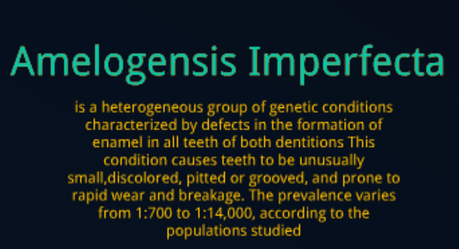
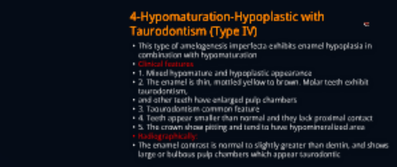
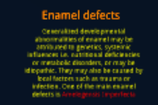




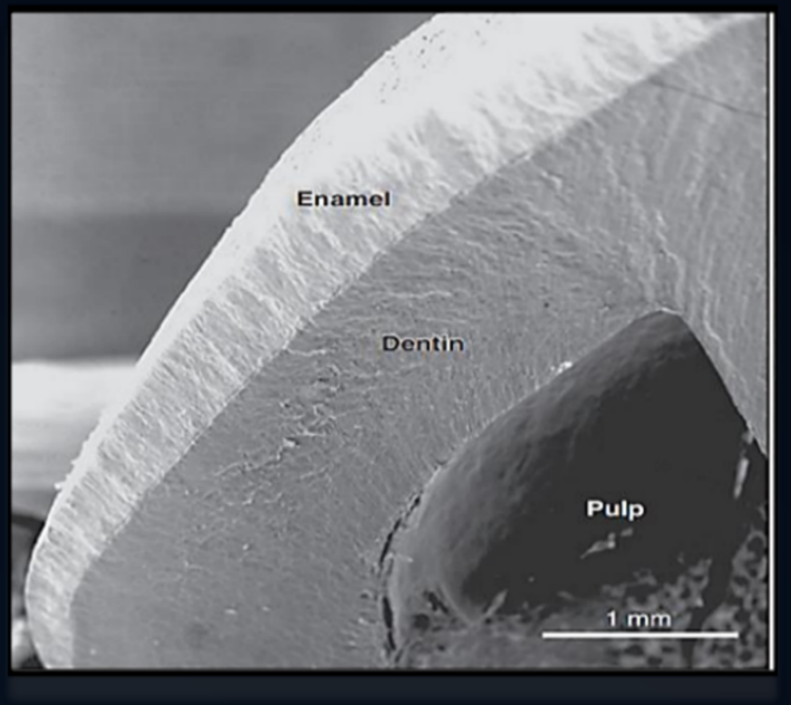
Amelogenesis Imperfecta

Assist. Lect. Rusul Jaffar Hadi



Enamel

- Enamel is the hardest calcified matrix of the body. The cells that are responsible for formation of enamel, are the ameloblasts, are lost as the tooth erupts into the oral cavity, and hence enamel cannot renew itself. Enamel brittle is therefore an underlying layer of more resilient dentin is necessary to maintain its integrity, if this supported layer of dentin is destroyed by caries or improper cavity preparation, the unsupported enamel fracture easily



Chemical Properties of enamel

The enamel consists mainly of inorganic material (96%) and only a small amount of organic substance and water (4%), organic material consists of some unique proteins, amelogenins (90%) and the non-amelogenins such as enamelin and ameloblastin (10%), The inorganic material of the enamel is hydroxyapatite crystals and also contain various elements such as magnesium, and fluoride

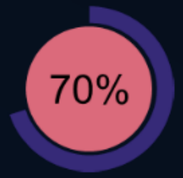
The stages of amelogenesis

1- Enamel matrix deposition

Ameloblast synthesize and secrete enamel matrix

- Hydroxyl apatite crystal

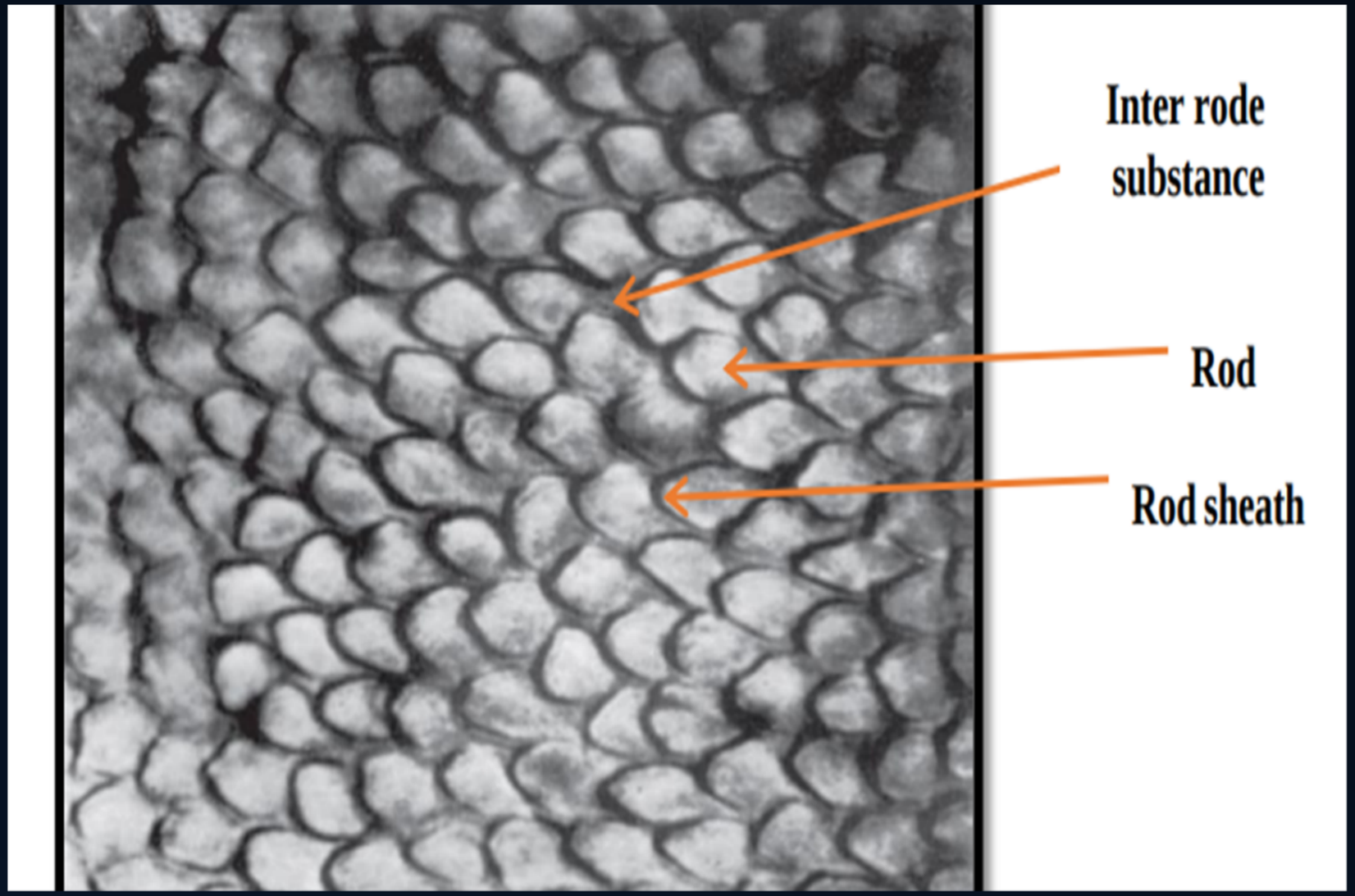
- water and proteins such as amelogenin, ameloblastin, enamelin, and enzymes like enamelisin, also called Matrix Metalloproteinase 20 (MMP_20), these proteins are responsible for creating and maintaining an extracellular environment favorable to mineral deposition



2- Mineralization of the Enamel matrix

When the E. matrix reach the full thickness, mineralization will be started,This process involved additional minerals with the removal of organic material and water to reach 96% mineral content. This minerals makes the initial E. crystals that formed in first stage to grow wider and thicker due to the deposition of large amount of hydroxy apatite crystals

The basic structure of Enamel



Enamel defects

Generalized developmental abnormalities of enamel may be attributed to genetics, systemic influences i.e. nutritional deficiencies or metabolic disorders, or may be idiopathic. They may also be caused by local factors such as trauma or infection. One of the main enamel defects is Amelogensis Imperfecta

Amelogensis Imperfecta

is a heterogeneous group of genetic conditions characterized by defects in the formation of enamel in all teeth of both dentitions This condition causes teeth to be unusually small,discolored, pitted or grooved, and prone to rapid wear and breakage. The prevalence varies from 1:700 to 1:14,000, according to the populations studied

Causes of Amelogenesis imperfecta

Amelogenesis imperfecta presents large variability in its clinical expression. Mutations have been reported in different genes. Some of them encode for enamel proteins, either structural (amelogenin, enamelin,ameloblastin) or enzymatic (kallikrein 4, MMP20); some others encode for transcription factors , cellular proteins, cellular receptor and calcium carrier.These genes provide instructions for making proteins that are essential for normal tooth development. These proteins are involved in the formation of enamel, which is the hard, calcium-rich material that forms the protective outer layer of each tooth. Mutations in any of these genes alter the structure of these proteins or prevent the genes from making any protein at all. As a result, tooth enamel is abnormally thin or soft and may have a yellow or brown color. Teeth with defective enamel are weak and easily damaged

Classification of amelogenesis imperfecta

- Clinical researchers usually classify AI into four main types of which 17 subtypes are recognized. The most commonly used classification of AI was proposed by Witkop (1988) which was later revised by Nusier (2004). The main types are based on clinical appearance, radiographic appearance and enamel thickness, and the subtypes are based on mode of inheritance. The main types are:
- hypoplastic (type I)
- hypomaturation (typeII)
- hypocalcified(typeIII)
- hypomaturation/hypoplasia/taurodontism (type IV).

