INTERNATIONAL JOURNAL OF HEALTH & MEDİCAL RESEARCH

ISSN(print): 2833-213X, ISSN(online): 2833-2148

Volume 04 Issue 12 December 2025

DOI : 10.58806/ijhmr.2025.v4i12n05

Page No. 670 - 677

Dental Anomalies: an Updated Review on Development, Morphology, and Clinical Management

Büşra Almas
Necmettin Erbakan University, Faculty of Dentistry, Department of Pediatric Dentistry

ABSTRACT: Changes in the number, size, shape, structure, and eruption of teeth constitute dental anomalies. Disorders that occur after teeth have completed their normal formation are called "acquired dental anomalies," while disorders that occur during the tooth formation process are defined as "developmental dental anomalies." Developmental anomalies can be caused by genetic factors such as heredity, metabolic disorders, and mutations, and/or environmental factors including physical, chemical, and biological factors. These anomalies can also occur as a symptom of a disease or syndrome, and early diagnosis can be significant for the treatment of the associated disease or syndrome.

KEYWORDS: Dental anomalies, Tooth development, Dental genetics

DENTAL ANOMALYS:

Significant changes in the structure and structure of teeth, such as the number, shape, and size of permanent and primary teeth, can occur. Dental anomalies can arise as a result of genetic, environmental, systemic, or local changes, or a combination of these. Anomalies during the shedding and eruption of primary teeth can provide examples for determining the eruption of permanent teeth [1].

1. NUMERICAL ANOMALIES OF TEETH

1.1. Hyperdontia

Hyperdontia is an anomaly characterized by an increase in the number of teeth. An excess of teeth may be termed supernumerary teeth, regardless of their location and morphology. This condition can be seen as single or multiple, unilateral or bilateral, erupted or impacted, in the primary or permanent dentition, and in one or both jaws [2]. The incidence of supernumerary teeth varies between 0.1% and 1.9% in the primary dentition and 0.1% and 3.6% in the permanent dentition [3]. In the permanent dentition, they are twice as common in males as in females. They are 8.2 to 10 times more common in the upper jaw than in the lower jaw. The most common supernumerary tooth is the mesiodentate tooth, which can be seen in various shapes and positions between the central incisors in the maxilla. This is followed by the fourth molars, premolars, and upper lateral teeth, respectively [4]. The probability of seeing a large number of supernumerary teeth is reported to be less than 1% in the literature. It is associated with systemic diseases such as Gardner syndrome, Ehlers-Danlos syndrome, cleidocranial dysplasia, cleft lip and palate, Ellis-van Creveld syndrome, Fabri-Anderon syndrome, incontinentia pigmenti, and megadontia [5].

1.2. Hypodontia

Hypodontia is the most common developmental dental anomaly. Hypodontia is the term used to describe the developmental absence of one or more primary or permanent teeth, excluding the third molar. Furthermore, the absence of six or more teeth is called oligodontia, and the absence of all teeth is called anadontia. The incidence of hypodontia can vary depending on ethnicity. This rate is 1% in Africans and Aboriginals, while it increases to 30% in Japanese. The third molar is the tooth with the highest incidence of agenesis. This is followed by the lower second premolars (2.8%), upper lateral teeth (1.6%), upper second premolars, and lower incisors (0.08%-0.23%) [6].

2. SIZE ANOMALIES

2.1. Macrodontia

Macrodontia (megadontia, megalodontia, macrodontism) is a term used to describe teeth that are larger than normal and are characterized by an increase in the mesio-distal and facio-lingual dimensions of the tooth [7]. The prevalence of permanent macrodontia varies between 1.1% and 1.9% depending on the population studied, with a higher incidence in the primary dentition. The prevalence of macrodontia is 1-2% in males and 0.9% in females, while macrodontia of the mandibular second premolars

affects both sexes equally. Macrodontia can be observed in three different types: Generalized macrodontia: In generalized macrodontia, all teeth in the dentition or the teeth in the lower jaw are larger than normal. This condition is often associated with certain systemic conditions and syndromes. General relative macrodontia: Although the teeth are of normal size, relative macrodontia exists due to a small maxilla and mandible. This can cause crowding and eruption disorders due to limited space in the dental arch [8]. Local macrodontia: This refers to macrodontia of a single tooth and is an extremely rare condition that can manifest as simple enlargement of all dental structures or may be associated with morphological anomalies.

