are often placed on permanent first molars [39] and glass ionomers on the occlusal surface while waiting for full eruption may help protect the tooth from further damage until the entire crown is exposed and can proceed with a restoration [39]. As the permanent incisors erupt, composite veneers may improve the esthetics while reducing wear and sensitivity, however young patients and their caregivers must be aware that restorative margins may become visible with continued eruption of the teeth and gingival maturation, requiring additional treatment to order to maintain esthetics [39].

Orthodontic evaluation at a young age is also crucial to overall success and management of patients with AI. However, treating patients with AI orthodontically does come with challenges such as bonding to defective enamel, and whether the enamel can withstand forces that are applied during tooth movement, and removal of fixed appliances [33,41]. Clear aligner therapy for patients with AI [42] should also be considered as the aligners form a pseudo-seal over the defective enamel and/or exposed dentin which may reduce sensitivity and improve QOL during orthodontic therapy.

Traditional orthodontic treatment goals likely need modification as perfect occlusion is no longer the goal, but rather achieving adequate tooth positioning that facilitates final restorations which maximize proper function, esthetics, and stability [33,41]. Visualization of the final prosthetic plan and type of AI must also be considered during orthodontic therapy. Orthodontic treatment will often close the interdental spaces of the teeth [33], but, in cases with a decreased enamel thickness such as hypoplastic AI, if the teeth are all brought together, at the time of restorative care the crown size will inevitably be smaller, the crowns will have longer contacts, and more tooth structure may have to be prepped in order to create restorative room for materials [43]. With hypomature AI, the teeth have a normal enamel thickness and crown size, therefore if the spacing between the teeth are closed it will not affect the final restorative plan.

In the permanent dentition, treatment goals are to restore function at a proper VDO, satisfy esthetics, and eliminate tooth sensitivity [33]. Prosthodontic full mouth reconstruction can begin once the clinical crown height and gingival tissue has stabilized and is mature [33]. Gingival contouring and crown lengthening may be needed in cases with shortened clinical crown heights (Fig 1a) or extensive gingival hyperplasia [33] (Fig. 7a). Depending on the remaining structural condition of the teeth, whether due to caries or severe attrition, endodontic therapy or extractions may be needed [33]. As skeletal discrepancies and malocclusion is commonly observed in this patient population, orthodontic therapy is crucial for best possible rehabilitation outcomes [44],

in addition, severe cases may require orthognathic surgery [33]. The comprehensive treatment plan during the permanent dentition will vary depending on the specific type of AI, healthy remaining tooth structure, and psychosocial status and readiness for full mouth reconstruction. Although many patients with AI would have preferred to have their full mouth reconstruction before the age of 16 [36], it does not mean that every teenage patient with AI is mature enough and psychologically capable of taking on all that comes with prosthodontic rehabilitation.

4. Prosthodontic management

4.1. Prosthetic considerations

Throughout these patient's lives, clinicians face the challenges of improving/restoring the function, esthetics, and occlusal stability. It becomes simultaneously challenging to consider being as conservative with treatment as possible [45] but still restoring and maintaining the vitality of the patient's natural dentition while avoiding or delaying the need for extractions [45]. However, this may not always be possible, especially when patients have not received proper care starting at a young age.

Historically, extractions and removable prostheses such as complete or partial dentures were often made for patients with AI [45]. However, removable prosthetics, especially for younger patients can have a negative psychological effect on patients. With vast advances in materials, digital, and restorative techniques, removable prosthetics should no longer be considered the standard of care even when all of the teeth are deemed unrestorable, and in such instances, implant therapy should be considered [45]. For teeth that are deemed restorable, studies have shown the use various materials such as composite resin veneers, porcelain veneers, stainless steel crowns, and full coverage crowns to restore the teeth [33,45,46].

