Enamel pretreatment with sodium hypochlorite to enhance bonding in hypocalcified amelogenesis imperfecta: case report and SEM analysis

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Abstract

Bonding composite resin to enamel of teeth affected by amelogenesis imperfecta (AI) is often problematic, especially in cases with poorly mineralized, friable enamel. Difficulty in bonding hypomineralized enamel can significantly limit the restorative and orthodontic treatment options for AI patients. In this report, we document a novel approach to bonding AI enamel by pretreating the tooth surface with 5% sodium hypochlorite (NaOCl), resulting in improved bonding of an orthodontic bracket to a previously impacted maxillary canine. (Pediatr Dent 16:433–36, 1994)

Introduction

Hypocalcified AI (HCAI) types are thought to result primarily from defects in nucleation and early enamel mineralization. However, later stages of enamel mineralization also may be abnormal.[1] The inheritance pattern for HCAI is reported as being autosomal dominant or recessive. The typical clinical features of affected enamel include a yellow to brown color and normal enamel thickness. Affected enamel may be variably located on the tooth. Cervical enamel frequently is less affected than more coronally located enamel.[2,3] Ultrastructurally, HCAI enamel has been shown to be more porous and have a lower mineral content per volume than normal enamel.[2] Differences in enamel protein content and composition have been demonstrated and could be diagnostic for the different AI types.[4–7] Certain types of AI can have an enamel protein content much greater than normal enamel. For example, HCAI enamel may have 3 to 4% protein by weight compared with 0.5% for normal enamel. There may be an association between higher protein content and more severely affected enamel.

It is believed that bonding composite resin by the acid etch technique to enamel affected by AI is more difficult than bonding to normal enamel (reviewed by Seow[8]). Sodium hypochlorite (NaOCl) is known to be an excellent protein denaturant that should be capable of removing excess enamel protein.[9] Thus, we predicted that pretreating AI enamel with sodium hypochlorite would make the enamel crystals more accessible to the etching solution, resulting in a clinically more favorable etched surface.

The purposes of this report were to: present a novel method for enhancing the bonding of an orthodontic bracket to a tooth affected with HCAI by pretreating the tooth for 1 min with 5% NaOCl, and examine the effect of 5% NaOCl on the surface topography of HCAI enamel by scanning electron microscopy.

Case report

An 11-year-old white female was referred to the Department of Pediatric Dentistry of the UNC School of Dentistry for treatment of "hypoplastic" teeth. Upon further evaluation, the patient was diagnosed as having hypocalcified amelogenesis imperfecta.

After completing interim restorative care, limited orthodontic treatment of the maxillary arch was undertaken due to an impacted maxillary left canine. After obtaining initial orthodontic alignment of the arch, the patient had a mucoperiosteal flap procedure to expose the tooth. In addition, the maxillary left second premolar was extracted. An orthodontic button was bonded (Transbond™, Unitek/3M, Monrovia, CA) near the incisal tip of the canine. The flap was sutured into a position apical to the exposed canine. A gold chain soldered to the orthodontic button was left exposed to enable delivery of extrusive force through an auxiliary wire (0.016x0.022-in. stainless steel) soldered to the base arch wire (0.018x0.025-in. stainless steel). The auxiliary wire rested in the mandibular vestibule in its passive state. The tooth was extruded over the next 6 months. After the facial surface of the canine was sufficiently exposed, the button and residual adhesive were removed, and a prophylaxis was performed using a rubber cup and a slurry of pumice in order to place a preangulated, pretorqued orthodontic bracket.

