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Unveiling The Anomalies Of The Teeth Based On Size And Shape- A Brief Review Literature

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ABSTRACT:

The formation of the human primary and permanent dentition is a strictly regulated process. A spatiotemporal cascade of signalling pathways leads to the development and eruption of the human dentition. Any irregularities in the shape, size, quantity, and position of teeth might cause deviations from their normal development. This chapter covers all developmental dental anomalies in both dentitions, emphasising the genetic, systemic, environmental, and local variables that contribute to them. A overview of the findings, clinical consequences, and current treatments is provided for each aberration. Dentists should be aware of these developmental dental defects because they may be connected to systemic diseases or disorders.

Keywords: primary and permanent dentition, dental malformations, tooth abnormalities, and developmental disturbances

INTRODUCTION:

The formation of the tooth, a specialized component of the human body, is mysterious and difficult to grasp. A complicated reciprocal relationship between the underlying ectomesenchyme and dental epithelium is necessary for a tooth to form successfully[1]. Numerous intricate chemical signals, receptors, and transcription regulatory systems are involved in the interaction. A variation from what is considered normal is called an anomaly (Gk, anomalos; irregular). Teeth developing abnormalities can result from disruptions of the epithelial and mesenchymal interactions, which can significantly change normal odontogenesis. Anomalies of quantity, structure, size, and/or shape may occur, depending on the developmental period at which the alteration may have occurred.[2]

DEVELOPMENTAL DISTURBANCES IN SIZE OF TEETH

GEMINATION:

The maxillary anterior region[3] [4], gemination—also known as double teeth, double formations, linked teeth, fused teeth, or dental twinning—occurs frequently.
Geminated teeth develop from either a big, partially separated crown with a single root and root canal , which may be validated radiographically, or an effort to divide a single tooth germ by invagination, resulting in a single tooth with two totally split crowns. The abnormal tooth is numbered[4] and has a greater mesial-distal diameter than usual. Nonetheless, the dental arch's overall tooth count of [4] is typical.

Although the exact cause of geminated teeth is unknown, factors such as dietary deficiencies, hormonal influences, viral or inflammatory processes, excessive medication use, inherited or congenital illnesses, local trauma.

FUSION:

The merger of the dentin and enamel of two or more distinct growing teeth is known as fusion, according to Pindborg [5]. Complete union of crowns or union of roots only can result in the formation of one excessively big tooth. With a preference for the anterior area [4], this anatomic abnormality is more common in deciduous dentition.

Depending on the developmental stage, fusion can be either partial or late or complete (total/true fusion) [6][4],. The teeth will fully combine and the crown will incorporate the pulp, cementum, dentin, and enamel of both teeth if fusion starts before the calcification stage. Later stages of incomplete fusion might result in a tooth with fused or separate pulp canals, distinct crowns, and only the root.

The etiology of fusion has been explained by a number of theories. It has been proposed that the physical pressure or force created during growing causes the tooth germs to come into touch and fuse when they are close to one another. The usage of thalidomide or the presence of a viral infection during pregnancy were proposed by other theories . There has also been discussion of a genetic etiology . Fusion has been documented in Xlinked congenital diseases as well as congenital defects such as cleft lip. Supernumerary teeth, hypodontia, peg-shaped incisors, dens in dente, nail problems, syndactyly, successional conical teeth, macrodontia, and double permanent teeth are among the dental and non-dental anomalies that have been linked to fusion [8].

CONCRESCENCE:

Concrescence is the cementum's ability to fuse neighboring pre-existing teeth together. It could happen before or after the eruption. In this type of fusion, just the cementum holds the teeth together. It is believed to be caused by dental crowding or trauma.[5]

Clinical significance: This problem is not very important until you try to pull one of the affected teeth. Again, radiography must be done before any extraction is done.[6]

Due to crowding, fusion may result in occlusal disruptions and cosmetic issues.

DILACERATION:

A severe bend, curvature, or angulation in a tooth's root or crown is referred to as dilatation. Acute mechanical stress during tooth development is typically the cause, changing the position of the calcified section and causing the remaining tooth to form at an angle. Anywhere along the tooth's length, there could be a bend or curve. Only a small percentage of cases are thought to involve hereditary factors.[10]

Clinical significance: A crown that is displaced could be an aesthetic issue. When a root is dilacerated,

extraction or endodontic therapy may be challenging. Teeth that are severely dislocated might not be able to erupt.

DENS INVAGINATUS:

There are very few occurrences of this rare tooth abnormality reported in the veterinary literature.[11]It symbolizes the invagination of dentin and enamel into the tooth pulp. It might be deep (crown and root) or superficial (crown). The condition's genesis is uncertain. Up to 5% of people have the mild version of the disease.[10]

Clinical significance: Depending on the extent of the lesion, which might range from pulpal necrosis and periapical inflammation to increased susceptibility to caries.