Particular care should be made into taking consideration of the specific type of AI, and the consequent defects affecting the dentition [33]. This becomes especially important during bonding and placement of direct restorations. With hypoplastic AI, the enamel will have an insufficient quantity but still be of acceptable quality for bonding [33]. However, for final restorations, the pitted enamel should be completely removed in order to eliminate sensitivity and not have a rough surface that may be susceptible to secondary caries. In cases of hypomaturation AI, the enamel is of poor quality and the defective enamel and restorative margin can become porous over time and is susceptible to chipping, flaking, and wear [33]. Therefore, all of the defective enamel should be removed prior to final placement of restorations [33]. With hypocalcified AI, which some may consider the weakest quality and most severe breakdown of enamel, bonding would be insufficient [33], therefore all of this defective enamel should also be removed prior to final placement of restorations. If restorative margins are left in the defective enamel, the enamel is likely to breakdown [33] ultimately leading to a failed restoration.

4.2. Fixed therapy

Typically, in the permanent dentition, fixed therapy with full coverage crowns is suggested in order to circumferentially seal and protect the teeth long-term, while establishing a functional occlusion at an acceptable VDO that also balances facial harmony and esthetics. Clinical research has shown that indirect restorations in patients with AI have predictable success rates and longevity [13].Unlike direct restorations that rely on bonding, the type of AI does not appear to effect the longevity of full coverage indirect restorations [13,36,43]. With fixed crown rehabilitation, gingival inflammation and bleeding has been shown to significantly reduce [43]. This is likely due to the fact that patients are now able to properly brush, floss, and clean their teeth without hypersensitivity that occurs during oral hygiene when there is defective enamel and/or exposed dentin. Complications observed with full coverage restorations in patients with AI include fracture of the material, loss of cementation, caries, and endodontic therapy [36,45].

Metal ceramic crowns have been reported frequently in the literature to restore patients with AI [47,48], however, with advances in dental materials, the use of glass based all ceramic crowns has increased [49]. Minimal preparations can be made for these restorations, however even though these are conservative options, severe discoloration may be difficult to mask without more aggressive preparation [45]. High strength all ceramic restorations with zirconia or alumina [50] have also been used for the rehabilitation of patients with AI. The concern with these restorations is that they require removal of more tooth structure in their preparation, however, most rehabilitations of patients with AI require removal of most if not all defective enamel and already present with attrition and a decreased VDO.

4.3. Surgical therapy

In severe advanced cases of AI where the dentition is failing (Fig. 19a,b), implant therapy should be considered. With adult patients who do not receive proper dental care until they are in their third or fourth decade of life, the breakdown is often unsalvageable. Younger adults or adolescents may also have non-restorable teeth needing extraction. In these cases, the timing of implant placement is important. Clinicians must plan for continued skeletal growth that would result such things such as hyper-eruption of the adjacent natural dentition, esthetic complications, open proximal contacts, or implant restorations that are in infra-occlusion when placed too early [51]. In such cases, bone grafting should be done in order to preserve as much alveolar bone width as possible in order to avoid more invasive grafting later in life such as maxillary sinus or block grafting.

Due to high dental anxiety and increased sensitivity, patients with AI often undergo their treatment needs under sedation or general anesthesia [44,52]. It has been reported that treating teeth with molar incisor hypomineralization has been more difficult to achieve adequate anesthesia [53–55] and speculated that the porosity found in the hypomineralized enamel causes a constant subclinical level of inflammation in the pulpal cells, thus making it harder to anesthetize [53]. If this rationale is applied to patients with AI, difficulty anesthetizing these patients is expected (as the authors have found in their own clinical practice). Many patients with AI even opt to have dental cleanings done under sedation due dental anxiety and pain from the air, water, and difficulty anesthetizing.

4.4. Prognosis

Early diagnosis and proper management at a young age is vital for long term tooth vitality and successful rehabilitation. Most reported literature on patients with AI has been case reports and there are limited studies present with long-term follow up of full mouth rehabilitation in patients with AI. However, a retrospective study did show that restorations performed well and patients had positive experiences regarding their prosthodontic rehabilitation [36]. The data does suggest that indirect restorations show more longevity and predictability compared to direct restorations [13].