Several attempts made by two operators (RDV and JRC) to bond the bracket to the facial surface of the canine using composite resin (Transbond) and the acid etch technique failed despite rigorous efforts to maintain a dry field with cotton roll isolation. Several attempts to bond the bracket using glass ionomer cement (Ketac-Cem®, Espe-Premier, Norristown, PA) also were unsuccessful, so the patient returned the following month for rebonding. Neither dentin bonding agent (Gluma®, Miles Dental Products, South Bend, IN) plus composite resin nor glass ionomer cement yielded a

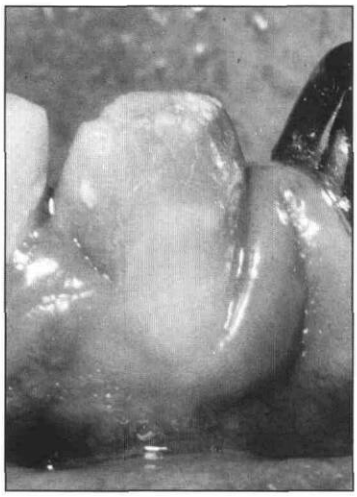

Fig 1. Mandibular left canine visibly affected by amelogenesis imperfecta and depicted in scanning electron micrographs (Figs 2–5).

bond that would remain intact for longer than 15 min after arch wire placement.

In laboratory studies of AI-affected enamel, NaOCl has been used to remove protein and to permit better analysis of enamel crystallite structure.[2] We hypothesized that using NaOCl to remove excess protein from the enamel would lead to a stronger bond. The canine was cleaned with pumice, rinsed with water spray, and carefully isolated with cotton rolls. A solution of 5% NaOCl was applied liberally with a brush for 1 min, and the tooth was rinsed with water spray. After air drying, 37% phosphoric acid solution was applied to the tooth surface for 1 min. The tooth was rinsed and air dried again, and a thin layer of enamel bonding agent was applied to the etched enamel surface. The bracket, loaded with composite resin (Transbond), was positioned on the tooth, and excess resin was removed. The resin was light cured for 2 min. The resulting bond was immediately subjected to normal orthodontic forces and was successful for the remainder of the orthodontic treatment.

Scanning electron microscopy procedure

The improved bonding after NaOCl pretreatment of HCAI enamel led us to investigate possible reasons for this success. There was no apparent difference in surface texture discernible to the naked eye due to NaOCl pretreatment plus acid etching compared with acid etching alone. However, it seemed likely that NaOCl followed by acid etching produced an ultrastructural topography more conducive to bonding.

The mandibular canines remained unrestored during the course of orthodontic treatment, so the more severely affected mandibular left canine was selected for SEM analysis (Fig 1). The labial surface was cleaned with a rubber cup and slurry of pumice, rinsed with water, and air dried. A baseline silicone impression (Silene®, Harry J. Bosworth, Skokie, IL) of the unetched tooth was obtained using the manufacturer's protocol and cotton roll isolation. Upon removing the polymerized impression, cotton roll isolation was maintained to prevent any salivary contamination of the tooth surface. The tooth was acid-etched for 1 min, rinsed, and dried. A second impression was made as described above. The silicone impressions were poured in epoxy resin, and casts were analyzed by SEM using standard techniques.[10] Analysis of these casts yielded control data on the topographic changes due to acid etch alone.

Several months passed to allow the etched tooth to remineralize before initial prophylaxis and baseline impression procedures were repeated exactly as above for the unetched tooth. The tooth surface was treated with 5% NaOCl applied with a brush for 1 min, rinsed, and dried. A second impression was obtained. Finally, the tooth was acid etched, rinsed and dried as described above, and a third impression was obtained. The casts of these impressions were processed and analyzed in the same manner as the control casts from the first appointment. Representative photomicrographs are depicted in Figs 2–5.

Results

The photomicrographs obtained from replicas of the unetched tooth at two different appointments (Figs 2A, B) confirmed that the period of remineralization was sufficient to ensure that the etched tooth regained a relatively normal SEM appearance. Thus, it seemed reasonable to compare photomicrographs of casts obtained during either appointment.

Acid etching alone created a sparse etch pattern separated by large areas exhibiting no etched appearance (Fig 3). These nonetched areas appeared to be coated with an amorphous surface layer. It is possible that this amorphous coating was protein adsorbed from saliva. It should be noted that the cervical enamel, which is less severely affected by AI, appears to exhibit a more normal acid etch pattern.

