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RESEARCH ARTICLE

IDIOPATHIC IMPAIRED TOOTH ERUPTION - AN ORTHODONTIC PERSPECTIVE

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Abstract

Eruption is the continuous process of movement of a tooth from its developmental location inside the jaw to its functional location in the mouth. Impaired tooth eruption, where this process is disturbed, is common in dental practice. It may manifest either as delayed or complete absence of eruption. This article reviews the local and systemic conditions under which IITE has been reported to occur. The terminology related to disturbances in tooth eruption is also reviewed and clarified. A diagnostic algorithm is proposed to aid the clinician in the diagnosis and treatment planning of IITE. The sequential and timely eruption of teeth is critical to the timing of treatment and the selection of an orthodontic treatment modality. This review addresses the need for a more in-depth understanding of the underlying pathophysiology of IITE and gives the clinician a methodology to approach its diagnosis and treatment.

Introduction:-

The normal eruptive process of the tooth involves navigation through bone and oral epithelium in a precise, bilaterally-timed sequence that must be coordinated with the growth of the jaws in all three planes of space. The eruption process is a result of a complex interaction between osteoblast, osteoclast, and the dental follicle involving many genetic factors. Eruption failure can be attributed to a variety of environmental and/or genetic factors, of which mechanical obstruction is the most common Obstacles can be any of a great variety of objects peripheral to the tooth, including cysts, other teeth, bone, unfavorable tongue posture, or digit habit. The obstruction can also be integral to the tooth in the form of fusion of cementum to bone. The resulting ankylosis prevents further eruption.1

This complex freely regulated process influences the normal development of the craniofacial region. A disturbance in the eruption process can occur in the context of systemic or genetic disorders, the clinical picture may range from a simple, delayed, eruption to complex agenesis. The precise identification of the cause of distribution of the eruption process helps to refine the orthodontic diagnosis define the overall orthodontic treatment plan and orthodontic treatment schedule

Eruption Disturbances And Attributing Factors

Eruption disturbances can be broadly classified as disturbances related to time, disturbances related to the position.

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Time related disturbances include premature eruption, delayed eruption, or impactions. A common feature of these is a significant deviation in eruption time within the established norms for a particular age, sex, race, or ethnicity. Although root development represents the fundamental biologic parameter for tooth eruption, chronological age at presentation is used as the first criteria in the establishment of the diagnosis of prematurity or a delay in eruption.2

Eruption disturbances related to the position include ectopic eruption and transpositions. Positional deviations too can cause a delay in eruption time; however, more commonly the involved tooth erupts within the expected time frame with an abnormality in position. Eruption failure due to mechanical obstruction may be thought of as a "secondary" failure because the eruption mechanism is normal. If the obstruction is removed, eruption usually resumes.3

"Primary Failure of Eruption" (PFE) was coined by Proffit and Vig to describe a condition in which nonankylosed teeth fail to erupt along an eruption path that has been cleared by normal resorption, because of a malfunction of the eruption mechanism. Only posterior teeth are affected, so the result is a posterior open bite.

Clinical Scenario Of Primary Failure Of Eruption

Tooth eruption is a tightly coordinated process, regulated by a series of signalling events between the dental follicle and the osteoblast and osteoclast cells found in the alveolar bone. A disruption in this process can occur as part of a syndrome or as a nonsyndromic disorder (isolated or familial), ranging from delayed eruption to a complete failure of eruption. The prevalence of PFE is approximately 0.06% in the population and a gender predilection of Male: Female of 1:2.25, the occurrence of PFE is relatively low. The main characteristic features of this condition are the failure of an affected tooth to erupt along its path of eruption even without any mechanical hindrance.

Posterior teeth are more commonly involved than the anterior teeth

Involved teeth may erupt into the initial occlusion and then cease to erupt further and affected teeth will be in infra occlusion.

Both primary and permanent molars may be affected involved permanent teeth tend to become ankylosed PFE occurs in isolation with an absence of affected family members

The significant problems in orthodontic management of PFE is the application of orthodontic force inevitably results in ankylosis causing the unerupted tooth remaining in its original submerged position while the other teeth supposed to serve as the anchorage are subject to intrusive force leading to deterioration rather than improvement in occlusal condition .