2.2. Microdontia

Microdontia is a rare anomaly. It can be defined as the presence of abnormally small teeth. Diastema is inevitable in the presence of small teeth. This condition can primarily cause aesthetic concerns and functional difficulties such as food retention. Microdontia can accompany malocclusion problems in cases of impacted canines. When evaluating the prevalence of microdontia, most data in the literature relate to maxillary lateral incisors. While the prevalence is less than 0.5% in the primary dentition, it averages 2.0% in the permanent dentition. Microdontia is more common in women [9]. Microdontia can be seen in three different types:

Generalized microdontia: All teeth in the dentition are smaller than normal. It is observed in pituitary dwarfism and is extremely rare otherwise [9].

General relative microdontia: When a parent inherits a large jaw from one parent and small teeth from the other, due to hereditary factors, the teeth may appear to have general microdontia, even though they are normal or near-normal in size. In such cases, polydiastema occurs. Local microdontia: This is the appearance of a single tooth being smaller than normal and smaller than the other teeth. It is the most common volume anomaly in teeth. It is most commonly seen in maxillary lateral teeth (peg laterals), third molars, and supernumerary teeth [9].

3. TOOTH FORMATION ANOMALIES

Fusion: Fusion is the embryonic union of two or three developing tooth germs to form a single tooth. When fusion occurs before calcification, the resulting tooth is usually normal in size. However, fusion in later stages of development can result in a tooth twice the normal size or a tooth with a split crown. Fusion can occur between normal teeth or between normal and supernumerary teeth. It occurs more frequently in primary teeth and in the anterior region [10].

Gemination: Gemination is the inability to fully separate the bud of a single tooth attempting to divide, depending on the duration and severity of the local irritant reaching the region during development. Therefore, they contain a crown with an enlarged mesio-distal diameter, a single root, and a single canal. The crown parts separated by the developmental groove extending from the incisal edge to the cervical margin on the crown resemble each other as mirror images. There is no decrease in the number of teeth in the dental arch. However, considering that the number of teeth in the arch will not change as a result of the fusion of an extra tooth and a normal tooth, the appearance of the crown halves separated by the developmental groove must be evaluated to diagnose gemination [11].

Concrescence: The fusion of the roots of adjacent fully developed teeth with their cementum due to trauma and crowding. As the interdental bone between the roots resorbs under environmental pressure, the roots approach each other, resulting in fusion through cementum apposition. This can occur before or after the teeth eruption. Occasionally, the roots of three teeth may fuse. It is most commonly seen between the maxillary second and third molars [12].

Dilaceration: The root or crown of an immature tooth has a sharp, elbow-like curvature or curve. It is also called root angulation. While coronal angulations can be detected by clinical examination, radiographic examination is necessary to determine root angulations. The curvature or curvature can be cervical, mid-root, or apical. The two most frequently reported etiologies of dilaceration, one of the causes of impaction of permanent incisors, are traumatic injuries to primary teeth and ectopic development of the tooth germ. Trauma during tooth development causes the calcified portion of the tooth to shift position, allowing the remaining portion of the tooth to continue developing at an angle [13].

Hutchinson's Teeth: Due to congenital syphilis affecting the tooth germs during fetal life, the front teeth are somewhat smaller than normal. The marginal edges are converged toward the center, the lateral edges are curved, and the incisal edges are serrated. This shape resembles a spoon and is called "Hutchinson's teeth." The molars are called "mulberry molars." These teeth exhibit hypertrophic enamel, along with abnormal enamel structure and rounded cusps [14].

Accessory Tubercle: Accessory cusps are common on primary and permanent teeth. Accessory cusps are more common on permanent teeth. In the primary dentition, accessory cusps are seen on the mesiobuccal aspect of the maxillary first molar and on the mesiopalatinal aspect of the maxillary second molar. These accessory cusps can be compared to the Karabelli cusp seen on the maxillary first molar [15].

Talon Cubercule: The talon cusp is a dental anomaly seen in the cementoenamel junction or cingulum of maxillary and mandibular anterior teeth. Although composed of normal enamel and dentin, it can sometimes contain pulp tissue. Its eagle-claw appearance gives it the name talon cusp. It is most commonly seen in maxillary lateral teeth. The maxillary central incisor is the most commonly affected tooth in the primary dentition, while the maxillary lateral incisor is the most commonly affected tooth in the permanent dentition. Although its etiology is not fully understood, genetic and environmental factors are thought to influence its formation

[16]. It is thought to form during the morphological differentiation phase of tooth development, as a result of trauma or other localized injuries to the tooth germ. The larger talon cusp, located further from the tooth crown, has been reported to be more likely to contain pulp tissue [17].

Dens Invaginatus: It occurs when the enamel organ invaginates into the dental papilla before calcification is complete. The incidence ranges from 0.25% to 9.66%. It is less common in Black people. It is equally common in both sexes. Various etiological factors play a role in its formation. These include dislocation of the enamel organ relative to the dental papilla, excessive pressure from surrounding tissues during tooth development, and focal stimulation of the tooth bud in specific areas. Dens invaginatus is most commonly seen in the maxillary permanent lateral incisors [18].