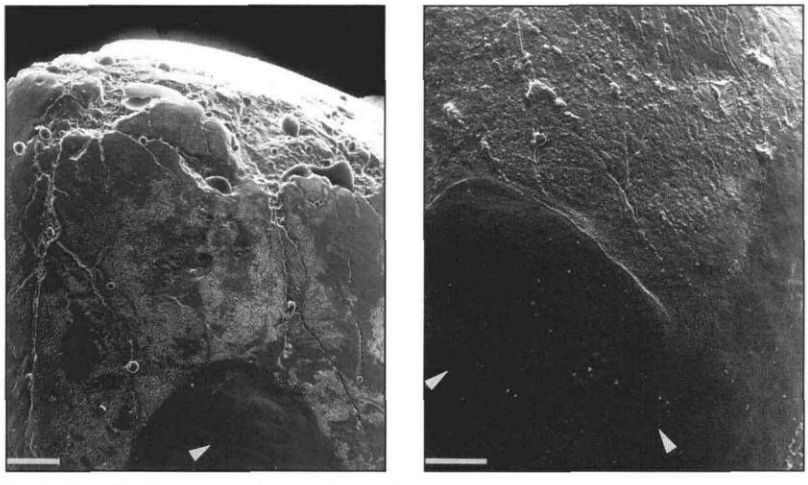

Fig 2. Mandibular left canine replicas of: A) unetched tooth, initial appointment, and B) unetched tooth, second appointment. Less affected enamel is located cervically (arrowheads). Scale bars represent 500 µm.

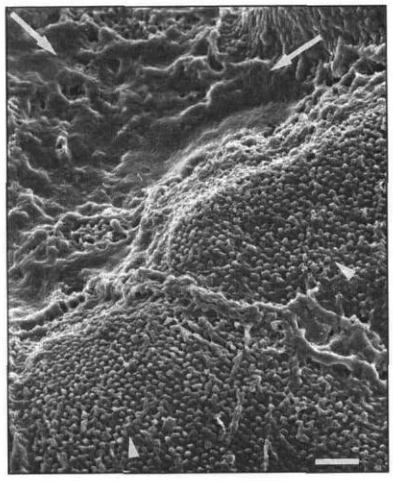

Fig 3. Mandibular left canine replica after acid etch alone. Note well-etched enamel (arrowheads) and amorphous, poorly etched areas (arrows). Scale bar represents 50 μm.

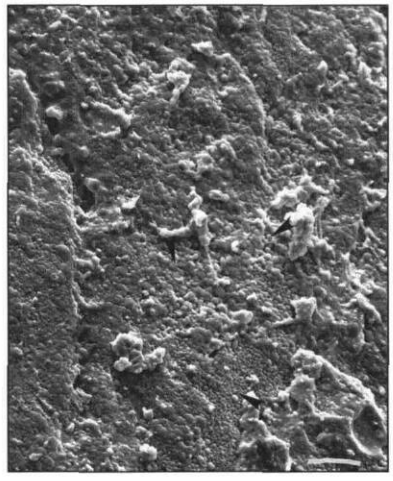

Fig 4. Mandibular left canine replica after 5% NaOCl alone. Note the globular surface pattern (arrowheads). Scale bar represents 50 μm.

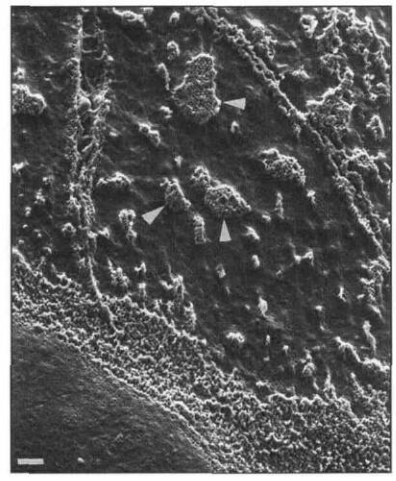

Fig 5. Mandibular left canine replica after 5% NaOCl plus acid etch. Note the raised islands of well-etched enamel (arrowheads). Scale bar represents 50 μm.