ETIO Pathogenesis Of Primary Eruption Failure

The role of the dental follicle has emerged as a central mediator of tooth eruption, facilitating the bone resorption necessary for normal tooth eruption and has since been shown to provide the environment and chemoattractants for monocytes to differentiate into osteoclasts, facilitating the bone resorption necessary for normal tooth eruption.4 Stellate reticulum cells found in the dental follicle are observed to secrete parathyroid hormone related peptide (PTHrP).PTHrP induces expression of colony stimulating factor-1 (CSF1) and receptor activator of NF-kappa B ligand (RANKL), which are primary factors involved in osteoclastogenesis. At the apical end of the dental follicle, concomitant expression of Bone Morphogenic Protein (BMP) promotes osteogenesis in a temporally and spatially coordinated fashion, genes involved in mineralization, e.g., amelogenin (AMELX) and ameloblastin (AMBN) may act in concert with those involved in osteoclastogenesis, such as RANKL, CSF1 and C-Fos. The apparent connection between PTH1R and PTHrP, which is secreted in the stellate reticulum and responsible for the induction of CSF1 and RANKL, activity. The established link between PTH1R and PTHrP provides significant evidence of the relationship between PFE, PTHrP signaling and the mediators of eruption necessary for normal bone remodeling.5 Consequently, one might hypothesize that some variants in one of these two focal genes (e.g., PTH1R) could disrupt the balance between bone resorption, necessary to establish the passageway for an erupting tooth, and bone formation, necessary to rebuild bone through which the tooth has transited, thus contributing to PFE. This relationship of PFE with PTH1R and PTHrP therefore provides clues to the possible mechanism of tooth eruption and may be important deducing an appropriate treatment modality. Because PTH1R and PTHrP act in the vitamin D

receptor – retinoid X receptor (VDR/RXR) activation pathway – it is plausible that a critical target of the genetic defect in PFE is the alveolar bone.4,5 The VDR/RXR pathway primarily affects cell signaling, molecular transport, and vitamin and mineral metabolism. Yet VDR/RXR signaling also plays a key role in balancing bone formation with bone resorption such as that seen in bone remodeling.6 In addition to influencing calcium homeostasis in general, the focal genes, PTH1R and PTHrP, and the pathway in which they belong, have been shown to affect the number, quality and function both of osteoclasts and osteoblasts as well as the volume, thickness and density of trabecular bone.4,6