DEVELOPMENTAL DISTURBANCES IN SIZE OF TEETH:

MICRODONTIA:

Teeth that seem smaller than the typically anticipated differences in tooth size are referred to as microdontia. Microtodontia has a genetic basis, as evidenced by its etiology, which includes disruptions in the odontogenesis process and familial inheritance patterns. In other situations, isolated, spontaneous cases that have no known origin may arise. In decreasing order, third molars, supernumerary teeth, and maxillary lateral incisors are the teeth most commonly impacted by microdontia . This disorder may affect all of the teeth (generalized) or only one tooth or a small group of teeth (localized) [12]. The tooth that is impacted by localized microdontia may present with a changed form in addition to being decreased in size.

When microdontia affects every tooth, it can be categorized as either relative or pseudo generalized microdontia, in which the affected dentition appears smaller than normal because of the abnormally enlarged size of the maxilla or mandible, or true generalized microdontia, in which all affected teeth appear smaller than normal [12]. Rare cases of true widespread microdontia have been documented in people with pituitary dwarfism [13]and pediatric kids undergoing chemotherapy or radiation therapy during tooth development [14]. Down syndrome, Hallerman-Streiff syndrome, and Williams syndrome[15] are further disorders linked to both localized and generalized microdoria [15]. Additionally, there is a strong correlation between microdontia and other dental developmental abnormalities including hypodontia. A long-term comprehensive interdisciplinary approach involving restorative, orthodontic, surgical, and prosthetic treatments to create a more normal shape of the affected tooth, thereby improving the overall esthetics, or conservative (esthetic or restorative treatment) management of microdontia, depending on the severity of the condition [14] . A thorough, long-term, multidisciplinary strategy that includes prosthetic, orthodontic, restorative, and surgical procedures to improve the overall appearance of the damaged tooth by giving it a more normal form [16].

MACRODONTIA:

When a single tooth or collection of teeth present with a greater than typical size, it is referred to as macrodontia, megadontia, or megalodontia. It is still unclear what causes macrodontia. Macrodontia is classified similarly to microdontia: real generalized (all teeth are larger than normal), localized or isolated (single tooth or groups of teeth), and relative or faux generalized (all teeth are either normal or slightly larger than relatively smaller jaws). A rare disorder known as isolated macrodontia is more common in mandibular third molars, permanent incisors, and canines. Hemifacial hypertrophy is a unilateral presentation of macrodontia involving discrete groups of teeth, where the teeth on the affected side appear abnormally larger than the unaffected side.

Generalized macrodontia is an uncommon disease associated with pituitary gigantism. Oto-dental syndrome, KBG syndrome, XYY syndrome, and insulin-resistant diabetes are other systemic diseases linked to macrodontia [17]

. Macrodontia affecting permanent teeth has an overall reported prevalence of 0.03–1.9%, with a higher incidence in men [18]. A macrodont tooth's clinically larger crown size is visible when it erupts, but its deformed appearance may mimic gemination or fusion. In some cases, the dentist may take the proper radiograph following a clinical observation of an unerupted tooth in order to identify an unerupted macrodont, necessitating additional procedures, such as primary tooth extractions to facilitate eruption.The treatment options are

- Dependent on the size and shape of the affected tooth .
- Teeth and the related functional or esthetic concerns.
- Complex endodontic, surgical, and restorative procedures
- May be performed to restore esthetics and function.

TAURODONTISM:

A form abnormality known as taurodontism results from improper Hertwig's epithelial root sheath invagination. This leads to the anomaly's distinctive characteristics, which include an expanded pulp chamber, pulpal floor apical displacement, and a lack of constriction at the cemento-enamel junction. The Latin word "tauros," which means "bull," and the Greek word "odus," which means "tooth," are the sources of the term "taurodontism" (bull tooth) [19].

In order of increasing severity, there are three forms of taurodontism: hypotaurodontism, mesotaurodontism, and hypertaurodontism [20]. Only the pulp chamber is enlarged in the mild form of hypotaurodontism, while the crown and pulp chamber of the affected tooth are disproportionately enlarged in comparison to the roots, causing the root furcation to occur in close proximity to the root apices in the most severe form, hypertaurodontism. Since the damaged teeth typically present with a clinically normal morphological look of the crown, radiographic imaging is used to determine taurodontism . The tooth's tapered rectangular shape (tapering towards the roots), the pulp chamber's expanded appearance, and the root furcation's apical displacement are among the radiographic findings. There is no discernible gender bias in the prevalence rate of taurodontism affecting permanent dentition, which has been estimated to range from 2.5% to 11.5% [21]. Taurodontism may manifest alone or in combination with other developmental disorders or syndromes, such as hypophosphatasia, Van der Woude syndrome, Down syndrome, Klinefelter syndrome, and Tricho-Dento-Osseous syndrome [21]. Clinical issues linked to taurodontism include an increased risk of pulpal exposure during dental procedures or if the tooth is impacted by dental caries because of the expanded pulp chamber. When endodontic therapy is necessary, it can be quite difficult for the endodontist and may need for a different filling technique in order to achieve sufficient obturation [22]. Taurodontism can occasionally cause issues during extractions or post-core prosthodontic treatment.