NaOCl pretreatment appeared to remove the amorphous surface material revealing a globular pattern (Fig 4). These globular structures could represent blunted prism ends, ectopic surface mineralizations, or surface deposits of calculus. Given the morphologic variability of these surface features, they likely represent several diverse structures.

Acid etching that followed NaOCl pretreatment produced islands of well-etched enamel apparently surrounded by shallow, depressed areas with featureless to slightly etched bases (Fig 5). There appeared to be preferential etching of the periphery of enamel prisms.

Low-magnification photomicrographs suggested that NaOCl pretreatment resulted in more etched surface area interspersed with smaller nonetched areas (data not shown). However, we undertook no morphometric analysis. Therefore, we cannot conclude definitively that NaOCl pretreatment increased etched surface area.

Discussion

NaOCl is an effective protein denaturant that does not appear to alter the structure or mineral content of normal or HCAI enamel crystallites.[2] SEM data suggest that NaOCl enhanced bonding in this case by removing excess protein, which interfered with establishing a clinically successful acid etch pattern. The excess protein may have been at least partially of salivary origin, since bond failures occurred only after the tooth had been exposed to the oral environment. Imbibition studies show that HCAI enamel can be more porous, and ultrastructural analyses show it has rougher crystallites than normal enamel.[2] Furthermore, HCAI enamel can have a markedly elevated protein content due to protein retention during development. In this case these factors apparently interfered with the development of a typical etch pattern using 37% phosphoric acid. NaOCl likely produced a more favorable acid etch by exposing the enamel mineral previously encased in acid-insoluble proteins.

One must exercise caution in interpreting the photomicrographs depicted in this report based on a single patient. In addition, no morphometric analysis of the photomicrographs was undertaken. Yet, the NaOCl pretreatment technique produced clinical success in bonding an orthodontic bracket to a tooth affected by HCAI.

We attempted several other methods of bonding an orthodontic bracket to the tooth in question and all resulted in failure. Still, other treatment options existed in the event of failure of NaOCl pretreatment in establishing a clinically successful bond. An orthodontic band could have been cemented to the tooth. Alternatively, a full coverage restoration (resin crown or prefabricated stainless steel crown) could have been placed, relying partly on macromechanical retention rather than solely on bonding to enamel and/or dentin. It is true that this tooth, severely affected by AI, will require a full coverage restoration at a later date. However, only the facial surface of the tooth was readily accessible. Thus, either of the above options would have required a second surgical procedure to permit access to the entire lingual surface.

This technique of enamel pretreatment with 5% NaOCl has been attempted in bonding orthodontic brackets for two other AI patients with apparent clinical success. One could speculate also about broader applicability for this pretreatment technique. The success of bonding sealants and composite resin restorations to AI-affected enamel could be enhanced. Yet we

have reason to believe that the technique would be ineffective — or possibly detrimental — in certain situations. For example, some AI enamel has a normal protein content,[6] and NaOCl pretreatment would probably have no effect on its surface topography. On the other hand, hypomaturation AI (HMAI) enamel exhibits a very high protein content with small, disorganized enamel crystals.[9] It is possible that NaOCl pretreatment of HMAI enamel could result in excessive destruction of enamel due to removal of large quantities of protein. Moreover, the enamel mineral content may be so low in these teeth as to make bonding unsuitable. In other words, enamel that is severely deficient in mineral content (e.g., less than 70% mineral per volume) would probably be a poor risk for any composite bonding technique due to the inherent weakness of the enamel. As a rule of thumb, we propose that enamel that can be penetrated easily with an explorer would not be a good candidate for NaOCl pretreatment and bonding.

Even normal enamel may fracture during orthodontic appliance removal. For patients affected with AI, this risk may be dramatically increased. A clinician should inform AI patients and/or parents in very clear terms of potential difficulties involved in orthodontic treatment, including the possibility of fracture or loss of affected enamel. These discussions should be well documented during the informed consent process.

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