Another aspect to consider when planning full mouth rehabilitation in patients with AI, especially younger adolescent patients, is the need for re-treatment later in life. Since there is likely extensive removal of the defective enamel and tooth structure in circumferential preparations [13], abutment teeth may be aggressively prepared the first time around. Therefore, when a rehabilitation needs to be re-done, there is a higher potential for pulpal complications or non-restorability. Ensuring that patients and their parents understand the risks and potential long term complications that come with re-treatment is imperative. This includes stressing the importance of oral hygiene throughout treatment in order to maintain restorations as patients with AI are also likely in a higher caries risk category and therefore close maintenance and

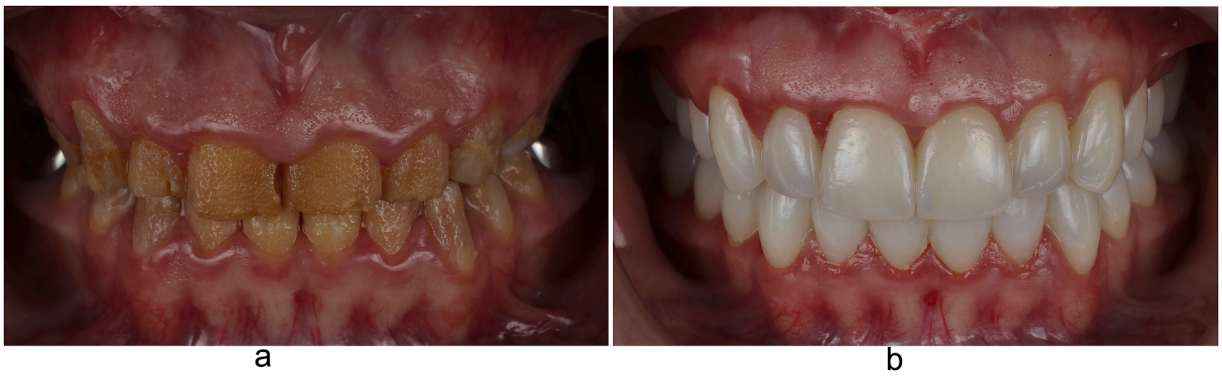

Fig. 14. Full mouth reconstruction on a 16-year old patient with hypoplastic (type 1) amelogenesis imperfecta.
(a) Intra-oral view, pre-operative presentation. (b) Intra-oral view, post-operative presentation.

follow-up is warranted.

More high level research with long-term follow up and prognosis is needed for patients with AI. However, with proper diagnosis and management starting at a young age, patients with AI can be successfully restored and have vast improvements in their quality of life. Life-long maintenance and close follow-up is needed on patients with AI throughout all phases of treatment from infancy through adulthood.

5. Clinical and radiographic examples

5.1. Hypoplastic (type 1) amelogenesis imperfecta

Fig. 1. A 16-year old patient with hypoplastic (type 1) amelogenesis imperfecta.

(a) Intra-oral view. Generalized pitting with an orange/brown appearance and shortened clinical crown heights noted. (b) CBCT slice of #8 exhibiting a generalized decreased thickness of enamel.

Fig. 2. A 17-year old with hypoplastic (type 1) amelogenesis imperfecta.

(a) Intra-oral view. Generalized pitting and diastemas noted. (b) Radiograph exhibiting a generalized decrease in enamel thickness.

Fig. 3. A 14-year old patient with hypoplastic (type 1) amelogenesis imperfecta.

(a) Intra-oral view. Pitting, rough surface texture, and areas of exposed dentin noted. (b) CBCT slice of #8 exhibiting a generalized decrease in enamel thickness. (c) CBCT slice of unerupted #1, 16 with a decrease in enamel thickness, confirming type 1 AI.