HYPODONTIA:

A dental anomaly known as hypodontia occurs when six teeth—aside from third molars—are absent. About one fifth of people have it, making it the most prevalent dental oddity. Different populations have varying prevalences of hypodontia . Compared to men, women are more frequently impacted. Nonetheless, research indicates that it is more prevalent in men . Less than 1% of Caucasian populations have hypodontia in their main teeth. Hypodontia frequently affects both the mandibular central incisors and the primary maxillary

incisors . Agenesis of the permanent successor is frequently the result of agenesis of the primary teeth. Hypodontia is more prevalent in the stages of permanent teeth. The mandibular second premolar and lateral incisors are the teeth that are most frequently lost during permanent hypoodontia . Even when all of the primary teeth are present, hypodontia may still exist in the phases of the permanent dentition. Compared to unilateral agenesis, bilateral agenesis is more prevalent . Down syndrome, ectodermal dysplasia, Pierre-robin sequence, Van der Woude syndrome, and cleft lip and palate can all be associated with either syndromic or non-syndromic hypodontia. Dental rotations, ectopic eruption of neighboring teeth, and gaps between teeth can all result from hypodontia. One potential cause of hypodontia is the agenesis of tooth buds. Hypodontia may result from some regions of the dental lamina being vulnerable to environmental and polygenetic influences.

It is advised to undergo prosthetic rehabilitation, which includes replacing lost teeth. Treatment planning can be staggered to include temporary treatments such a resin-supported bridge (Carolina bridge) or a Maryland bridge , depending on the patient's age, available space, and future cosmetic needs. Depending on the patient's age, future permanent restorative solutions incorporating crowns supported by implants can be planned. In the past, space closure or canine replacements for missing lateral incisors have been used, depending on the skeletal or orthodontic findings .

HYPERDONTIA:

Hyperdontia is defined as the presence of a tooth or Teeth in addition to the full complement of permanent or Primary teeth. The erupted or unerupted additional tooth Is termed as a supernumerary tooth. Hyperdontia can be Present unilaterally or bilaterally. Supernumerary teeth Are most commonly noted in the anterior region of the Maxilla. The prevalence of hyperdontia in the permanent Dentition Is Approximately 2% [23]. Localized Hyperactivation of dental lamina is considered as possible Etiologic basis for development of hyperdontia . Supernumerary tooth/teeth can be classified based on the Number of extra teeth, shape, size, site and laterality [24] Atavism theory, dichotomy theory, and the theory of the epithelial remnant of dental lamina are further potential explanations for the origin of hyperdontia, in addition to the dental lamina hyperactivation theory . According to atavism hypothesis, humans may gain extra teeth as a result of phylogenetic reversion to primates, which have three pairs of incisors. According to the dichotomy theory, a single tooth bud might divide into two equal (twin) or uneven (rudimentary) pieces, which may result in the development of an extra tooth or teeth. The most widely accepted explanation for the emergence of hyperdontia is the presence of epithelial remains of the dental lamina .When erupted or unerupted extra teeth are found during a clinical or radiographic examination, hyperdontia is clinically diagnosed . Regarding the care of extra teeth, there are no established rules or opinions . Clinically, crowding, displacement, or root resorption of erupted teeth can result from extra teeth. Additional issues with extra teeth include the potential for pathology (cysts) to develop. A number of syndromes, such as Cleidocranial Dysplasia, Gardner's Syndrome, Enamel-renal-gingival Syndrome, Crouzon's Syndrome, Ehlers-Danlos Syndrome, Hallerman-Streiff Syndrome, Incontinentia Pigmenti, Noonan Syndrome, and Robinow Syndrome, can coexist with hyperdontia or present non-syndromically . Supernumerary premolars have been linked to a mutation in the sequence family FAM10a .

It is not necessary to extract the additional tooth right away if there is no pathology or other orthodontic issues with it . A few medical professionals have advised that supernumerary teeth be extracted right away.

OLIGODONTIA:

A dental condition known as oligodontia occurs when more than six teeth, excluding third molars, are absent .

Oligodontia may be non-syndromic or associated with syndromes like Down syndrome, Van der Woude syndrome, Ellis van Creveld syndrome, Rieger syndrome, Ectodermal dysplasia, Orofacial digital syndrome, Witkop-tooth-nail syndrome, Klinefelter syndrome, Incontinent Pigmenti , or Emanuel syndrome .