AI may be inherited as an X-linked, autosomal dominant, or autosomal recessive genetic trait, depending on the mode of inheritance

1-Hypoplastic AIH (type I)

Inadequate deposition of enamel matrix with normal mineralization in which that quantitative defect in enamel matrix formation

Clinical features

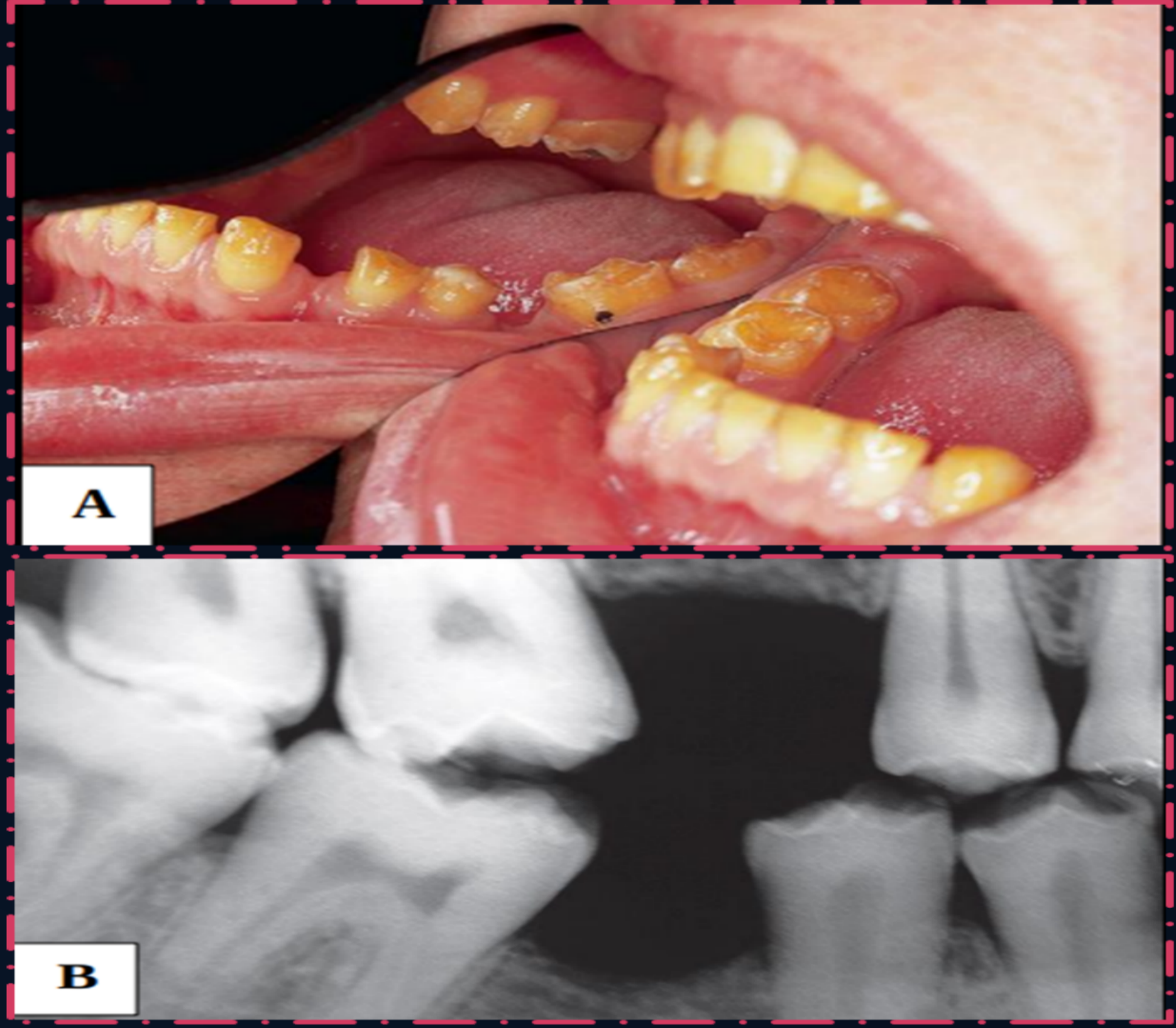
1_-Teeth are yellow to light brown

2_surface is rough with pits or larger area defects

3_-No pain is associated with this AI, although some slight thermal sensitivity may sometimes be reported

4_-Because of reduced enamel thickness in some cases, abnormal contour and absent interproximal contact points may be evident

Radiographically Thin enamel but normal radiodensity will be seen, Enamel can be distinguished from the underlying dentin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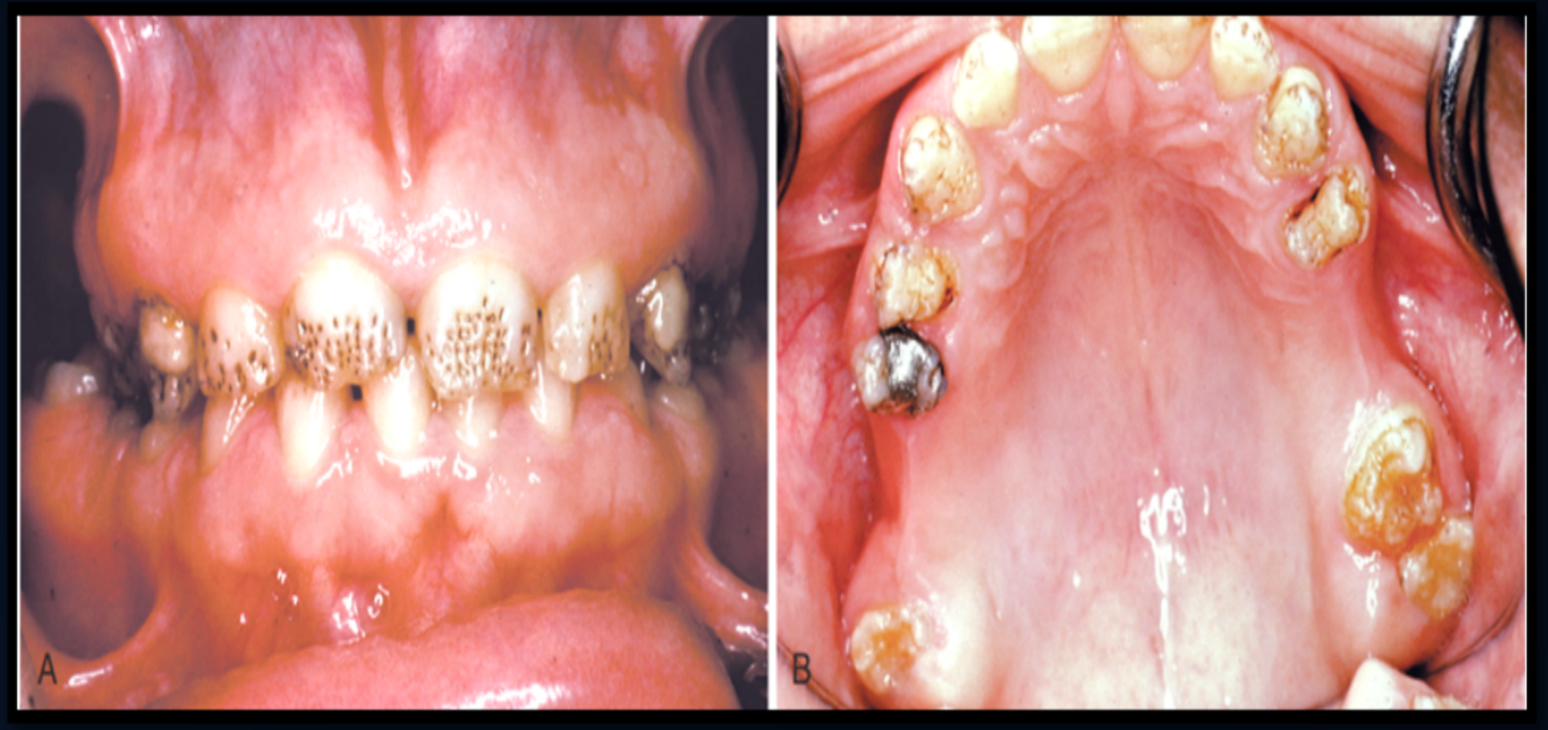
Subtype classification of hypoplastic AI according to the clinical appearance

1-Hypoplastic generalized pattern affects the entire dentition. Pin point pits are presents on the buccal surface of the teeth and later on these pits may be stained. The enamel between these pits is of normal thickness, hardness and colouration.
2-Hypoplastic localized pattern affects only some teeth in the oral cavity. Large defects on the buccal middle third of the teeth are seen.
3-Hypoplastic smooth pattern exhibit smooth surface which is thin, hard and glossy.
4-Hypoplastic rough pattern the enamel is thin, hard and rough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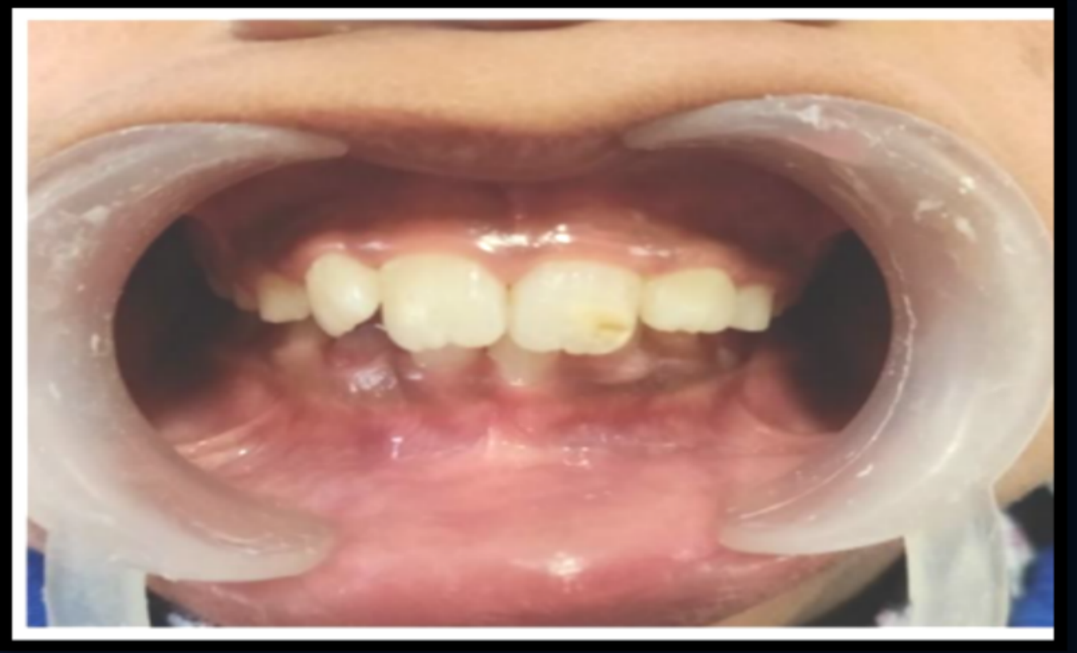