Dens Evaginatus: Dens evaginatus is characterized by a cone-shaped accessory cusp on the occlusal surfaces of teeth. This cusp contains enamel, dentin, and often pulp tissue. The presence of pulp tissue is a key clinical distinction between this accessory cusp and the cusp of Carabelli. Early diagnosis and treatment of this cusp is crucial because trauma during mastication can cause abrasion or fracture of the cusp, leading to pulp necrosis and periapical infection. This anomaly is encountered in the primary and permanent dentition, most commonly in premolars [19].

4. ROOT MORPHOLOGY ANOMALIES

Taurodontism: Taurodontism manifests itself on radiographs with a vertically elongated pulp chamber, an apically displaced pulp floor, and two or three shorter than normal root/canal structures. The narrowing at the cementoenamel junction in normal teeth is reduced in taurodontized teeth, giving them a rectangular shape. Taurodontism can occur alone or in association with syndromes and anomalies such as amelogenesis imperfecta, Down syndrome, ectodermal disorders, Klinefelter syndrome, ora fascia-digital syndrome type 2, osteoporosis, and trichomoniasis. It can be seen in both the permanent and primary dentition, but permanent teeth are more commonly affected. Taurodont teeth, which can appear unilaterally or bilaterally, are more commonly encountered in molars [20].

Accessory Roots: Accessory roots can be found in any tooth. Tree-branch-like accessory roots can be seen in primary canines, permanent upper incisors, and mandibular and maxillary molars. They are very rare in permanent lower incisors and maxillary canines. They are common in mandibular canines, premolars, and molars, especially in the mandibular first molars. While accessory roots are generally seen distolingually, they can also be seen in bifurcation areas. Accessory roots can be thin, rounded, tapering toward the tip, or curved [21].

Hypercementosis: This is the accumulation of excessive layers of secondary cementum on the roots of one or more teeth. The exact cause is unknown, but it is thought to result from hypofunction associated with periapical inflammation and the absence of an antagonist tooth. In Paget's disease, widespread hypercementosis is observed in all teeth [22].

5. POSITIONAL ANOMALIES OF TEETH

Transposition: Tooth transposition is defined as the displacement between two adjacent teeth in the same half-jaw [16-17]. It is divided into two types: complete transposition, in which the crown and root of the tooth are displaced in the dental arch, and incomplete transposition, in which the root remains in its normal position but the crown is displaced. The incidence is around 0.4% [18]. Tooth transpositions are more common in the maxilla than in the mandible, and the upper permanent canines are the most frequently transposed teeth [23]. It can occur unilaterally or bilaterally, more commonly in women [24].

Ectopic Eruption: In ectopia, teeth are located on the jaws but away from the dental arches. For example, the maxillary canine erupts in the infraorbital region, the mandibular third molar in the condyle, and the maxillary central incisor in the nasal floor region. Ectopic teeth may remain impacted or erupt like normal teeth. The frequency of ectopic eruption is, respectively, maxillary permanent molars and canines, mandibular second premolars and canines, and maxillary lateral incisors. The prevalence of ectopic eruption of maxillary permanent first molars varies between 3-6%. Ectopic eruption of canines occurs in 1.5-2% of the population and is more common in males at a ratio of 2:1 [25]. Factors causing ectopic eruption include: These may include bone-tooth size discrepancies or anomalies in the crown morphology of the primary second molar, ankylosing of the primary teeth, chronological changes in the growth of bone in the tuber region associated with calcification and eruption of the molar, abnormal eruption angle of the permanent first molar, and supernumerary teeth [26].

Heterotopia: Heterotopia is the development of a tooth within an organ other than the jaw. For example, teeth can be found within the orbital cavity, within dermoid ovarian cysts, and alongside other epidermal formations [27].

Displacement: Displacement occurs when a tooth is located within the jaw arch but not in its normal location. For example, maxillary canines are in a vestibular or palatal position, or premolars are in a palatal/lingual position [28].

Inversion: Inversion occurs when a tooth completely reverses its normal eruption direction. These teeth can erupt into the nasal cavity, sinus, and orbit. This type of anomaly is frequently encountered in supernumerary teeth and wisdom teeth [29].

Diastema Between The Upper Central Incisors: When the upper permanent incisors erupt, a diastema sometimes forms between them, which gradually closes as eruption is completed. The persistence of this diastema is pathological. Local causes include the presence of mesiodens and hypertrophy of the upper lip frenulum, while general causes include rickets, hormonal disorders, heredity,

and cleft lip and palate. If a supernumerary tooth is present, extraction is mandatory. Removal of the frenulum or attempts to close the diastema through orthodontic means are not always successful [30].