Fig. 4. An 11-year old patient with hypoplastic (type 1) amelogenesis imperfecta.

(a) Intra-oral view. Dentin cores exposed in the mandible with little enamel remaining and crossbite in the anterior noted. Without a clinical and radiographic examination, the phenotypic presentation here could be interpreted as type 3 AI. (b) Panoramic radiograph exhibiting a generalized decrease in enamel thickness on both erupted and unerupted teeth, confirming type 1 AI.

Fig. 5. A 16-year old with hypoplastic (type 1) amelogenesis imperfecta.

(a) Intra-oral view. Areas of rough surface texture and pitting noted, with some areas of smooth surface texture noted in the anterior mandible. (b) Radiograph exhibiting a generalized normal thickness of enamel, however a quantitative loss of enamel from pitting as seen on #4, 5 is noted.

Fig. 6. A 23-year old patient with hypoplastic (type 1) amelogenesis imperfecta. Genetic testing revealed a pathogenic variant in the *ENAM* gene, confirming autosomal dominant amelogenesis imperfecta type 1B.

(a) Intra-oral view. Irregular surface texture, linear depressions, and localized areas of pitting observed. (b) CBCT slive of the anterior teeth exhibiting a generalized decrease in enamel thickness.

Fig. 7. An 11-year old patient with hypoplastic (type 1) amelogenesis imperfecta. Genetic testing revealed a pathogenic variant in the *FAM20A* gene, confirming autosomal recessive amelogenesis imperfecta type 1G (Enamel-renal syndrome).

(a) Intra-oral view. Gingival hyperplasia, anterior open bite, and delayed tooth eruption noted. (b) CBCT slice of #9 exhibiting a generalized decrease in enamel thickness. (c) Panoramic radiograph exhibiting a generalized decrease in enamel thickness on both erupted and unerupted teeth, confirming type 1 AI.

5.2. Hypomaturation (type 2) amelogenesis imperfecta

Fig. 8. A 23-year old patient with hypomaturation (type 2) amelogenesis imperfecta.

(a) Intra-oral view. White opaque appearance with a lack of translucency noted on the unrestored mandibular teeth. Incisal wear and loss of defective enamel due to a qualitative alteration of the enamel observed on the incisal and occlusal surfaces. Spotting and staining observed on the facial surface in round areas of exposed dentin. (b) CBCT slice exhibiting a normal thickness of the enamel.

Fig. 9. An 8-year old patient with hypomaturation (type 2) amelogenesis imperfecta.

(a) Intra-oral view. White opaque appearance with a lack of translucency on the partially erupted permanent teeth noted. Due to a qualitative alteration of the enamel, areas of the defective enamel have worn or been abraded away. (b) Panoramic radiograph exhibiting a normal thickness of enamel on the erupted and non-erupted adult dentition. Thin enamel noted on the primary teeth from abrasion of the defective enamel over time.

Fig. 10. A 15-year old patient with hypomaturation (type 2) amelogenesis imperfecta.

(a) Intra-oral view. Generalized white, yellow, brown opaque appearance of the teeth with a loss of translucency. Exposed dentin in the posterior where defective enamel has worn or broken off due to a qualitative alteration in the enamel. (b) CBCT slice exhibiting a normal thickness of enamel.

5.3. Hypocalcified (type 3) amelogenesis imperfecta

Fig. 11. An 8-year old with hypocalcified (type 3) amelogenesis imperfecta. Genetic testing revealed a pathogenic variant in the *FAM83H* gene, confirming autosomal dominant amelogenesis imperfecta type IIIA.

(a) Intra-oral view. Exposed dentin cored with minimal enamel remaining on the unrestored teeth is noted. Defective enamel is highly susceptible to post-eruptive breakdown due to a significant qualitative alteration of the enamel. Without a clinical and radiographic examination, the phenotypic presentation here could be interpreted as type 1 AI.