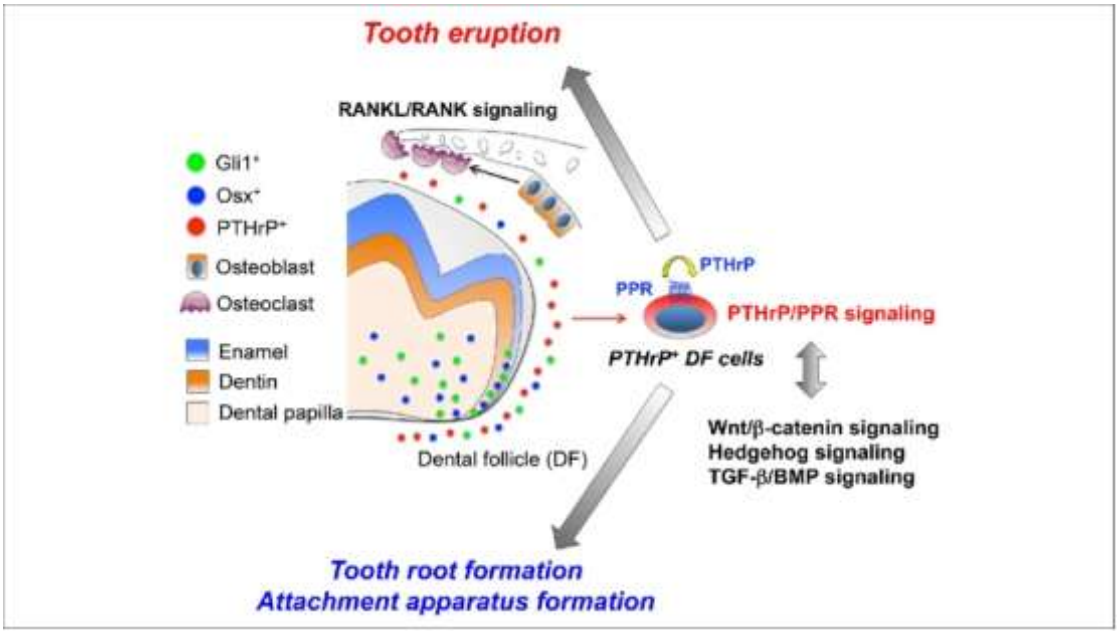


Figure 13:- Role of PTH in tooth eruption.

TNF ligand family RANKL was found to be in membrane bound protein present on osteoblast and stromal cells. Cell to cell signaling between cells with RANKL on their surface and osteoclast precursor carrying the receptor RANK induced both osteoclast formation and activation.

Primary Failure Of Eruption –Classification

The identification of mutations in the PTH1R gene as the cause of PFE and the connection between osteoclasts and osteoblast cells as discussed above also provides the basis for potential clinical management approaches. Although PFE is relatively rare (estimated incidence of 0.6%), the occurrence of eruption problems in the dental/orthodontic setting is not uncommon. A significant challenge in the accurate diagnoses of PFE is the high degree of clinical variability observed in familial and isolated cases. With respect to the pattern viewed from an anteroposterior gradient two types of PFE have been previously described.

Type I is marked by a progressive open bite from the anterior to the posterior of the dental arches.7

Type II presents as a progressive open bite from the anterior to the posterior; however, there also is a more varied expression of eruption failure in more than one quadrant and greater although inadequate eruption of a second molar. Type II is even more challenging to recognize than Type I because the second molar may appear erupted but upon careful examination will lack the ideal interdigitation with the opposing tooth.7

Radiological Finding

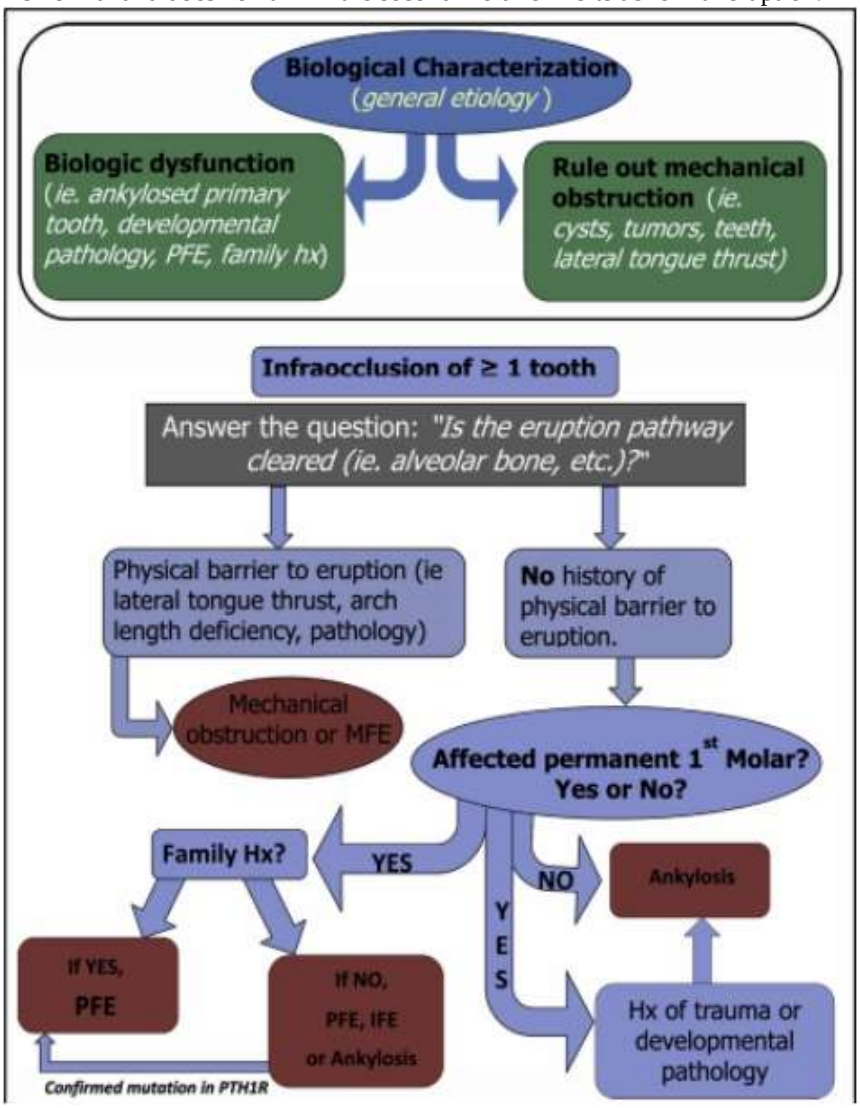
PFE presents characteristic radiological image as well. Enlarged bony crypts around the tooth-germ crowns formed due to resorption of the alveolar process called as resorption chimneys are seen. These “chimneys” suggests a proper resorption process and deficiency of eruptive force to move the tooth-germ along the path of eruption. Primary tooth ankylosis and hypodontia are some characteristic features presented more frequently in patients with PFE than general population.8