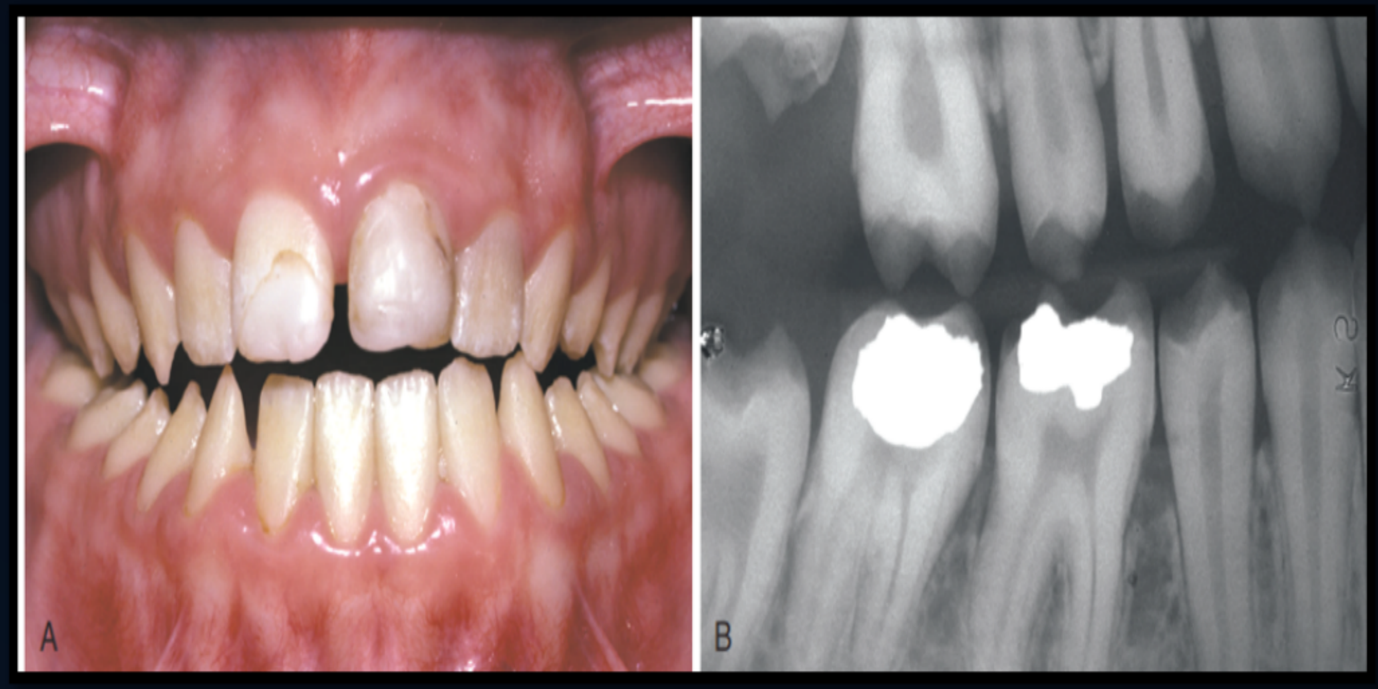
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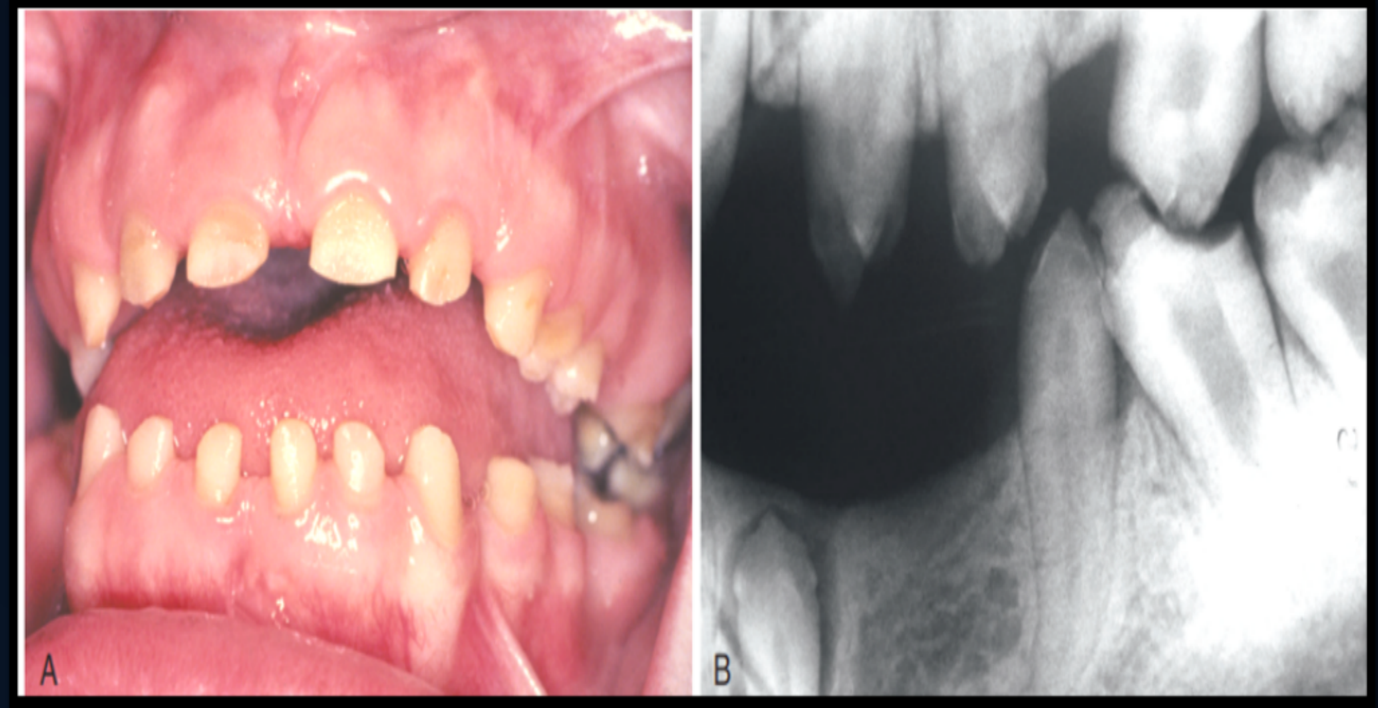
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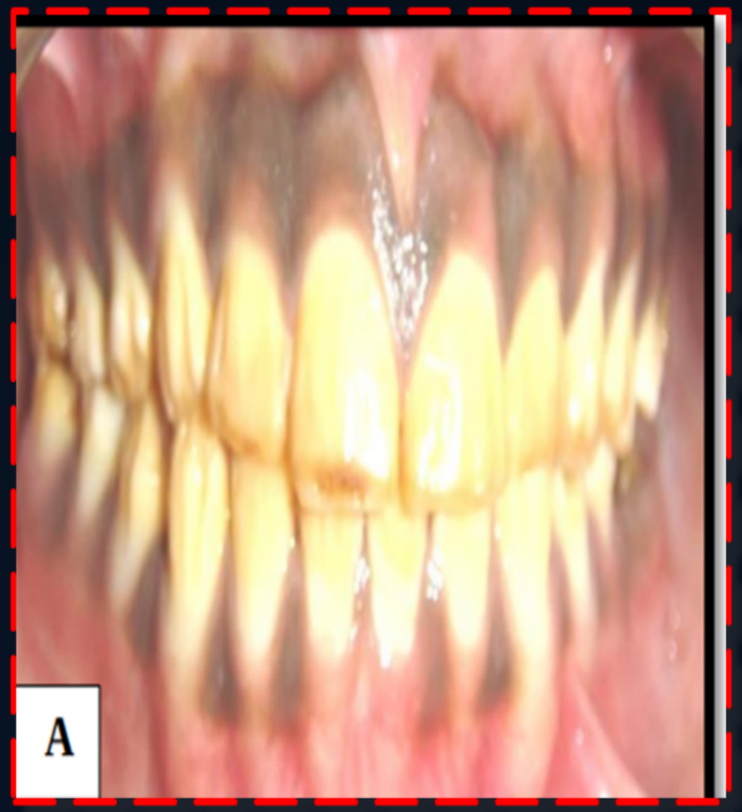
•2-Hypomaturation