6. STRUCTURAL ANOMALYS

Structural anomalies of teeth are examined under four headings: enamel defects, dentin defects, cementum defects, and odontodysplasia.

6.1. Enamel Defects

Structural anomalies of enamel can occur due to genetic or environmental factors, resulting in hypomineralization due to insufficient mineralization of matrix proteins, or hypoplasia due to a defect in matrix production. Enamel can be primarily affected as a result of genetic influences, as in amelogenesis imperfecta. Enamel anomalies can also result from many acquired environmental and systemic factors, such as metabolic conditions, infections, drugs, chemicals, radiation, and trauma. Additionally, congenital anomalies that affect mineralization pathways, such as parathyroid gland disorders, can also cause enamel anomalies. Localized enamel defects are usually seen in permanent teeth due to trauma or infection in primary teeth [31].

6.1.1. Amelogenesis Imperfecta

Amelogenesis imperfecta is a genetic disorder characterized by enamel formation disorders affecting all teeth, affecting both dentitions. Disease classification is based on clinical appearance and heredity. According to the most accepted classification currently, there are four types of amelogenesis imperfecta and subgroups within these types [32].

Type I (Hypoplastic Type) AI: This describes thin but mineralized translucent enamel. In some cases, it results from problems in the secretion phase, characterized by complete absence of enamel. During this phase, ameloblasts undergo a transformation, reducing enamel protein secretion. Proteinases are secreted in place of structural proteins (amelogenin, ameloblastin, enamelin, amelotin, tuftelin, and dentinal phosphoprotein), and the organic matrix is destroyed. As a result of these changes, enamel crystals cease to grow, and the enamel becomes thinner than normal. The teeth are yellowish-brown, with rough and pitted surfaces. Radiographically, like normal enamel, they are more radiopaque than dentin [33].

Type II (Hypomaturated Type) AI: Enamel is of normal thickness. It forms as a result of inadequate degradation of proteins in the enamel matrix during the maturation phase. The enamel has a mottled dark brown-yellow appearance. It is slightly softer than normal enamel. Because the enamel tissue has not yet fully matured, severe wear and tear and fractures can occur. Radiographically, it exhibits a radiodensity similar to dentin [33].

Type III (Hypocalcified Type) AI: Enamel calcification is problematic. The enamel is still of normal thickness but very soft and is destroyed immediately after tooth eruption. It also results from inadequate calcium (Ca+2) ion transport during the maturation phase. It has an opaque or chalky appearance. Rapid destruction is observed in the teeth. Sensitivity to thermal stimuli is very high. Radiographically, enamel is less radiopaque than dentin. In some cases, it is quite difficult to definitively differentiate between hypomature and hypocalcified amelogenesis imperfecta. Therefore, both types can be referred to collectively as hypomineralized amelogenesis imperfecta [33].

Type IV (Hypomature-Hypoplasic Type Seen With Taurodontism) AI: A mixture of hypoplastic and hypomature types is observed (Figure 16). Radiographs show signs of taurodontism, with the pulp chamber being excessively enlarged and the furcation point approaching the apical part of the root [33].

6.1.2. Systemic Enamel Defects

Systemic factors that can affect enamel development can occur pre-, peri-, and postnatally, depending on the timing of the event. Systemic factors can be classified as metabolic disorders, infections, chemicals, and medications. Prenatal factors that can cause enamel hypoplasia include maternal smoking and vitamin D deficiency during pregnancy and the neonatal period, while postnatal factors include nutritional deficiencies. Preterm infants and those with low birth weight have a higher prevalence of enamel hypoplasia than children born with normal birth weights. Defects found in preterm children often result from systemic conditions related to preterm birth, such as respiratory problems, cardiovascular, gastrointestinal, and renal abnormalities, intracranial hemorrhage, and anemia. Additionally, hypocalcemia, osteopenia, and hyperbilirubinemia may increase the risk of enamel defects in preterm children [34].

6.1.3. Localized Enamel Defects

Unlike systemic factors, which generally affect all developing teeth, local factors such as trauma affect only the teeth in the immediate vicinity of the injury. For example, trauma to the maxillary alveolus during laryngoscopy in a newborn can cause localized defects ranging from mild enamel opacities to severe enamel hypoplasia and even dilaceration. Similarly, trauma to the thin buccal cortical bone is thought to be the cause of the circumscribed opacities commonly seen on the labial surfaces of primary canines.