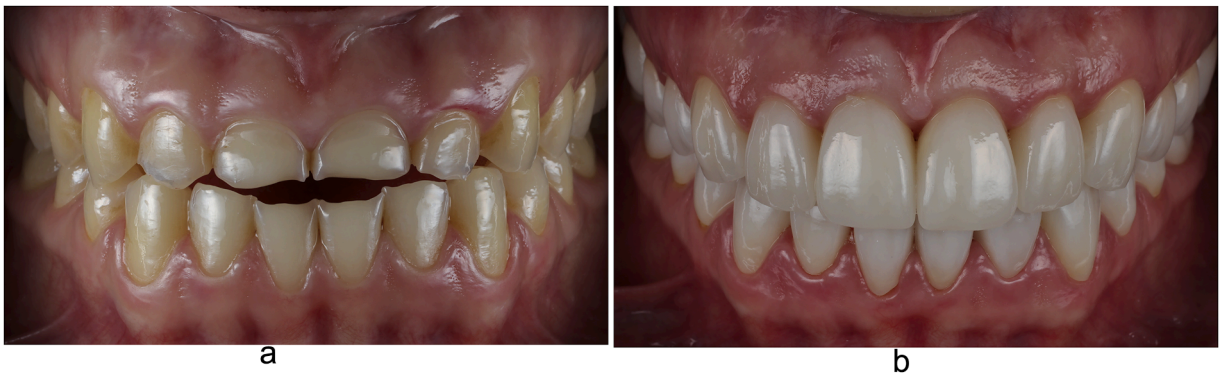

Fig. 15. Full mouth reconstruction on a 20-year old patient with hypoplastic (type 1) amelogenesis imperfecta.
(a) Intra-oral view, pre-operative presentation. (b) Intra-oral view, post-operative presentation.

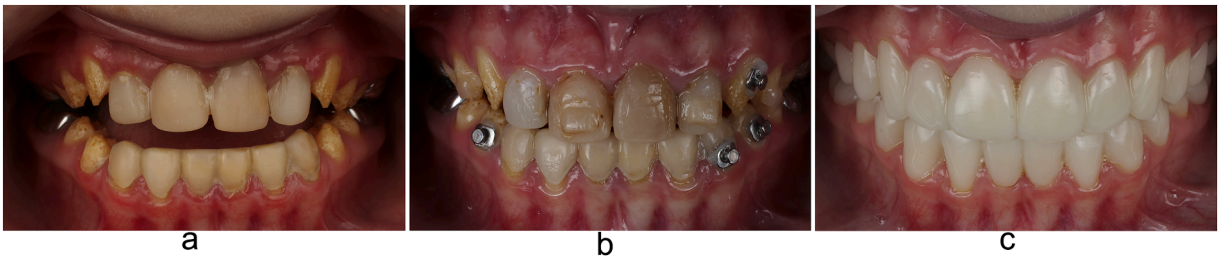

Fig. 16. Full mouth reconstruction on a 17-year old patient with hypoplastic (type 1) amelogenesis imperfecta, post-orthodontic therapy, mid-treatment.
(a) Intra-oral view, pre-operative presentation. (b) Intra-oral view, prior to orthodontic completion. (c) Intra-oral view, mid-treatment, provisionalization phase.

(b) Panoramic radiograph exhibiting a normal thickness of enamel on the toothbuds, confirming type 3 AI. A decreased contrast and difficulty differentiating the dentin-enamel junction is noted.

Fig. 12. A 14-year old patient with hypocalcified (type 3) amelogenesis imperfecta.

(a) Intra-oral view. Severe wear with minimal enamel remaining and generalized exposed dentin. (b) Radiograph exhibiting a normal thickness of enamel on the adult teeth prior to eruption. (c) Radiograph exhibiting minimal enamel remaining and severe breakdown and loss of the defective enamel on both the primary and adult first molars.