In non-syndromic situations, complete prosthetic management is advised to replace lost teeth. Young patients with oligodontia with syndromic association may present certain difficulties because of recalcitrant patient conduct and other systemic abnormalities.

ANODONTIA:

Anodontia is a rare dental anomaly in which there is absence of all teeth. It is commonly associated with syndromes such as Witkop's tooth-nail syndrome Rieger syndrome, Holoprosencephaly, Kabuki syndrome, WolfHirschhorn syndrome and Hair-nail-teeth dysplasia [36]. Depending on the age, cooperation, and syndromic or systemic findings, prosthetic rehabilitation can be done using an implant supported complete denture.

DENTINOGENESIS IMPERFECTA:

The second-hardest tissue in the human body is dentin. Minerals make up 70% of its composition, followed by organic matrix (20%) and water (10%). While type I collagen and a trace amount of non-collagenous dentin phosphoprotein compose the organic phase, hydroxyapatite makes up the majority of the mineral phase. A genetic disorder called dentinogenesis imperfecta (DI) affects the mineralization and/or production of dentin. It may occur alone or in conjunction with systemic bone abnormalities such osteogenesis imperfecta (OI) and impact both the primary and permanent dentition. DI is also known as Capdepont dysplasia or inherited opalescent dentin. DI is divided into three groups: Type II DI is typically present as a distinct entity, but Type I DI is typically linked to OI. C) kind III DI is an uncommon kind distinguished by teeth that resemble shells and have several pulp exposures in the primary dentition. According to reports, the incidence is 1 in 8000 . A mutation in the genes COL1A1 and COL1A2 (type I collagen) is the etiologic basis. Additionally, DI has been linked to mutations in the dentin sialophosphoprotein (DSPP) gene . Osteodysplasia, Goldblatt syndrome, and Ehlers-Danlos syndrome are examples of related syndromes.

Restoring function and reducing pain or infection are the goals of treatment, depending on the severity of the ailment and the primary or permanent impacted teeth. Full-coverage, stainless steel crowns are advised for the primary dentition. For primary teeth with significant attrition, pulp treatment could be recommended before stainless steel crown restoration. Endodontic therapy and beautiful full-coverage restorations are included in comprehensive care for the permanent teeth. To restore the patient's occlusion in cases of extreme attrition, onlays may be designed with potential overdentures. Preventive management involves routine reminder visits and expert fluoride administration in addition to restorative therapy.

AMELOGENESIS IMPERFECTA:

With over 90% mineral content and fewer than 2% organic matrix and water, enamel is the toughest tissue in the human body. Imperfect Amelogenesis
refers to a diverse collection of disorders caused by abnormal enamel production, each with a range of clinical signs. It is characterized by changes in enamel composition in addition to altering the morphological look of enamel .

AI impacts both the primary and permanent dentition and can be either sex-linked or autosomal dominant. The incidence varies according to the population under study, ranging from 1:700 to 1:14000 . Amelogenin (AMELX), ameloblastin (AMBN), enamelin (ENAM), dentin sialophosprotein (DSPP), tuftelin (TUFT1), Kallikrein (KLK4), matrix metalloproteinase 20 or enamelysin (MMP20), and other organic matrix molecules interact to form enamel by ameloblasts. Numerous protein mutations, including those in enamelysin (MMP20), enamelin (ENAM), kallikrein (KLK4), amelogenin (AMELX), and FAM83H, are linked to the development of amelogenesis imperfecta. AI by itself may be regarded as a condition, and it may manifest alongside other concurrent developmental dental abnormalities such pulp calcifications, congenitally absent teeth, and impacted or ectopically erupted permanent teeth. There are typically no related systemic problems in isolated instances of AI. Additionally, AI is frequently linked to conditions like trico-dento-osseous syndrome. In suspected cases, a thorough collection of family history and pedigree analysis is advised due to the genetic etiology and possibility for syndromic connection. The dental team's standard care and diagnostic assessment do not include genetic analysis. Nonetheless, genetic analysis can be a helpful supplementary diagnostic and research tool when assistance is available.

Following a diagnosis, impacted people and their families should get genetic counseling.

CONCLUSION:

The general form, function, and development of the jaws can be impacted by developmental dental anomalies that alter the primary and permanent teeth's shape, number, position, and enamel or dentinal structures. During the primary, mixed, or permanent dentition stages, some developmental defects call for minor to extensive dental treatments employing an interdisciplinary approach, while others are minor departures from normal with no recommended interventions. To better serve impacted patients, clinicians should be knowledgeable about the different kinds of dental developmental anomalies, their relevant clinical and radiographic characteristics, related systemic conditions or syndromes, and the available management strategies.

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