6.2. Dentin Defects

Dentin defects can occur due to genetic and environmental factors. The most common genetically transmitted dentin defects are dentinogenesis imperfecta and dentin dysplasia. Dentin defects can be seen in association with syndromes such as osteogenesis imperfecta and Ehlers-Danlos syndrome.

6.2.1. Dentinogenesis Imperfecta

Dentinogenesis Imperfecta (DI) is defined as an autosomal dominant genetic disease characterized by abnormal dentin structure affecting both primary and permanent teeth. DI can occur alone or in association with a systemic hereditary bone disease known as osteogenesis imperfecta (OI). DI diagnosed in children with unexplained bone fractures may be an indicator of an undiagnosed case of OI [35].

Dentinogenesis Imperfecta Type I (DI-I): This is the syndromic form of DI that occurs in association with osteogenesis imperfecta. It has been reported that DI is the most prominent finding in some cases of osteogenesis imperfecta. Both primary and permanent dentition can be affected. Primary teeth are most commonly affected, followed by permanent first molars and incisors. Radiographically, a bell-shaped, bulbous crown, narrowing of the cervical region, short and thin roots, and narrowing and obstruction of the root canal and pulp chamber due to excessive dentin production are observed. Periapical radiolucencies and root fractures may occur. The teeth appear gray-blue, yellow-brown, or transparent. Due to changes in the enamel-dentin junction, hypomineralized dentin is exposed instead of enamel and is highly susceptible to abrasion. This condition is even more pronounced in primary teeth. Infection may develop in primary teeth due to abrasion, and abscesses may occur [36].

Dentinogenesis Imperfecta Type II (DI-II): This is a non-syndromic type of DI, also known as hereditary opalescent dentin. Both tooth rows are affected equally. Its characteristics are consistent with DI-I. When viewed with transillumination, the teeth are transparent and bluish-brownish. As in DI-I, primary teeth are more severely affected than permanent teeth, while the final permanent teeth are least affected. On radiographic examination, shrinkage of the pulp chamber can be observed from the moment the tooth erupts. Gum disease can be observed even in the absence of tooth decay. Following eruption, the enamel of the permanent dentition may appear normal, but histological studies have shown hypomineralized areas in the enamel in approximately one-third of cases [36].

Dentinogenesis Imperfecta Type III (DI-III): This is less common and affects both tooth rows. In DI-III, in addition to the typical color and size changes, bell-shaped crowns and enamel pits are particularly prominent in the permanent teeth. Clinically, it is very similar to D1-II. The main difference from D1-I and D2 is the appearance of "ghost (shell) teeth" on radiographs due to excessive pulp expansion. In primary teeth, the pulp chamber is greatly enlarged and the dentin is extremely thin, resulting in a normal-thin enamel structure and a blue-gray color. The pulp has extended to the occlusal dentin and can be exposed very quickly. In genetically determined dentin abnormalities, the severity of the damage can vary from individual to individual [36].

6.2.2. Dentin Dysplasia

Dentin dysplasia, a rare anomaly, is an autosomal dominant disease affecting the primary dentition, the permanent dentition, or both dentitions in one in every 100,000 patients. Based on radiological findings, Witkop in 1972 classified dentin dysplasia into two types: type I: Radicular dentin dysplasia and type II: Coronal dentin dysplasia. Type I dentin dysplasia is characterized by normal-appearing crowns or may have a slightly amber hue due to abnormal root development, reduced pulp space in permanent teeth, and abnormal growth of dentin adjacent to the pulp chamber, leading to partial or total obliteration of the pulp chambers. Periapical radiolucency or cysts that can lead to premature tooth loss may also be observed. Type II dentin dysplasia is characterized by yellow, brown, or gray-amber primary teeth with total pulpal obliteration. Permanent teeth may have a normal appearance or be amber in color. Roots are normal in size and shape, contain pulp stones, and are characterized by a "thistle tube"-shaped pulp chamber. Obliteration of the pulp chamber occurs after tooth eruption [37].

6.3. Cement Defects

Cementation defects are seen in association with genetically transmitted diseases. Bone sialoprotein (BSP) is a multifunctional extracellular matrix protein found in mineralized tissues, including bone, cartilage, cementum, and dentin. Deficiencies in the secretion of this protein cause cementation defects. Alkaline phosphatase is an ectoenzyme and is present in high concentrations in mineralizing tissues. Cementation defects are also observed in cases of hypophosphatasia, where alkaline phosphatase enzyme secretion is impaired. Early and spontaneous tooth loss may be an indicator of the disease. Prosthetic rehabilitation should be considered at an early age to maintain aesthetics and function [38].