- Hypomaturation AI (typeII) consists of a defect in matrix protein degradation.In enamel, which is the most calcified structure in the organism, proteins must be degraded and removed to achieve final crystal growth. So there is normal matrix formation and defective mineralization and occur during the maturation stage.
- Clinical features
- 1. The color of teeth here varies from creamy opaque to marked yellow/brown.
- 2. The surface of the teeth appear soft and rough leading to sensitivity due to dentinal exposure but not as severe as the hypocalcified type.
- 3. The enamel thickness is normal but often chips off and abrades away easily
- Radiographically
- Contrast between enamel and dentin is lost, similar radiodensity as dentine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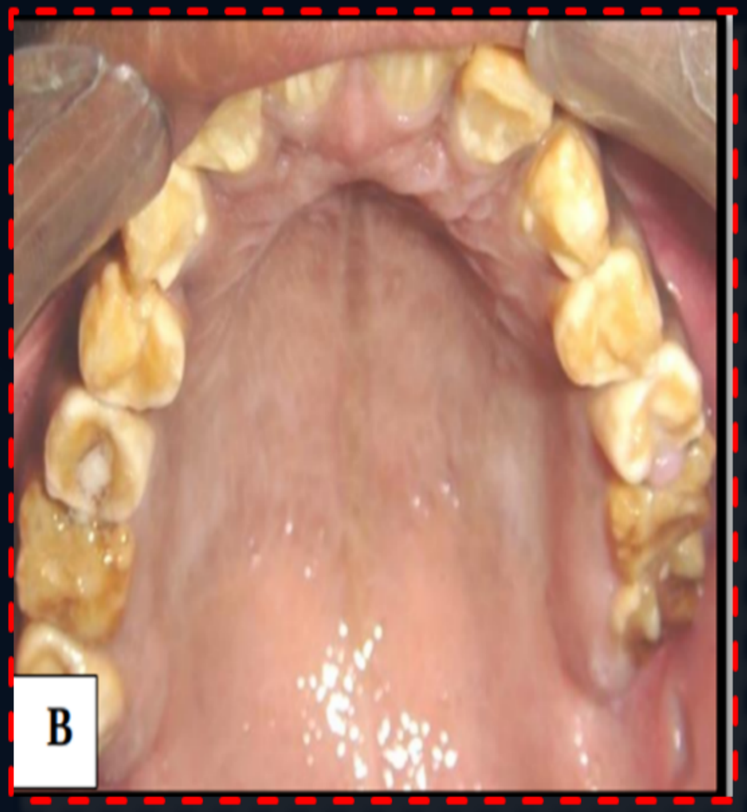
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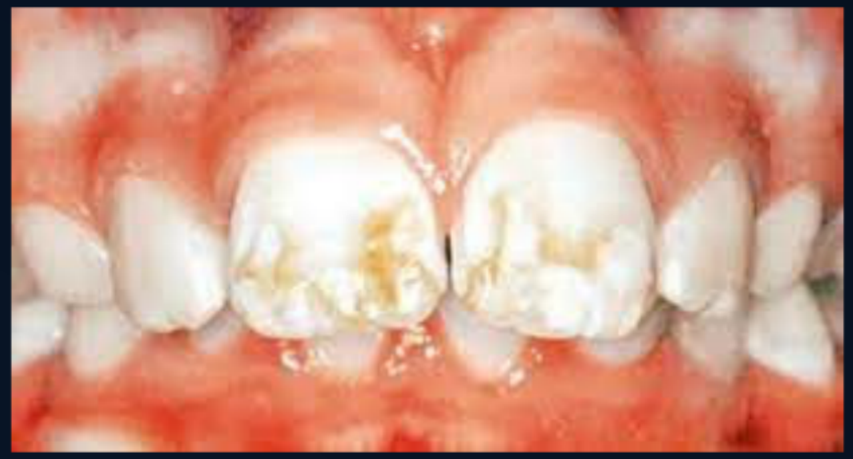
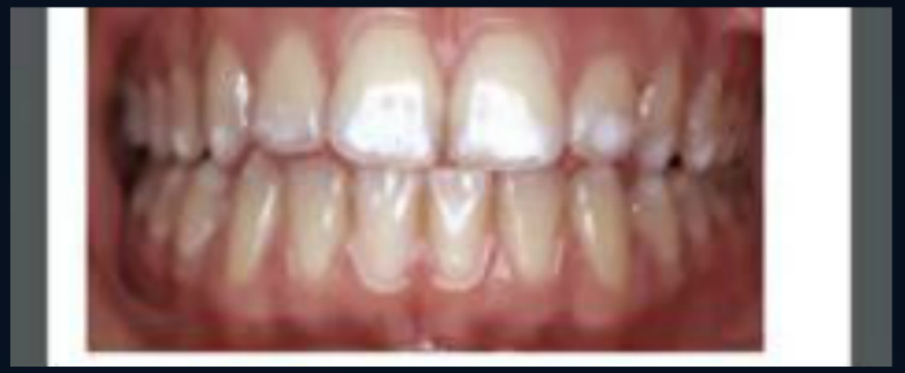
Subtype classification of hypomaturation AI according to the clinical appearance

1-The pigmented pattern : the enamel has mottled and brown appearance

2-Snow- capped pattern: the enamel has zone of opaque white on incisal and occlusal edges.





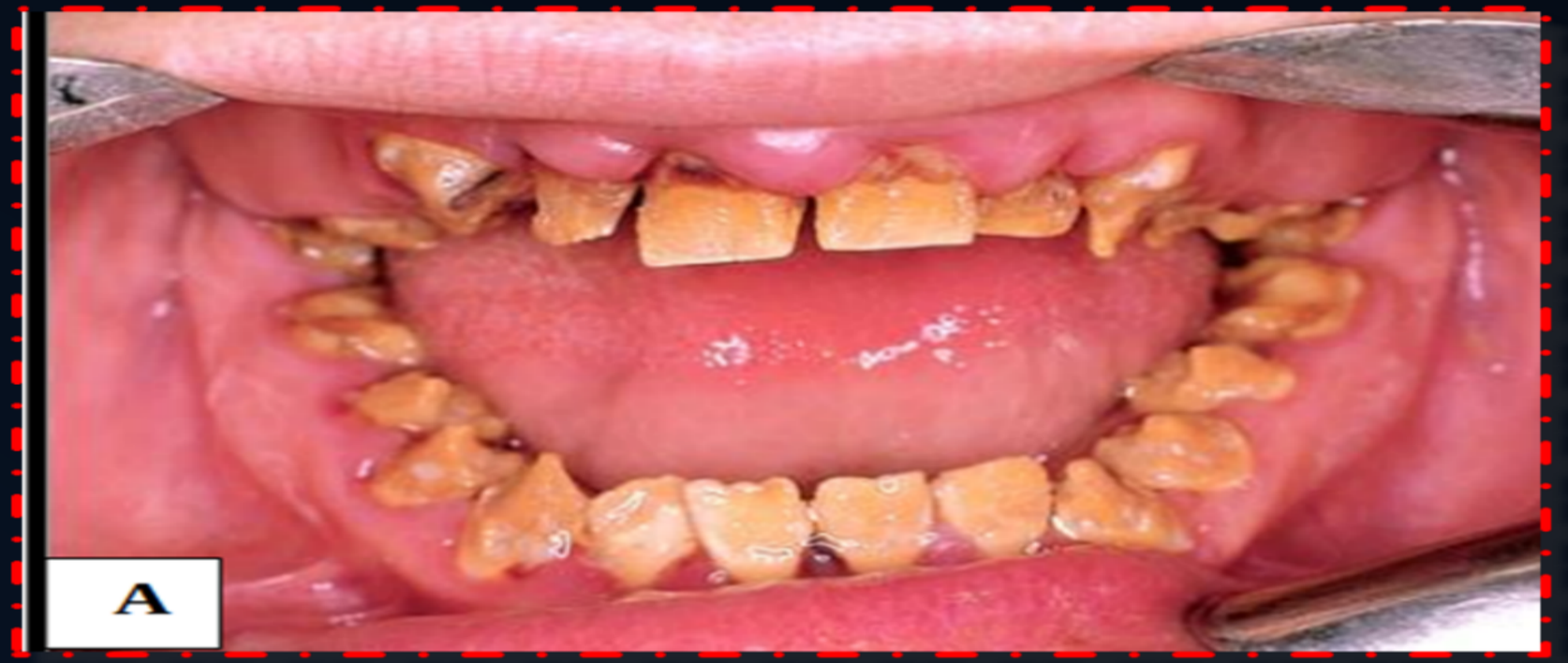
3-Hypocalcified AI (Type III)

Qualitative defect occurs when the enamel is insufficiently mineralised and soft with normal matrix formation. on comparison with hypomaturation variety, the degree of mineralization is markedly reduced.

Clinical features

1. the crowns of the teeth in such cases appears to be opaque white to yellowbrown, soft rough enamel surface.
2. dental sensitivity and very poor aesthetic
3. Due to severe hypomineralization, there may be early loss of enamel.
4. The thickness of enamel appears to be normal at eruption that often chips and but, tends to abrade easily post eruptively.