5.4. Hypomaturation-hypoplastic with taurodontism (type 4) amelogenesis imperfecta

Fig. 13. A 53-year old patient with hypomaturation-hypoplastic with taurodontism (type 4) amelogenesis imperfecta. Genetic testing revealed a pathogenic variant in the *DLX3* gene, confirming autosomal dominant amelogenesis imperfecta type IV and autosomal dominant tricho-dento-osseous (TDO) syndrome.

(a) Intra-oral view. Previously completed full mouth rehabilitation with failing restorations noted. (b) Radiograph exhibiting a decrease in enamel thickness on erupted #16.

6. Completed case examples

6.1. Fixed full mouth reconstruction

Fig. 14. Full mouth reconstruction on a 16-year old patient with hypoplastic (type 1) amelogenesis imperfecta.

(a) Intra-oral view, pre-operative presentation. (b) Intra-oral view, post-operative presentation.

Fig. 15. Full mouth reconstruction on a 20-year old patient with hypoplastic (type 1) amelogenesis imperfecta.

(a) Intra-oral view, pre-operative presentation. (b) Intra-oral view, post-operative presentation.

Fig. 16. Full mouth reconstruction on a 17-year old patient with

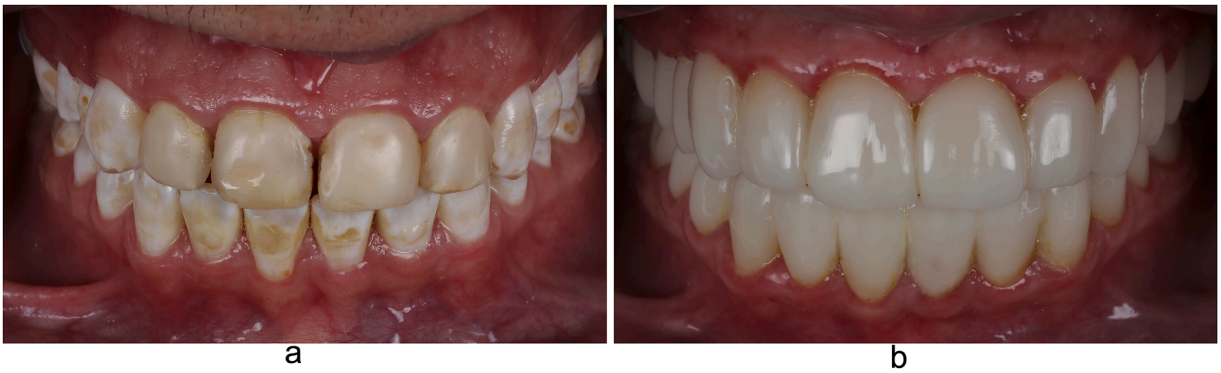

Fig. 17. Full mouth reconstruction on an 19-year old patient with hypomaturation (type 2) amelogenesis imperfecta, mid-treatment.
(a) Intra-oral view, pre-operative presentation. (b) Intra-oral view, mid-treatment, provisionalization phase.