6.4. Regional Odontodysplasia

Regional odontodysplasia is a rare developmental dental anomaly originating from both the mesoderm and ectoderm. Its etiology remains unknown, but viral infections, local trauma, vascular defects, x-rays, metabolic disorders, Rh incompatibility, and medications taken during pregnancy are considered possible causes. In some patients, it may be accompanied by facial asymmetry. Diagnosis is primarily based on clinical and radiographic findings, and sometimes histological findings [39].

7. ENDOGENOUS TOOTH DISCOLORATION

Intrinsic staining is a structural discoloration that affects the optical properties of enamel and dentin tissue, resulting from various factors that occur during tooth development. Intrinsic discoloration due to systemic factors:

Phenylketonuria (Alkaptanuria): This is a recessive metabolic disease. Hemogentistic acid, produced by a defect in tyrosine and phenylalanine oxidation, causes brown discoloration of the dentin [40].

Porphyria (Gunter's Disease): This is an autosomal recessive hereditary disorder characterized by pigmented bullae that leave scars. Hematoporphyria, resulting from a defect in porphyrin metabolism, is deposited in the bones and teeth. It affects both the primary and permanent dentition. The teeth present with a dark pink discoloration [40].

Erythroblastosis Fetalis (Neonatal Jaundice): This is a hemolytic disorder seen in newborns. Biliveridin pigment, released into the circulation as a result of erythrocyte agglutination and hemolysis, causes jaundice and stains developing teeth greenish-blue, bluish-black, or Brown [40].

Fluorosis: Dental fluorosis is a developmental anomaly of enamel caused by the deterioration of enamel formation in ameloblasts observed in teeth exposed to high concentrations of fluoride during development. The severity of fluorosis varies depending on the dose and duration of exposure, as well as individual characteristics. The effects on enamel structure are increased porosity and low mineral content. The discolorations observed in fluorosis are symmetrical. Depending on the severity of the case, discolorations can range from chalky white opaque patches that develop after teeth erupt to yellow-brown stripes. Tetracycline Staining: Tetracycline, a broad-spectrum antibiotic, can reach the pulp and dentin via the bloodstream and cause tooth discoloration. This effect occurs by forming a tetracycline-calcium-orthophosphate complex in dentin due to its high affinity for calcium. The severity of discoloration depends on the drug's dose, type, duration of use, and the tooth's developmental stage at the time of administration. Tetracycline, oxytetracycline, and dimethyltetracycline stain teeth in yellow-brownish-gray tones, while chlortetracycline stains teeth gray. Because tetracycline molecules can cross the placental barrier, the period from the fourth month of pregnancy, when primary teeth begin to calcify, to age 8, when calcification of permanent tooth crowns is complete, is a risky period for tetracycline discoloration. Therefore, tetracycline should not be used in pregnant women, breastfeeding women, or children at least until the age of 7-8. The typical appearance of tetracycline discoloration is band-like discoloration [41].

Pulp Necrosis: Necrosis of the pulp due to bacterial, mechanical, or chemical irritants can cause the release of harmful byproducts that can penetrate the tubules and change the color of the surrounding dentin. The degree of discoloration is directly proportional to the duration of necrotic tissue residue in the pulp cavity. This discoloration can often be removed with intracoronal bleaching [42].

Intrapulpal Hemorrhage: Hemorrhage can occur in the pulp tissue following pulp extirpation or severe dental trauma. Discoloration of the adjacent dentin can occur due to the passage of blood components into the dentin tubules. While a pink discoloration is observed in the crown in the early stages, the iron released by the destruction of red blood cells is converted to iron sulfate by bacteria in the later stages. This causes a gray discoloration of the tooth. If pulp necrosis occurs as a result of trauma, the discoloration gradually increases [42].

Pulp Tissue Residuals İn Endodontically Treated Teeth: Similar to intrapulpal hemorrhage, tissue remnants remaining in the pulp chamber after pulp extirpation can cause discoloration. Preparing a suitable access cavity and completely removing the coronal pulp is important in preventing this condition. Intracoronal bleaching is a successful treatment.

Endodontic Material: Failure to completely remove root canal filling material residue from the pulp chamber can cause discoloration in endodontically treated teeth. This frequently encountered condition can be prevented by removing all material above the bone level [42].

Coronal Filling Materials: In older composite resin restorations, microleakage can cause dark discoloration at the margins and, over time, in the tooth tissues. Dark gray discoloration due to metallic components can also be observed in teeth restored with amalgam [42].

Root Resorption: Although root resorption is clinically asymptomatic in its early stages, it may exhibit a pink appearance at the cementoenamel junction [42].

Dystrophic Calcification: Secondary dentin, which forms physiologically with aging, affects the optical properties of the tooth. A gradual darkening and increased opacity of the tooth are observed due to the narrowing of the pulp space [42].