Radiographically Normal enamel thickness (immediately after tooth eruption) but less radiopacity compare to dentine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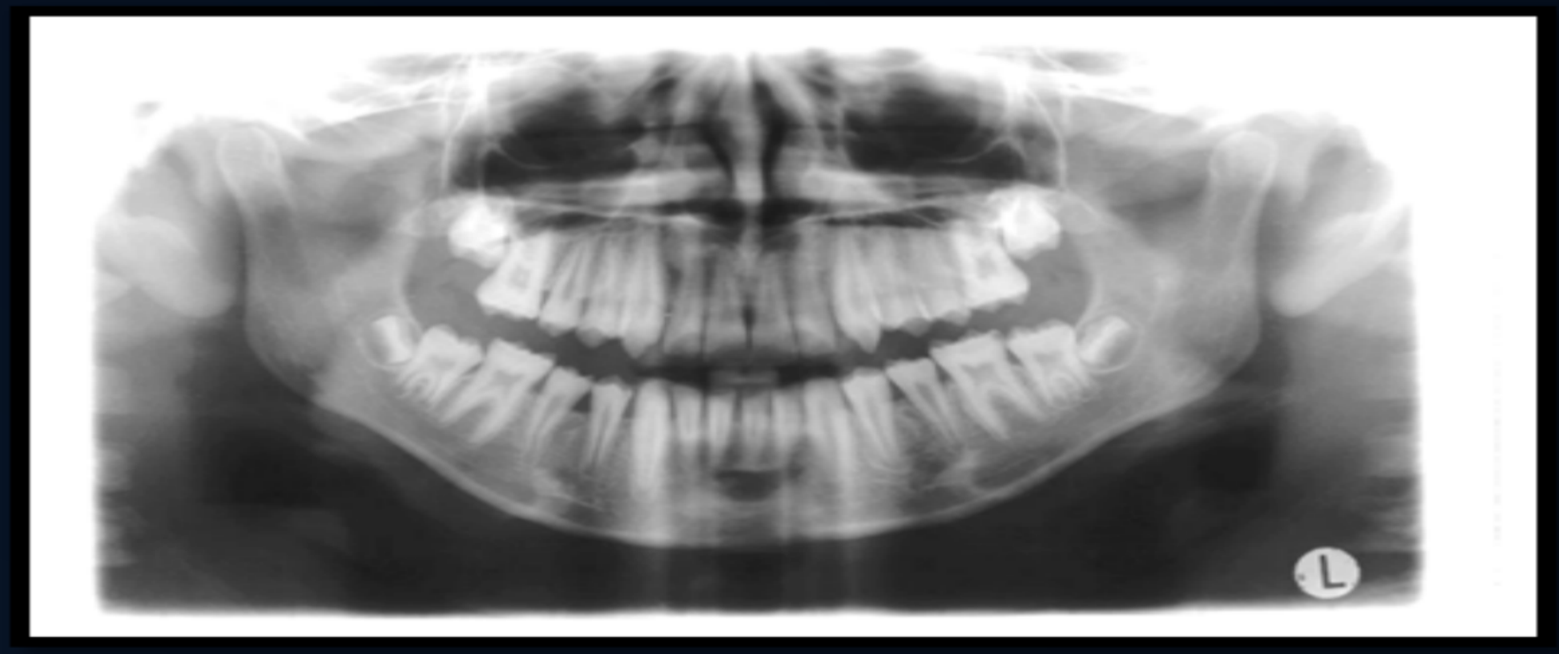
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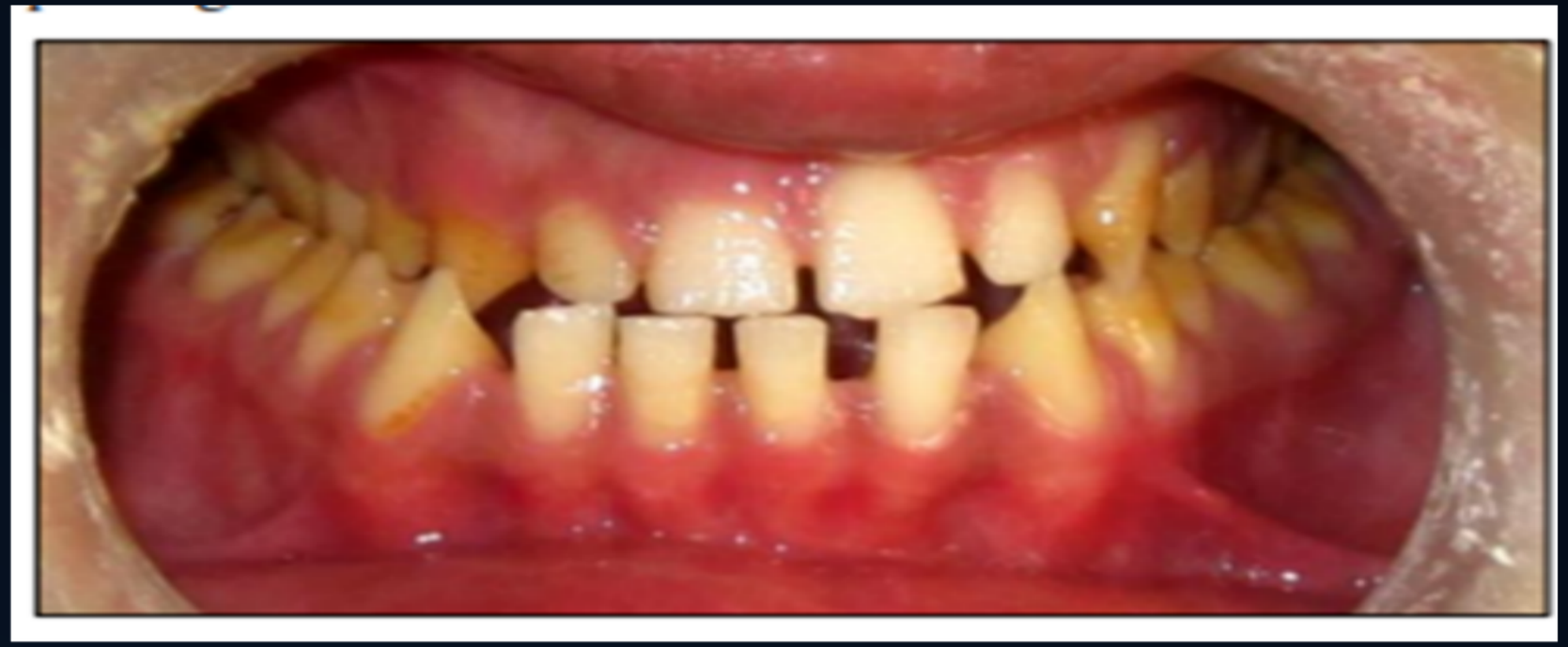
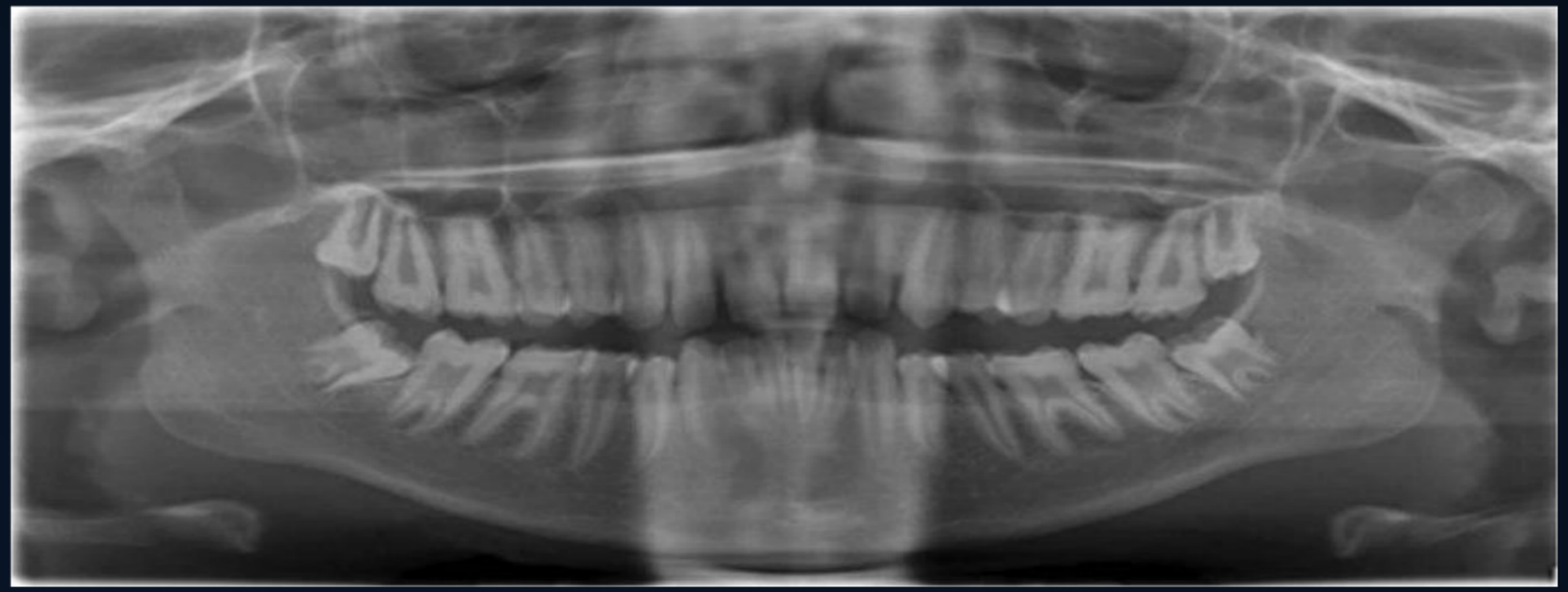
4-Hypomaturation-Hypoplastic with Taurodontism (Type IV)

- This type of amelogenesis imperfecta exhibits enamel hypoplasia in combination with hypomaturation
- Clinical features
- 1. Mixed hypomature and hypoplastic appearance
- 2. The enamel is thin, mottled yellow to brown. Molar teeth exhibit taurodontism,
- and other teeth have enlarged pulp chambers
- 3. Taourodontism common feature
- 4. Teeth appear smaller than normal and they lack proximal contact
- 5. The crown show pitting and tend to have hypomineralized area
- Radiographically:
- The enamel contrast is normal to slightly greater than dentin, and shows large or bulbous pulp chambers which appear taurodontic





Clinical consideration

Compromised periodental health

Asymmetric gingival contour

Hypersensitive tooth

High risk of cavities

Discolored and pitted surface

short clincal crown height

Malformed tooth

Congenital missing tooth

Pulp calcification

Taurodontisim

Anterior and posterior open bite

Multiple posterior diastemata

Loss of vertical dimension of occlusion

Impaired esthetics

Diagnosis of amelogenesis imperfecta

The severity of AI can vary significantly between patients and often it is difficult to make a diagnosis of the phenotype from clinical examination alone. In some cases the different phenotypes described may coexist in the same patient and on the same tooth

dentist can identify and diagnose amelogenesis imperfecta on the basis of the patient's family history , the signs and symptoms present in the affected individual and x_ray

Genetic testing is available for the genes AMELX, ENAM, and MMP20

Histological confirmation can be done, but required extraction of the affected tooth, which is not a good idea in all cases except where prognosis of that tooth is poor