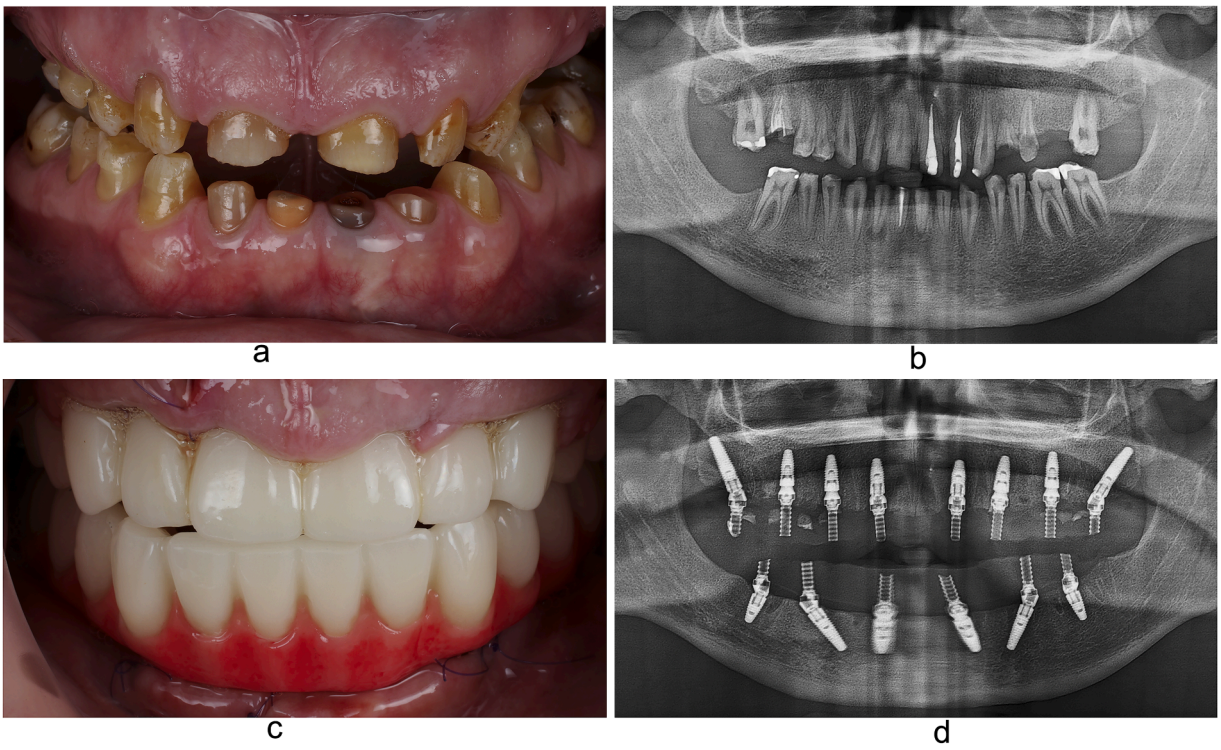

Fig. 18. Implant full mouth reconstruction on a 36-year old patient with hypomaturation-hypoplastic with taurodontism (type 4) amelogenesis imperfecta and tricho-dento-osseous (TDO) syndrome, mid-treatment.
(a) Intra-oral view, pre-operative presentation. (b) Panoramic radiograph, pre-operative presentation. (c) Intra-oral view, mid-treatment, provisionalization phase. (d) Panoramic radiograph, mid-treatment, provisionalization phase.