CONCLUSION

Dental anomalies can persist in the mouth for extended periods without any clinical symptoms and can cause various problems. Developmental anomalies of the enamel can cause problems such as tooth sensitivity and susceptibility to decay, while impaction, missing or supernumerary teeth can lead to orthodontic problems. They can also create aesthetic problems. Early diagnosis of dental anomalies and appropriate treatment planning after a comprehensive evaluation are crucial to reduce the need for longer, more costly treatments later. Only in cases where follow-up is recommended should patients be referred for routine checkups.

REFERENCES

1) Safety, R., & In, P. (2008). White SC, Pharoah MJ: Oral Radiology Principles and Interpretation.
2) Puri, K., Bansal, M., Jain, D., Kumar, A., Khatri, M., & Gupta, G. (2013). Nonsyndromic Multiple Supernumerary Premolars And Paramolars: An Overview And Report Of 2 Cases. Indian Journal of Dental Sciences, 5(5).
3) Kasat, V. O., Saluja, H., Kalburge, J. V., Kini, Y., Nikam, A., & Laddha, R. (2012). Multiple bilateral supernumerary mandibular premolars in a non-syndromic patient with associated orthokeratised odontogenic cyst-A case report and review of literature. Contemporary Clinical Dentistry, 3(Suppl 2), S248-S252.

4) Salcido-García, J. F., Ledesma-Montes, C., Hernández-Flores, F., Pérez, D., & Garcés-Ortíz, M. (2004). Frequency of supernumerary teeth in Mexican population. Medicina Oral, Patologia Oral y Cirugia Bucal, 9(5), 407-9.
5) Nayak, G., Shetty, S., Singh, I., & Pitalia, D. (2012). Paramolar–A supernumerary molar: A case report and an overview. Dental Research Journal, 9(6), 797.
6) Al-Ani, A. H., Antoun, J. S., Thomson, W. M., Merriman, T. R., & Farella, M. (2017). Hypodontia: an update on its etiology, classification, and clinical management. BioMed research international, 2017(1), 9378325.
7) Garib, D. G., & Peck, S. (2006). Extreme variations in the shape of mandibular premolars. American journal of orthodontics and dentofacial orthopedics, 130(3), 317-323.
8) McKinney, R., & Olmo, H. (2023). Developmental disturbances of the teeth, anomalies of structure. In StatPearls [Internet]. StatPearls Publishing.
9) Jurado, C. A., Villalobos-Tinoco, J., Alshabib, A., & Afrashtehfar, K. I. (2024). Advanced restorative management of focal microdontia: A brief review and case report. Dental and medical problems, 61(3), 455-462.
10) Goswami, M., & Lohia, S. (2024). Exploring Dental Fusion in Primary Dentition: A Pediatric Dental Perspective. Cureus, 16(9).
11) Altun, C., Güven, G., Başak, F., Akbulut, E., & Altuğ, H. A. (2005). Süt dişlerinde füzyon ve geminasyon: Beş olgu nedeniyle. European Annals of Dental Sciences, 32(3), 223-227.
12) Consolaro, A., Hadaya, O., Miranda, D. A. O., & Consolaro, R. B. (2020). Concrescence: can the teeth involved be moved or separated?. Dental press journal of orthodontics, 25, 20-25.
13) de Amorim, C. S., Americano, G. C. A., Moliterno, L. F. M., de Marsillac, M. D. W. S., Andrade, M. R. T. C., & Campos, V. (2018). Frequency of crown and root dilaceration of permanent incisors after dental trauma to their predecessor teeth. Dental Traumatology, 34(6), 401-405.
14) Sundararajan, D. (2025). Developmental Defects of Teeth. In Oral Histology and Oral Histopathology: A Practical Guide for Dental Students and a Companion to Pathologists (pp. 169-179). Cham: Springer Nature Switzerland.
15) Haberal, M., & Bayraktar, Y. (2024). Paramolar Tubercles (Bolk Cusps): Two Case Reports.
16) Çelenk, P. Maksiller 2. Molar Dişte İzlenen Dens Invajinatus: Olgu Sunumu.
17) Almak, Z., & Kotanlı, S. Talon Tuberculosis and Conservative Approaches: Two Case Reports. HRU International Journal of Dentistry and Oral Research, 2(1), 78-81.
18) Siqueira Jr, J. F., Rôças, I. N., Hernández, S. R., Brisson-Suárez, K., Baasch, A. C., Pérez, A. R., & Alves, F. R. (2022). Dens invaginatus: clinical implications and antimicrobial endodontic treatment considerations. Journal of endodontics, 48(2), 161-170.
19) Lerdrungroj, K., Banomyong, D., Songtrakul, K., Porkaew, P., & Nakornchai, S. (2023). Current management of dens evaginatus teeth based on pulpal diagnosis. Journal of Endodontics, 49(10), 1230-1237.
20) Chetty, M., Roomaney, I. A., & Beighton, P. (2021). Taurodontism in dental genetics. BDJ open, 7(1), 25.
21) Bueno, M. R., Azevedo, B. C., Estrela, C. R. D. A., Sousa-Neto, M. D., & Estrela, C. (2021). Method to identify accessory root canals using a new CBCT software. Brazilian Dental Journal, 32(6), 28-35.
22) Massé, L., Garot, E., Maureille, B., & Le Cabec, A. (2023). Insights into the aetiologies of hypercementosis: A systematic review and a scoring system. Archives of Oral Biology, 146, 105599.
23) Çam, K., & Zengin, A. Z. (2024). Prevalence of tooth transpositions and associated dental anomalies: A CBCT study. J Clin Images Med Case Rep, 5(10), 3316.
24) Tank, U., Saraf, T., & Jawdekar, A. (2022). Transposition of lateral incisor and canine in two sisters-a case report and literature review. Spec Ugdym, 2(43), 238-47.
25) Güven, Y. (2018). Prevalence of ectopic eruption of first permanent molars in a Turkish population. European Oral Research, 52(1), 1-5.
26) Demirel, T., & Bodrumlu, E. H. (2019). Sürme Anomalileri. Journal of International Dental Sciences (Uluslararası Diş Hekimliği Bilimleri Dergisi), (3), 141-146.
27) Kumar, D., Mahajan, D., & Sachdeva, J. (2013). Heterotopia. Journal of Evolution of Medical and Dental Sciences, 2(45), 8701-8704.
28) Laganà, G., Venza, N., Borzabadi-Farahani, A., Fabi, F., Danesi, C., & Cozza, P. (2017). Dental anomalies: prevalence and associations between them in a large sample of non-orthodontic subjects, a cross-sectional study. BMC oral health, 17(1), 62.
29) Tuna, E. B., Kurklu, E., Gencay, K., & Ak, G. (2013). Clinical and radiological evaluation of inverse impaction of supernumerary teeth. Medicina oral, patologia oral y cirugia bucal, 18(4), e613.
30) Abrahams, R., & Kamath, G. (2014). Midline diastema and its aetiology–a review. Dental update, 41(5), 457-464.
31) McCauley, L. K., & Martin, T. J. (2012). Twenty-five years of PTHrP progress: from cancer hormone to multifunctional cytokine. Journal of Bone and Mineral Research, 27(6), 1231-1239.