Management (Treatment) of AI

Objectives of the treatment

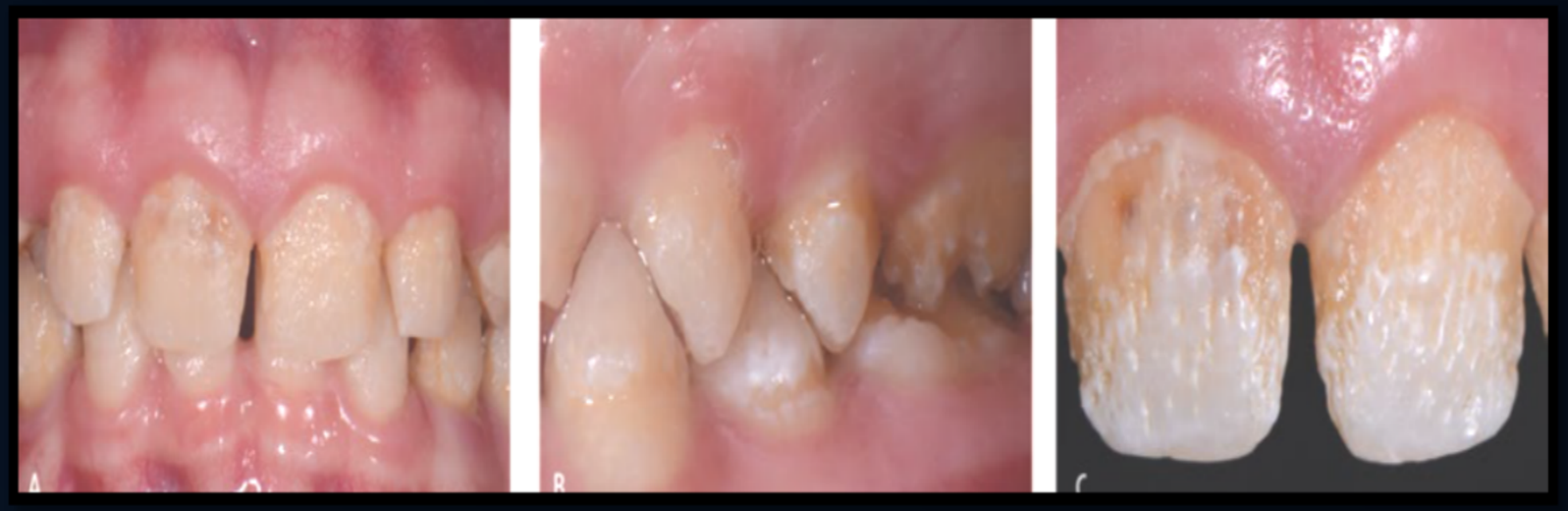
1. Pain prevention

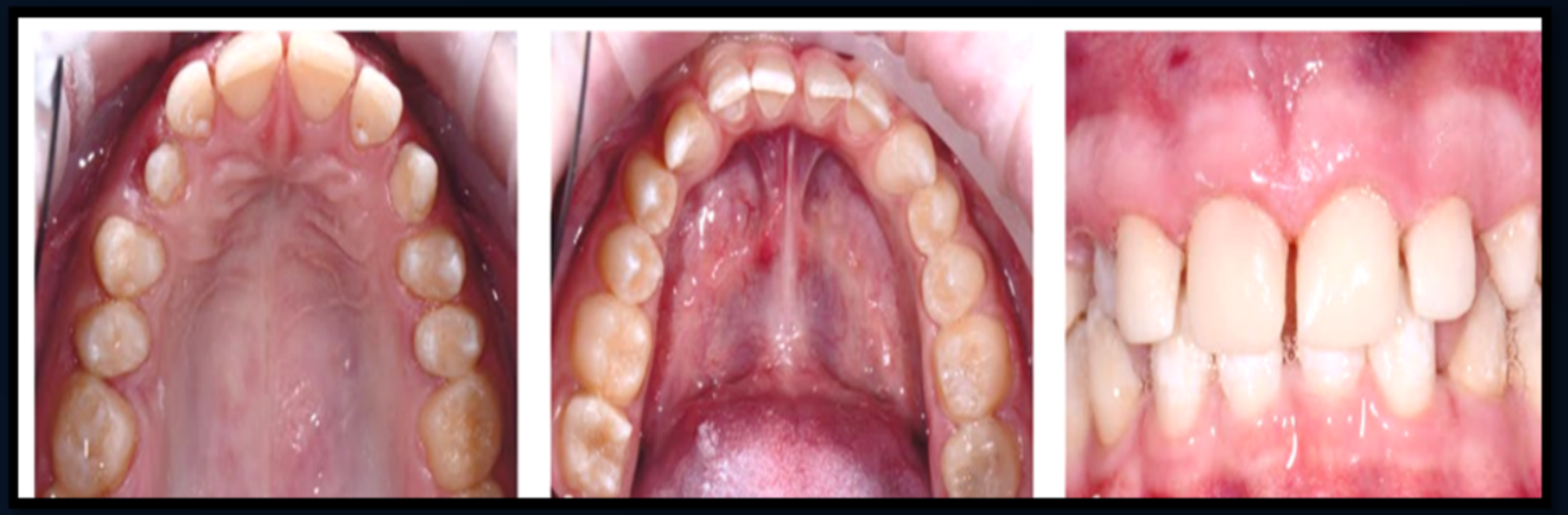
2. Protection of dental tissue integrity in order to maintain occlusal function and limit dental biofilm retention