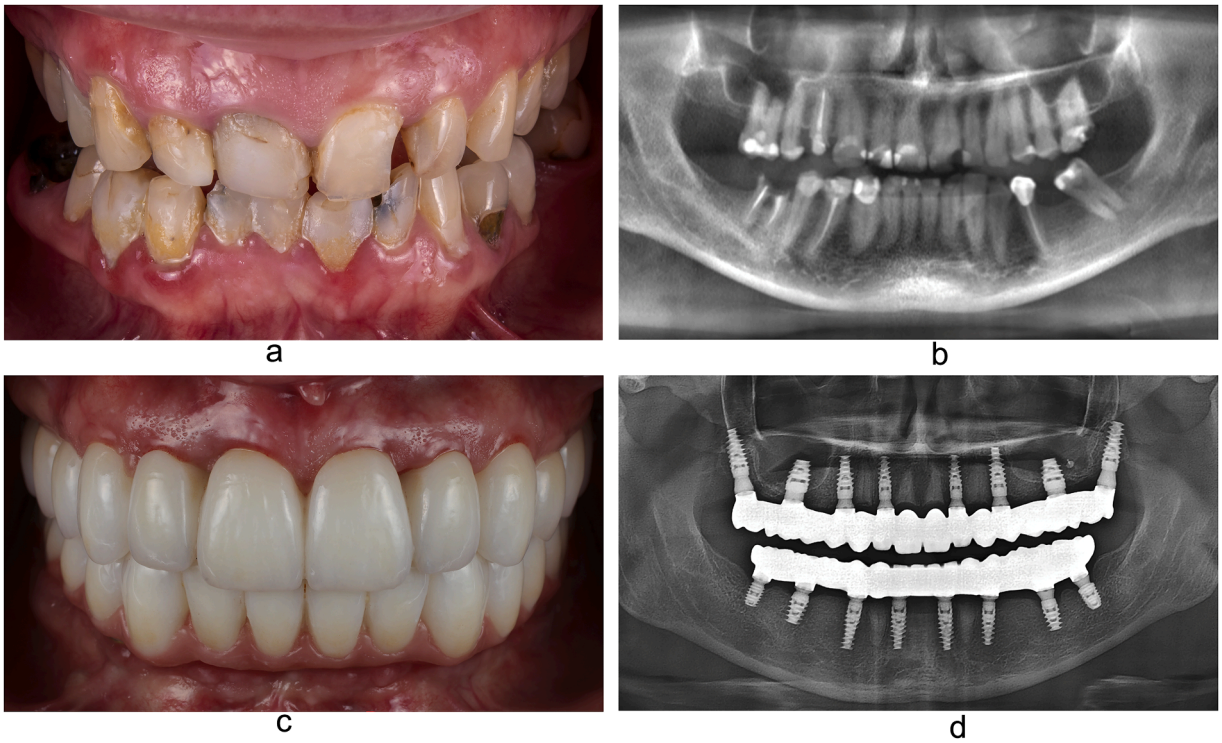

Fig. 19. Implant full mouth reconstruction on a 41-year old patient with hypoplastic (type 1) amelogenesis imperfecta.

hypoplastic (type 1) amelogenesis imperfecta, post-orthodontic therapy, mid-treatment.

(a) Intra-oral view, pre-operative presentation. (b) Intra-oral view, prior to orthodontic completion. (c) Intra-oral view, mid-treatment, provisionalization phase.

Fig. 17. Full mouth reconstruction on a 19-year old patient with

hypomaturation (type 2) amelogenesis imperfecta, mid-treatment.

(a) Intra-oral view, pre-operative presentation. (b) Intra-oral view, mid-treatment, provisionalization phase.

6.2. Implant full mouth reconstruction

Fig. 18. Implant full mouth reconstruction on a 36-year old patient with hypomaturation-hypoplastic with taurodontism (type 4) amelogenesis imperfecta and tricho-dento-osseous (TDO) syndrome, mid-treatment.

(a) Intra-oral view, pre-operative presentation. (b) Panoramic radiograph, pre-operative presentation. (c) Intra-oral view, mid-treatment, provisionalization phase. (d) Panoramic radiograph, mid-treatment, provisionalization phase.

Fig. 19. Implant full mouth reconstruction on a 41-year old patient with hypoplastic (type 1) amelogenesis imperfecta.

(a) Intra-oral view, pre-operative presentation. (b) Panoramic radiograph, pre-operative presentation. (c) Intra-oral view, post-operative presentation. (d) Panoramic radiograph, post-operative presentation.

7. Conclusion

Amelogenesis imperfecta is a rare genetic condition that affects normal enamel formation. Diagnosis of AI is difficult, often leading patients to go undiagnosed for many years, which can lead to further deterioration of the dentition. AI poses restorative challenges for providers but is further complicated by the psychosocial effects that AI can have on patients well-being and overall quality of life. Proper dental management starting at a young age and throughout all phases of life is essential in order to maximize patients dental needs and long-term prognosis. Further research on long term management and outcomes for patients with AI is needed in order to optimize evidence based, individualized, multidisciplinary treatment approaches for this patient population. Extremely close maintenance and follow-up care is warranted for patients with AI throughout all phases of life.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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