Enamel hypoplasia: A rare case report involving all permanent canines

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Abstract

Enamel hypoplasia (EH) is restricted to ectodermal disturbance, associated to alterations in the organic enamel matrix which can source the white flecks, narrow horizontal bands, lines of pits, grooves and discoloration of the teeth altering from yellow to dark brown. Enamel defects have been connected with a broad spectrum of etiologies together with genetic and epigenetic factors such as local, systemic and environmental factors. In our case EH involves all the permanent canines, which are found to be uncommon. Hence, we report a rare case of enamel hypoplasia.

Keywords: Ameloblasts, enamel hypoplasia, permanent canines, pitted type

Introduction

Enamel formation is a complex and extremely regulated process. Enamel is the covering of the tooth and is greatly mineralized tissue in the body. It consists of 96% inorganic hydroxyapatite crystallites. Cells responsible for the development of the enamel are called ameloblasts. They cover the whole surface of the enamel as it forms, but are lost as the tooth emerges. Partial or imperfect development of the organic enamel matrix of teeth associated with hypocalcification and hypomineralization of enamel is defined as enamel hypoplasia (EH).[1]

EH is restricted to ectodermal disturbance, associated to alterations in the organic enamel matrix which can source the white flecks, narrow horizontal bands, lines of pits, grooves and discoloration of the teeth altering from yellow to dark brown.[2] Tooth enamel is unique in that, remodeling does not occur after initial formation. Therefore, abnormalities in enamel formation are etched permanently on the tooth surface.

Hypoplasia marks merely if the injury occurs at some stage in enamel development. Just the once the enamel has calcified no such defect can be formed.[3]

Enamel defects have been connected with a broad spectrum of etiologies together with genetic and epigenetic factors such as local, systemic and environmental factors.[4] Generally causes of EH is difficult to determine. Since the ameloblast is a sensitive type of cell and easily damaged, it is likely that in cases in which the etiology cannot be determined, the causative agent may be some illness or systemic disturbance that has made no impression on the patient. Timing of ameloblastic damage has a great effect on location and appearance of the defect in the enamel.[5]

In our case, EH involves all the permanent canines which are found to be rare. Hence, we are reporting a rare case of EH.

Case Report

A 14-year-old patient who is female reported to the dental clinic with a chief complaint of pain in the upper left back region of her mouth.

On intra-oral examination deep carious lesion was associated with 26. On further examination, all the permanent canines 13, 23, 33 and 43 were associated with two brown pitted spots on the incisal 1/3rd of labial surface one on the mesial and the other on the distal half [Figures 1 and 2]. Radiographic examination of all canines through intraoral periapical showed two localized small radiolucencies in the incisal third of the tooth [Figures 3 and 4].

Patient was attended to the chief complaint and treated with root canal treatment followed by crown fixation. Clinical diagnosis for these canines was given as “EH,” pitted variety. Patient had no significant medical history.

Discussion

EH was first used by Zigmondy in 1894. Hypoplasia is preferable to the old term “enamel atrophy” because the condition is characterized by an underdevelopment of the enamel, whereas
The word "atrophy" indicates a wasting or reduction in size of a fully developed tissue or organ. \(^6\)

EH can be defined as a partial or imperfect development of the enamel matrix of teeth. Two fundamental types of EH subsist: \(^1,6\)

1. EH caused by environmental factors
2. EH caused by hereditary defects (amelogenesis imperfecta).

Almost all visible environmental enamel defects can be classified into one of the three patterns: \(^6\)

1. Hypoplasia
2. Diffuse opacities
3. Demarcated opacities.

Classification of enamel defects recorded in the study done by Littleton and Townsend based on the developmental defects of enamel: \(^7\)

<table>
<thead>
<tr>
<th>Types of defects (hypoplasia only)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pits</td>
</tr>
<tr>
<td>Grooves: Horizontal</td>
</tr>
<tr>
<td>Grooves: Vertical</td>
</tr>
<tr>
<td>Missing enamel</td>
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<tr>
<td>Number and demarcation</td>
</tr>
<tr>
<td>Single</td>
</tr>
<tr>
<td>Multiple</td>
</tr>
<tr>
<td>Diffuse: Fine white lines</td>
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<tr>
<td>Diffuse: Patchy</td>
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</tbody>
</table>

Environmental EH can be caused by a number of different factors each capable of producing injury to the ameloblasts which include: Nutritional insufficiency (vitamins A, B, C and D); Exanthematous diseases (e.g.: Measles, chicken pox, scarlet fever); birth injury; congenital syphilis; hypocalcemia; prematurity, Rh hemolytic disease; local infection or trauma and Ingestion of fluorides. \(^1\)

Hypoplasia marks barely if the damage occurs throughout the time the teeth are developing, or more in particular, during the formative stage of enamel maturity. Formerly if the enamel has
been calcified, no such defect can be formed. The timing of the ameloblastic damage has a great effect on the location and appearance of the defect in the enamel. The site of coronal damage correlates with the area of ameloblastic activity at the time of the injury; the affected enamel is restricted to the areas in which secretory activity or active maturation of the enamel was occurring.[1,3] If the interference takes place in the 1st year (called the infancy period), the permanent teeth affected are the first molars, the incisors (except the maxillary lateral incisors), and the canine teeth. It is striking that the maxillary lateral incisors mineralize after the central incisors and canines, approximately after the age of 10 months. If the interference takes place in early childhood (approximately 13-34 months) the maxillary lateral incisors and premolars which begin to calcify during this period are also affected.[6]

In mild environmental hypoplasia, there might be simply a few minute grooves, pits or fissures on top of the enamel surface. If the condition is further severe, the enamel may show evidence of rows of deep pits arranged horizontally diagonally on the surface of the tooth. Sometimes there might be only a single row of such pits or several rows signifying a sequence of injuries.[1,6] Thus, the present case is of mild environmental hypoplasia involving only permanent canines with two pits on the labial surface.

The treatment of dental problems of patients with EH presents an interesting challenge to the dental surgeon. Management of patients with EH should start with early diagnosis to prevent restorative problems at a later stage. The esthetic treatment of EH is limited to the removal of surface.[2]

**Conclusion**

EH is quantitative enamel defect associated with a broad spectrum of etiologies. Most defects in enamel are cosmetic rather than functional dental problems. The main clinical characteristic is extensive loss of tooth tissue, carious lesions, tooth sensitivity and poor aesthetics. Appropriate interventions and early dental treatment may help prevent destruction and loss of the dentition.

**References**