3. Restoration of smile aesthetics

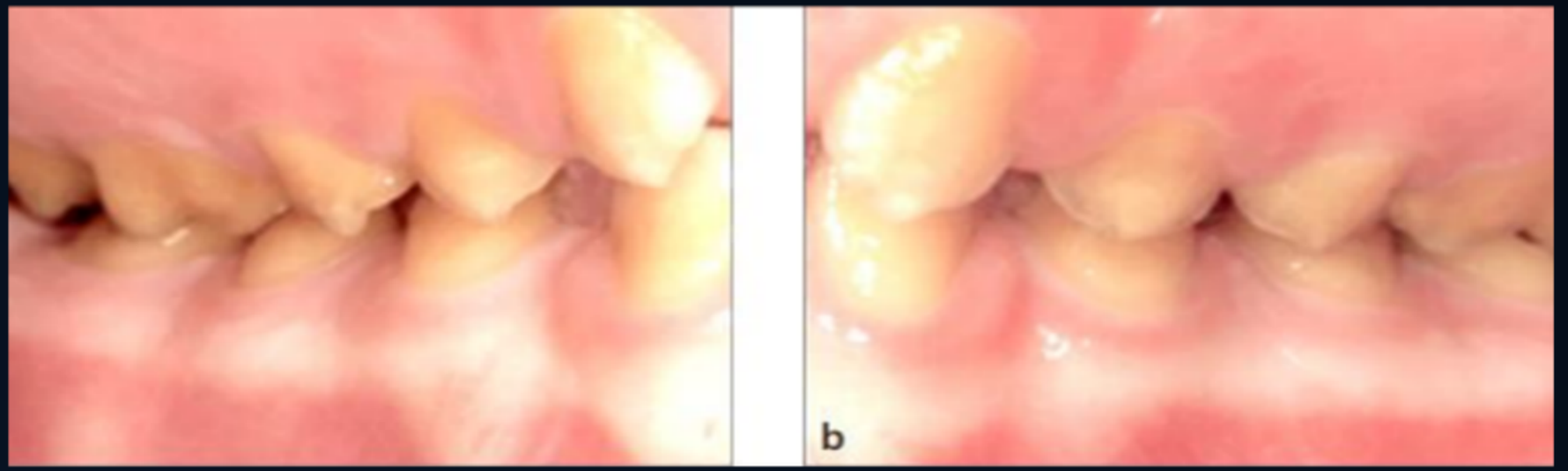
4. To alleviate the sensitivity

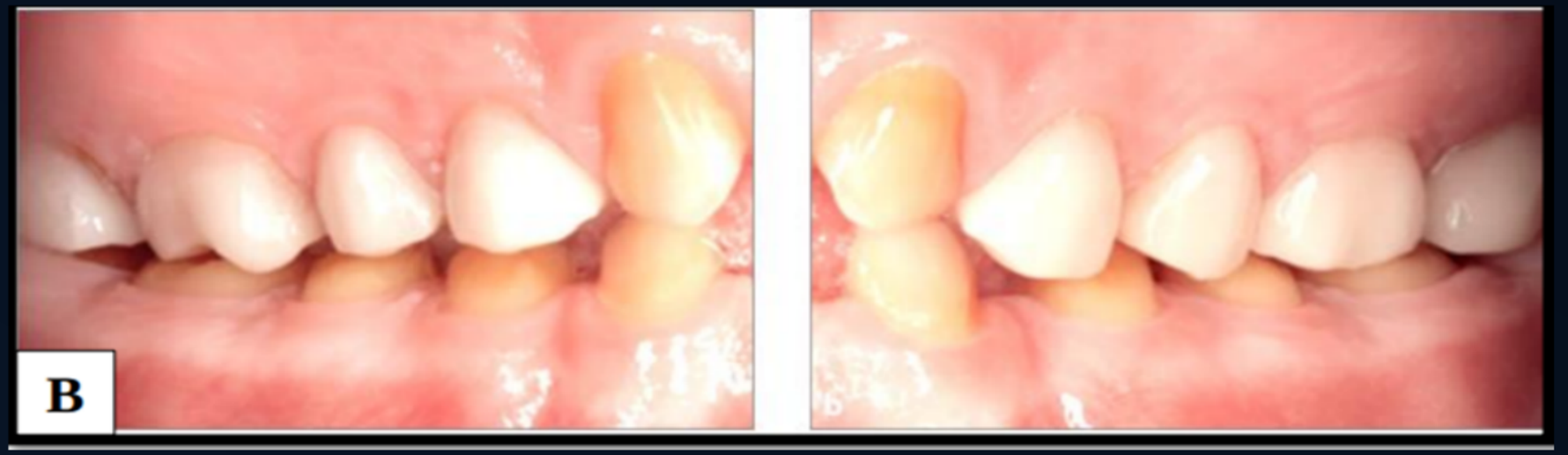
1-Direct composite



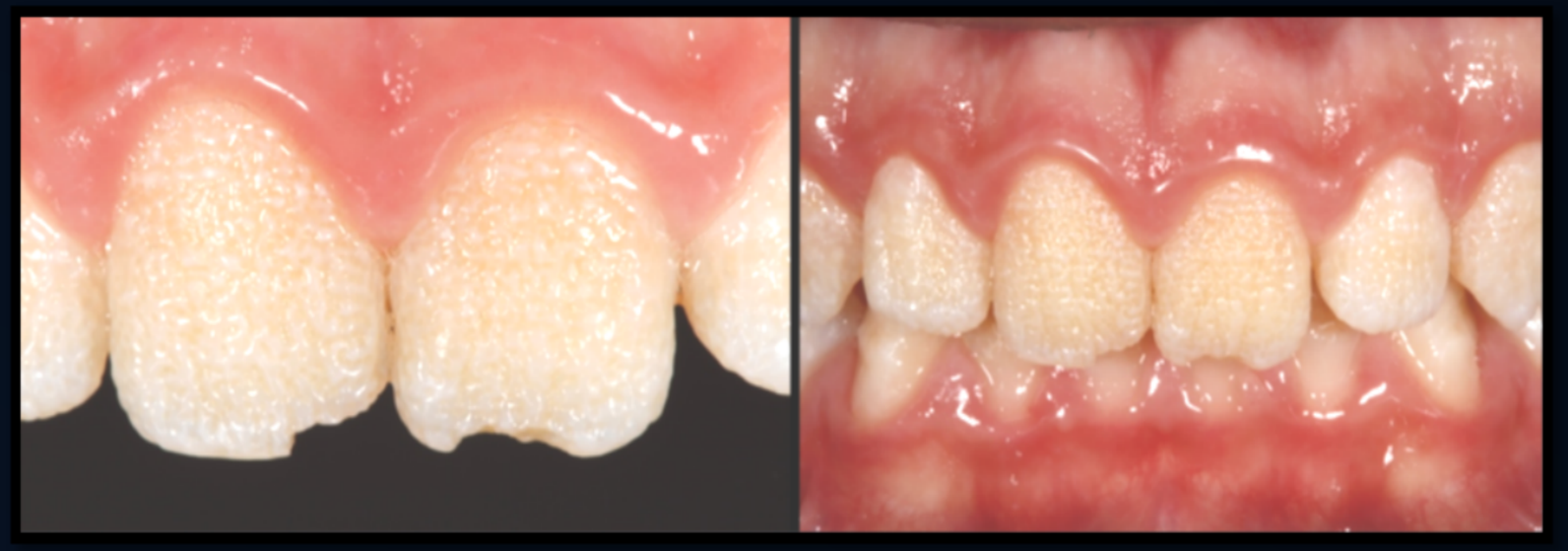


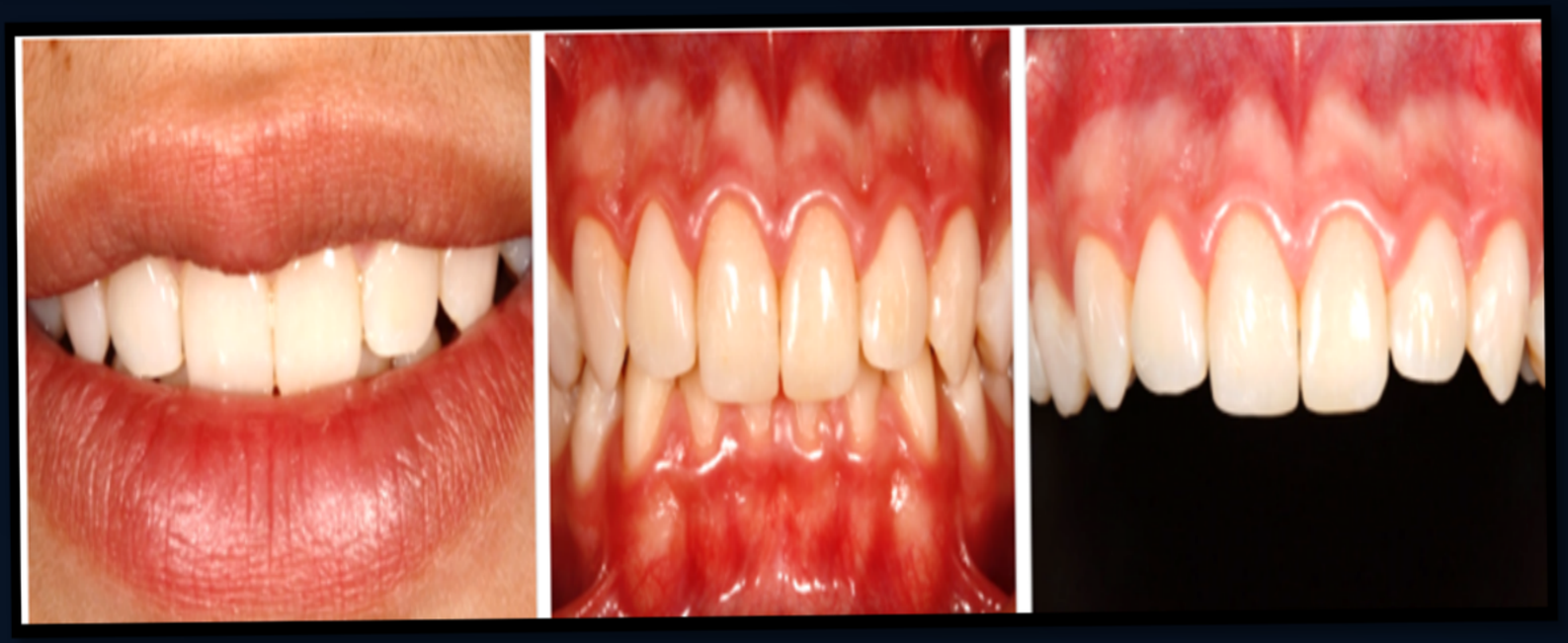
2-Indirect composite



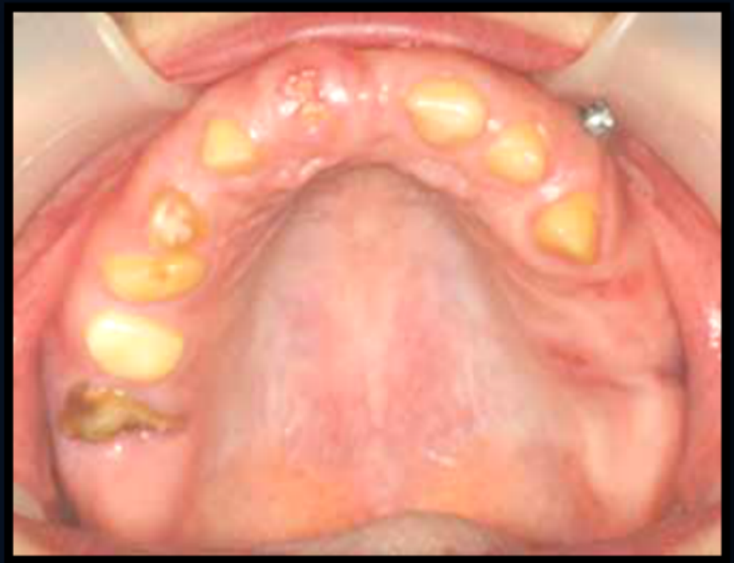


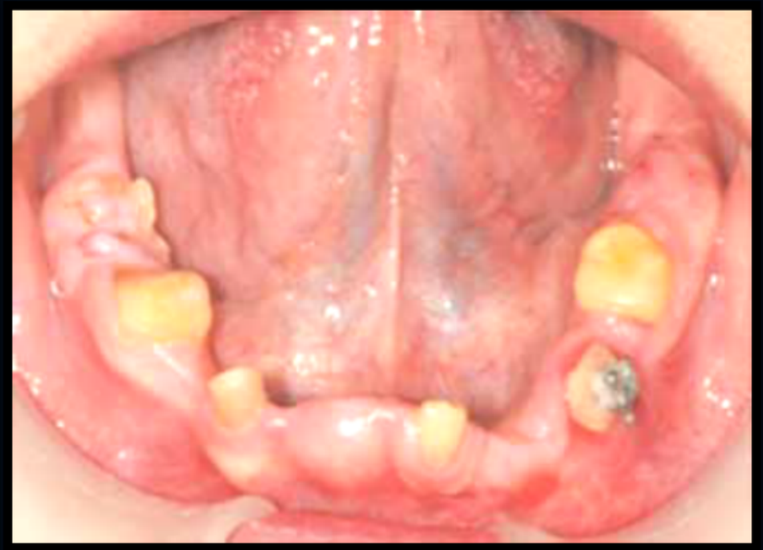