32) Crawford, P. J., Aldred, M., & Bloch-Zupan, A. (2007). Amelogenesis imperfecta. Orphanet journal of rare diseases, 2(1), 17.
33) Gadhia, K., McDonald, S., Arkutu, N., & Malik, K. (2012). Amelogenesis imperfecta: an introduction. British dental journal, 212(8), 377-379.
34) Kim Seow, W., Ford, D., Kazoullis, S., Newman, B., & Holcombe, T. (2011). Comparison of enamel defects in the primary and permanent dentitions of children from a low-fluoride District in Australia. Pediatric dentistry, 33(3).
35) Council, O. (2013). Guideline on dental management of heritable dental developmental anomalies. Pediatr Dent, 35(5), 179-84.
36) Council, O. (2013). Guideline on dental management of heritable dental developmental anomalies. Pediatr Dent, 35(5), 179-84.
37) Witkop Jr, C. J. (1975). Hereditary defects of dentin. Dental Clinics of North America, 19(1), 25-45.
38) Foster, B. L., Nagatomo, K. J., Tso, H. W., Tran, A. B., Nociti Jr, F. H., Narisawa, S., ... & Somerman, M. J. (2013). Tooth root dentin mineralization defects in a mouse model of hypophosphatasia. Journal of Bone and Mineral Research, 28(2), 271-282.
39) Crawford, P. J., & Aldred, M. J. (1989). Regional odontodysplasia: a bibliography. Journal of Oral Pathology & Medicine, 18(5), 251-263.
40) Manuel, S. T., Abhishek, P., & Kundabala, M. (2010). Etiology of tooth discoloration-a review. Nigerian Dental Journal, 18(2), 56-63.
41) Aoba, T., & Fejerskov, O. (2002). Dental fluorosis: chemistry and biology. Critical Reviews in Oral Biology & Medicine, 13(2), 155-170.
42) Greenwall, L. (2017). Discoloration of teeth. In Tooth Whitening Techniques (pp. 1-19). CRC Press.