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Review Article

Physiology of tooth eruption

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ABSTRACT

Basic biomedical sciences form foundation for enlightened rational clinical practice and provide substance for innovation and advance. Tooth eruption is highly complex yet finely regulated process of developmental biology. Integration of local, regional and systemic mechanisms is uniquely displayed in eruption physiology. Serial and reciprocal gene expressions in the process enable parallel progress of opposite biologic process in close proximity. Rational clinical address to eruption disorders can only be based on sound understanding of basic physiologic process. Scope for developing enhanced therapeutic capabilities and pharmaceuticals must emerge through understanding and investigation on the perspectives. This review attempts to briefly present simplified view of physiology of tooth eruption drawn on the current understanding.

1. Introduction

Tooth eruption disorder is worrisome impediment of developmental milestone with many adverse sequelae. The biologic basis of the same is essential in learning of dentistry toward correct scientific address and development of new, especially molecular therapeutics and management strategies. Rational preventive interventions and orthodontic procedures mandate biological insights beyond conceived norms of tooth eruption suggested in epidemiologic observations. Vision of cellular and molecular perspective would necessarily empower efficient address for both bud transplants, controlled retention of deciduous teeth when required, facilitation of impacted tooth eruption, tooth stabilization following orthodontic and surgical movement etc. Predictable clinical options for retention and induction of tooth eruption are complementary also to reconstructive surgery after trauma or tumour removal.[1] Biology of alveolar bone formation during tooth eruption is similarly essential to comprehend. Complex engagement of osteoblasts, osteoclasts and dental mesenchyme employs activities of regulatory genes, proteins and humoural factors. Major aspect of systems biology is thus created for refined learning and research in dentistry.[2]

2. The antecedent developments

Complex physiology of tooth development includes bud, cap and bell stages, root development and finally eruption. The developmental cum regenerative processes base on

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interactive contributions of dental epithelium and mesenchyme derived, from cranial neural crest, the dental papilla and dental follicle.[3] The conjoint cell lineage secrets basement membrane to play crucial part in differentiation of odontoblasts from the mesenchyme and form tooth root. Epithelial sheath around tooth root then disrupts at places to allow ingress of mesenchymal cells to make cementoblasts and lay cementum. Parallel growth of dental follicle mesenchyme within cementum and alveolar bone stabilizes the organ, secreting also the matrix substance and organize as periodontal ligament. Transforming growth factor beta (TGFB) derived from the mesenchyme plays role in sustained activation of epithelial cells, inducing expression of variety of transcription factors and proteins amid the participating tissues. Sequential and reciprocal coordinated functioning of cells and bio-molecules drives tooth formation and root development.[4]

3. Progress of tooth eruption

Eruption of permanent tooth is heavily genetically regulated. Rate of progress of the process of the in definable component phases (viz. Pro-functional phase, follicular growth, pre-emergent and post emergent spurts of eruption and post junctional phase) remains subject to influence of variations in local and systemic factors which include gender, craniofacial morphology, body composition, general health and accompaniments of socioeconomic status.

The process necessitates creation of path by resorption in alveolar bone by activity of osteoclasts, for tooth to erupt through. Regulation of osteoclast generation and activity is therefore fundamental consideration in tooth eruption. Relative expression of macrophage chemotactic factor and colony stimulating factor 1(CSF-1), versus expression of osteoprotegerin (OPG) in the dental follicle surrounding un-erupted tooth, determine osteoclast generation at a given instance.[5]

After creation of eruption path, periodontal ligament, the derivative of dental follicle continues with expression of OPG to check resorption of alveolar bone. Epithelio-mesenchyme interaction generates chemotactic factor which attracts monocytes from circulation and CSF-1 that promotes their reformation as osteoclast precursors and proliferation. Such cells then express OPG Ligand and osteoclast differentiation factor (ODF), which act via cell to cell contact to maximize osteoclast differentiation as well as survival. Lately understood expression of receptor activator of Nuclear Factor kappa B Ligand (RANKL) in the osteoclast precursors is crucial in osteoclast differentiation and activity.[6,7] OPG is secreted from osteogenic stromal cells, osteoclasts and even expressed in osteoclast precursors. It can bind and prevent function of RANKL, thereby controlling osteoclastogenesis and activity. Inhibition of osteoclastogenesis is also served differently by expression of another SFRP-1 protein (secreted frizzled related protein1).[8] The mechanisms check bone loss particularly under vulnerable states like oestrogen deficiency or glucocorticosteroid excess.

Diseases involving defect of osteoclast formation e.g. osteopetrosis associate failure of tooth eruption while osteoclast over activity in diseases like Paget's disease associates premature tooth loss and resorption.[9] RANKL/OPG ratio appears to be general determinant of skeletal integrity.[10]

4. Role of the dental follicle

Tooth eruption is localized bilaterally symmetrical process which displays both resorption and formation of alveolar bone on opposite sides of the tooth. Dental follicle co-ordinates such happening.[11] Coronal part of dental follicle participates in directed resorption and formation of eruption path. Basal part of the follicle takes up collaboration in laying down new bone under the tooth roots. RANKL is highly expressed in coronal part while basal part has prominent expression of bone morphogenic protein BMP2.[12] Epidermal growth factor (EGF) binds apical follicle associating intense proliferative activity and dynamic change in profile of proteins.[13] Matrix metalloproteinases (MMPs) are inhibited and increased proteoglycan formation is seen with active eruption. Chronologically differing expression of genes in dental follicle and widening expression of RANKL appear to initiate and propagate process of osteoclastogenesis and tooth eruption.[14]

Eccentric bone formation under root base serves as mechanism to guide eruption movement of tooth.[15,16] Transcription factor named core binding factor 1(Cbfa1) is master regulator of osteoblast differentiation. It is expressed in osteoblasts as well as the dental follicle and derived structures.[17] Under its stimulus number of osteoblast genes express driving synthesis of extracellular matrix. Greater Cbfa 1 expression induces osteoblast specific genes in fibroblasts and myoblasts also. Expression of Cbfa 1in osteoblasts is regulated by the osteo-inductive bone morphogenic protein.[18] Activation of osteoclasts via RANKL/OPG ratio mechanism requires participation of osteoblasts. While osteoblast driven bone formation is essential component of eruption process, osteoblasts play crucial role in osteoclast signalling during the events. Genetic disorder of Cbfa 1 expression adversely affects the skeletal system with failure of tooth eruption.[19]

5. Renovation function of structural elements

Root formation and mineralization of crown must precede onset of eruption. Resorption of root of deciduous tooth along matrix remodelling is necessary to allow proper creation of eruption path of permanent tooth. Odontoclasts in deciduous tooth residue accomplish this enjoying support of matrix metalloproteinases in the vicinity. Odontoclasts resemble osteoclasts in regards to stimulation by CSF-1 and activation by RANKL/OPG ratio profile. However, humoural systemic and local regulators, stimulators in particular, like PTHrP and Interleukin-1 selectively affect osteoclasts but not odontoclasts which function just once in lifetime. Follicular cells enmeshed in cementum of developing permanent tooth represent stellate reticulum. The latter secretes parathyroid hormone related protein PTHrP and Interleukin-1 at critical instance during eruption process. Their binding to dental follicle is crucial to release of chemotactic and colony stimulating factor toward recruiting the monocytes.[20]

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