3-Porcelain veneers

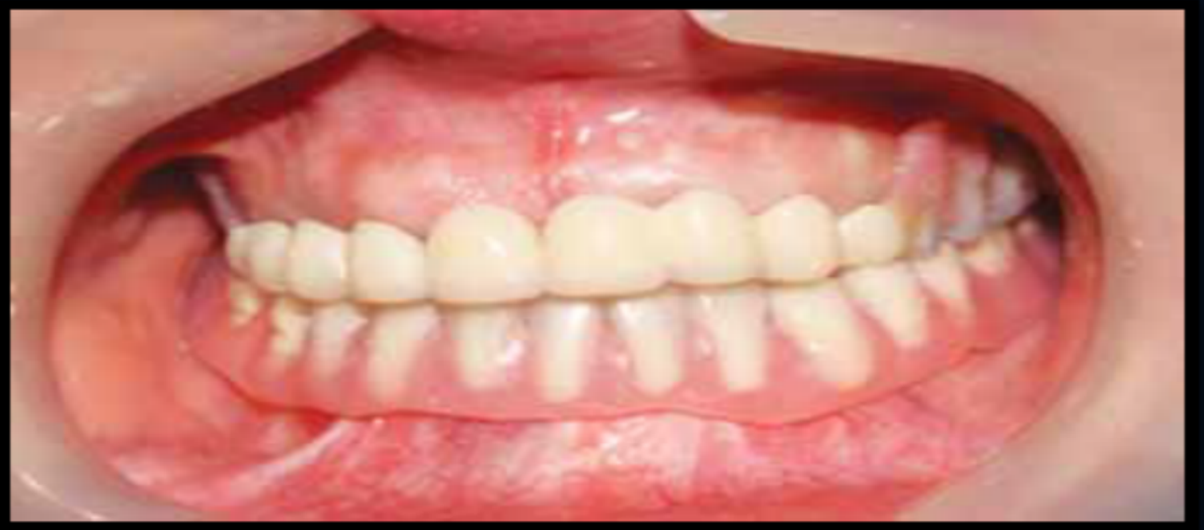




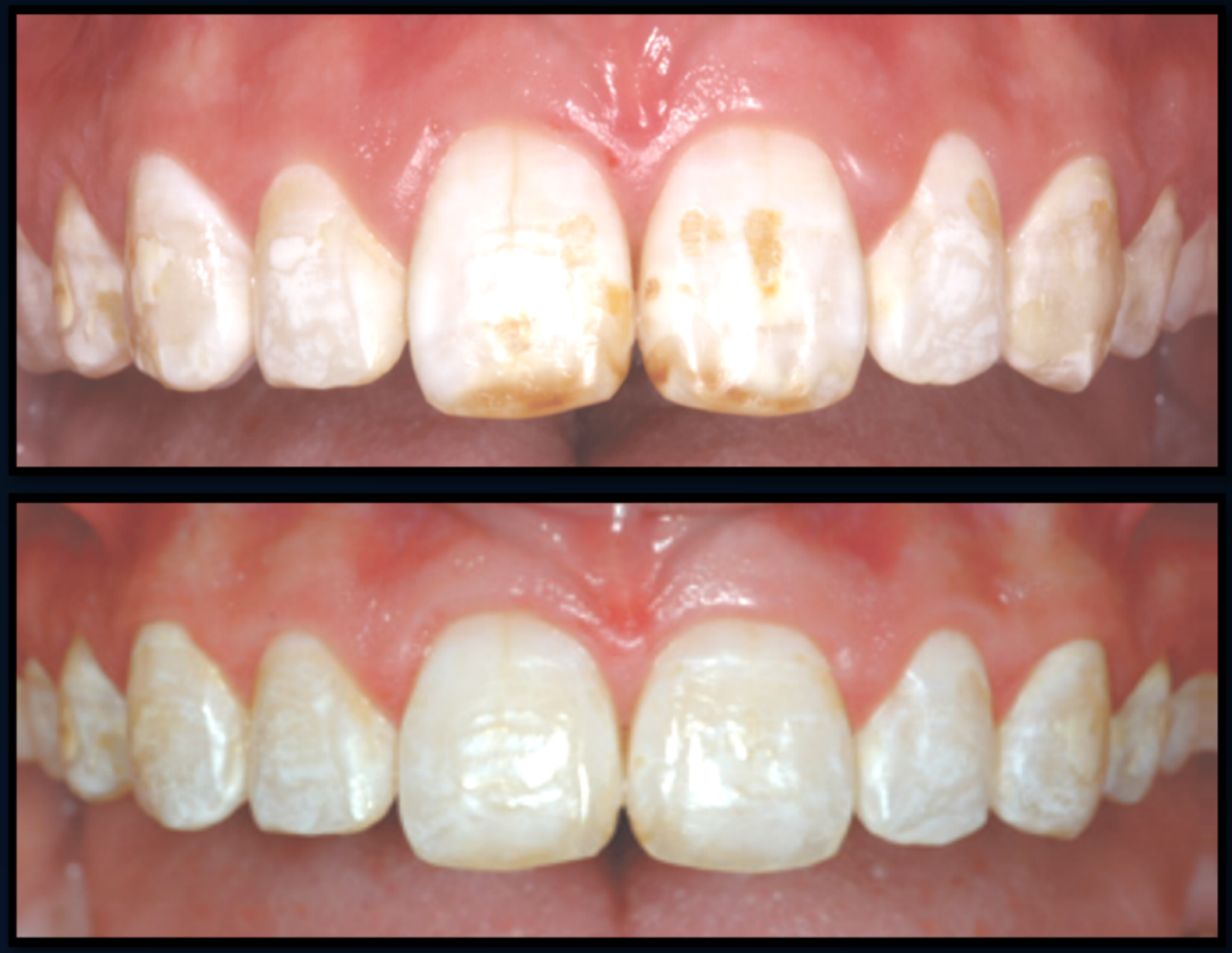
4-Dentures



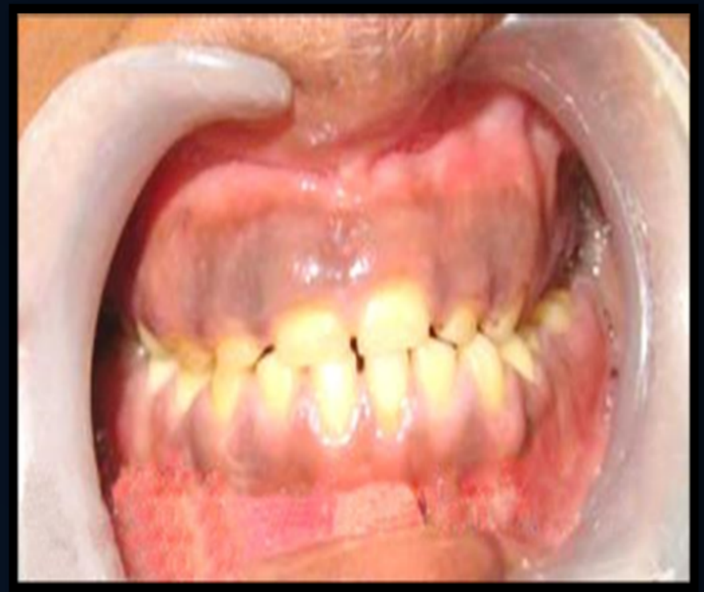


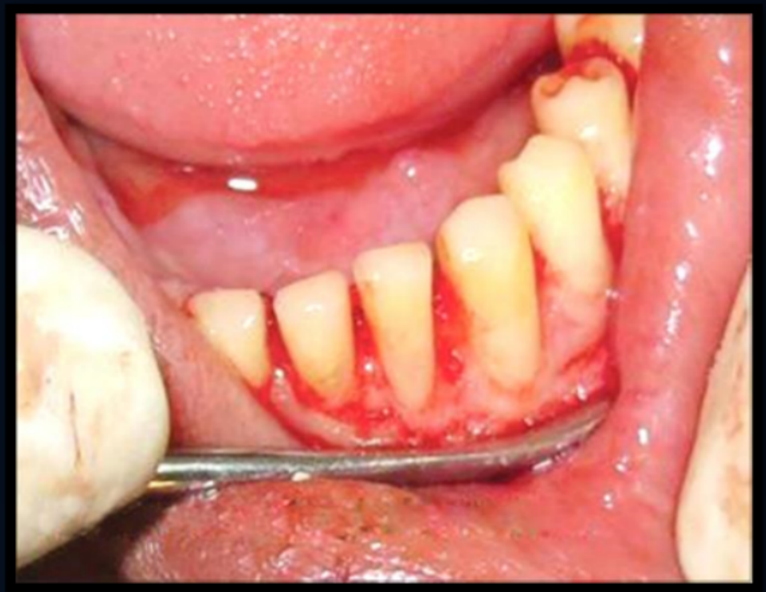


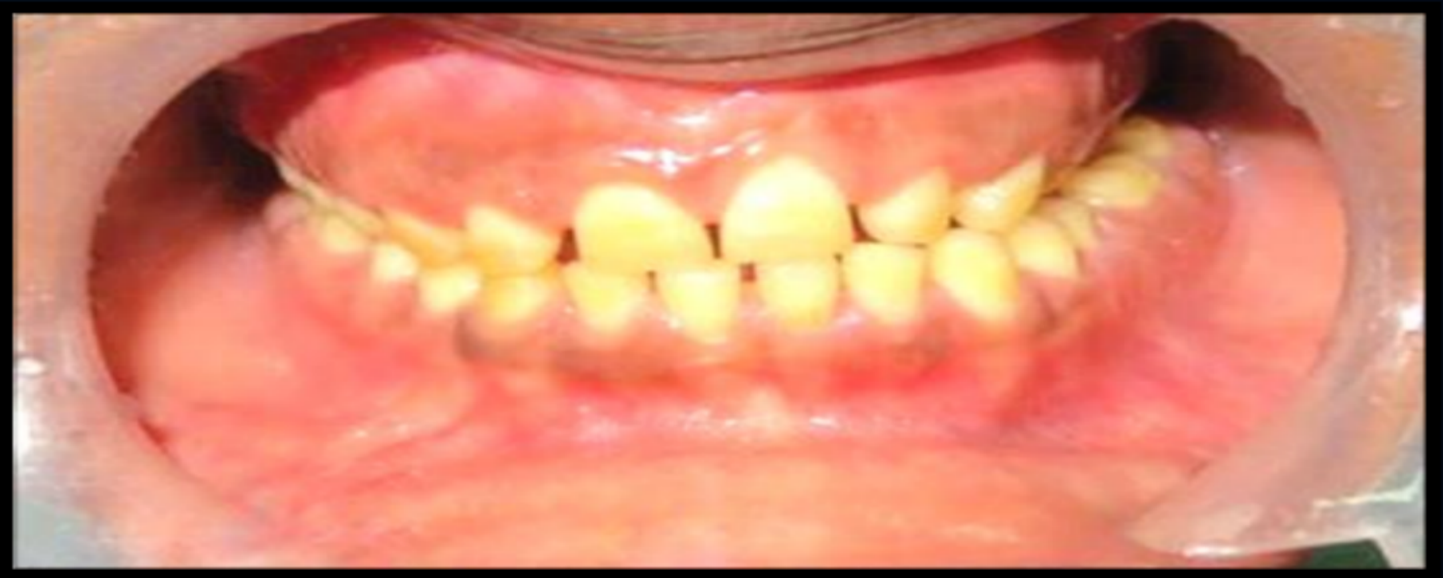
5-Bleaching and microabrasion



7- Crown lengthening surgery








THANK YOU
FOR YOUR ATTENTION