Diagnostic Criteria And Clinical Intervention

Ankylosis, strictly defined as the fusion of cementum to alveolar bone, represents an eruption defect most easily confused with PFE.4 The diagnosis of ankylosis relies largely on the clinical appearance of infraocclusion and radiographically by the absence of a periodontal ligament space, however, the determination of an absent periodontal ligament space often can be misinterpreted on a radiograph. The absence of physiologic mobility and the sharp solid sound on percussion of the tooth have also been suggested as diagnostic approaches but show great variation based on the operator.4

However, if a diagnosis of ankylosis is accurate then the affected tooth is the only tooth that will be unresponsive; the remaining teeth will be responsive to orthodontic treatment. Given the difficulty in diagnosing ankylosis accurately, if a physical or mechanical cause cannot be documented and a genetic etiology is discovered, then PFE more likely is the diagnosis. The diagnostic approach above will allow the clinician to follow two different treatment courses including:

1. If PFE can be confirmed, traditional orthodontic treatment with a continuous arch wire should be avoided. An accurate diagnosis of PFE prevents a futile effort by doctor and patient because the teeth will not respond.4
2. If a first molar fails to erupt, early extraction of the first molar will allow the second molar to drift mesially if the second molar is normal and does no harm if the second molar exhibits abnormal eruption.4



Flow chart 2:- Decision tree provided as a tool for clinicians to aid in an objective and systematic diagnosis of eruption disorders. This decision tree also assumes that dental development is sufficient to analyse the eruption potential of the first permanent molar.

MFE- mechanical failure of eruption
PFE - primary failure of eruption
IFE- idiopathic failure of eruption or idiopathic impaired tooth eruption (IITE)

Conclusion:-

Orthodontists are often in a sentry position to perform an early evaluation of craniofacial structures, both clinically and radiographically.9 Proper evaluation of IITE in orthodontic diagnosis and treatment requires a clear definition of the term and its significance.. With the advent of technology, and increased researches in the genetic field, it is possible to alter the eruption disturbances with genetic engineering. Genetic engineering helps in early detection of Primary failure of Eruption. With early identification it is possible to alter the mutations of PTHrP gene using localized gene therapy thus avoiding ankylosis and other disturbances during orthodontic tooth movement. The advances in our understanding of normal and abnormal tooth eruption allows for a systematic clinical diagnosis and management. Recent advances in molecular biology changes the treatment practices in future era.

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