ISSN: 0975-833X

Available online at http://www.journalcra.com

International Journal of Current Research
Vol. 9, Issue, 10, pp.59319-59322, October, 2017

INTERNATIONAL JOURNAL OF CURRENT RESEARCH

CASE STUDY

ALTERED PASSIVE ERUPTION: ANOTHER STUMBLING BLOCK TO A PLEASING SMILE

***Ketoulelhou Vizo**

Govt Dental College Patiala, India

ARTICLE INFO

Article History:
Received 11th July, 2017
Received in revised form
05th August, 2017
Accepted 16th September, 2017
Published online 31st October, 2017

Key words:

Altered Passive Eruption, Delayed Passive Eruption, Gummy Smile, Excess Gingiva display.

ABSTRACT

Excess gingival display while smiling is one of the most common causes for unaesthetic smile. Altered Passive Eruption (APE) is a clinical situation in which there is a coronally situated Dentogingival complex and excess overlap of gingival tissue over the limits of dental crown, presenting the sensation of hidden tooth and is a frequent finding for the cause of excess gingival display. Perioplastic surgery can play a central role in the correction of such problem. Proper treatment approach and analysis of the individual case with regard to crown-root-alveolar bone relationship should be included.

Citation: Ketoulelhou Vizo, 2017. "Altered passive eruption: Another stumbling block to a pleasing smile", *International Journal of Current Research*, 9, (10), 59319-59322.

INTRODUCTION

Patient awarenessregarding dental problem has increased rapidly in the past few years. The demand for aesthetic dental treatment has also increased as more people opt for a better and a pleasing smile. The aesthetic demand has reached to a point where less than optimal aesthetic are no longer considered as an acceptable outcome (Alexander, 1998). Excess gingival display while smiling is one of the most common causes for unaesthetic smile.There are many different reasons for gummy smile includingvertical maxillary bone excess, dentoalveolar extrusions, hypermobile upper lip, Altered Passive eruption (APE), or a combination of these (Robbins, 1999). APE is a clinical situation in which there is a coronally situated dentogingival complex (Dolt and Robbins, 1997) and excess overlap of gingival tissue over the limits of dental crown, presenting the sensation of hidden tooth (Alpiste-Illueca, 2011). APE presents a frequent finding for the cause of excess gingival display and can be corrected by perioplastic surgery. A typical characteristic of APE includes a short clinical crown length and a square appearance of the crown. It is reported that the prevalence of APE to be approximately 12% considering more than 1000 adults patients with mean age of 24 years (Volchansky and Cleaton-Jones, 1974). In a normally erupted tooth, the Dentogingival Junction is normally situated at the level of the CEJ with the gingival margin slightly covering the limits of dental crown (Ainamo and Löe, 1966).

Corresponding author:* **Ketoulelhou Vizo,
Govt Dental College Patiala, India.

The normal tooth eruption mechanism consists of two phases, active and passive eruption (Gottlieb and Orhan, 1933). The active phase is in which the tooth erupts into the oral cavity in the direction of occlusal plane and the passive phase is in which there is gradual apical migration of the soft tissues covering the tooth crown (Gottlieb and Orhan, 1933). During the initial active eruption stage, the gingival margin and the sulcus are at the tip of the tooth crown, and with gradual eruption process, they migrate at the limits of dental crown i.e CEJ. Simultaneously the junctional epithelium, the oral epithelium and the reduced enamel epithelium also undergo extensive alteration and remodeling, thereby maintaining a normal morphological relationship between gingiva, tooth and the bone. Once the crown reaches the opposing tooth, the occlusal forces acts as one of the most important factor in the regulation of the eruption mechanism of the tooth. Many animal and human studies have confirmed that the eruption restarts when teeth loses its contact with the functional antagonist tooth. However, even when the tooth reaches their functional antagonists, the gingival sulcus and junctional epithelium are still on the enamel, and the clinical crown is approximately two thirds of the anatomic crown. Gottlieb and Orban (1933) believed that active and passive eruption proceed together. The passive eruption phase is characterized by the apical migration of the dentogingival junction onto the cementum. Along with the apical migration of the complex, the length of the clinical crown also increases, (Gargiulo *et al*., 1961) which is coordinated with attrition; the teeth erupts to compensate for the loss of tooth substance worn away by the

attrition process. Historically, passive eruption has been divided into four stages (Gottlieb and Orhan, 1933);

First stage: the bottom of the gingival sulcus remains in the region of the enamel-covered crowns for some time, and the apical end of the attachment epithelium stays at the CEJ. This stage persists in primary teeth almost up to 1 year of age before shedding and in permanent tooth, usually to the age of 20 or 30 years. However, this relation is subjected to a wide range of variations.
Second stage: the bottom of the gingival sulcus is still on the enamel, and the apical end of the attachment epithelium has shifted to the surface of the cementum.
Third stage: when the bottom of the gingival sulcus is at the CEJ, the epithelium attachment is entirely on the cementum, and the enamel covered crown is fully exposed.
Fourth stage: When the entire attachment epithelium is on the cementum, the gingiva may appear normal but is believed to have receded as a result of pathology. This stage represents recession of the gingiva. The stage and rate of migration varies from different person and also in different teeth of the same jaw and on different surfaces of the same tooth.

Although originally thought to be a normal physiologic process, passive eruption is now considered a pathologic process, specially the latter two stages. When this normal physiological tooth eruption sequence is disturbed, then the gingival margin tend to occupy a much more coronal position, giving rise to the clinical condition of short crown. This variation from the normal morphological relationship of the periodontium to a more coronal position has also been referred to as Altered or Delayed passive eruption (Volchansky and Cleaton-Jones, 1974).

Classification of APE

Coslet *et al* in 1977 introduced the concept of APE and classified the APE into two different types according to the relationship between the gingiva and the clinical crown (Coslet *et al*., 1977) (Figure 1).

Type 1: the gingival margin is located incisal/occlusal to the CEJ with a wide keratinized gingiva and the mucogingival junction at a position more apical to the CEJ.
Type 2: the gingival margin is located incisal/occlusal to the CEJ with a normal width of keratinized gingiva and the mucogingival junction positioned at the level of CEJ.

Both the Type 1 and Type 2 is further sub divided into two subtype each, based on the relationship between CEJ and bone crest

Subtype A: the distance between alveolar crest and CEJ is approximately 1.5-2.0 mm, which allows for normalinsertion of connective tissue fiber attachment.
Subtype B: the alveolar crest is at the level of the CEJ or above.

The significance of the distance between bone crest to the CEJ is basically related to the insertion of the fibers into the cementum. With deceased or no cementum surface available for the fibers insertion, as in the case of subtype B, there is a failure of the dentogingival complex to passively migrate apically as the final stage of tooth eruption approaches.

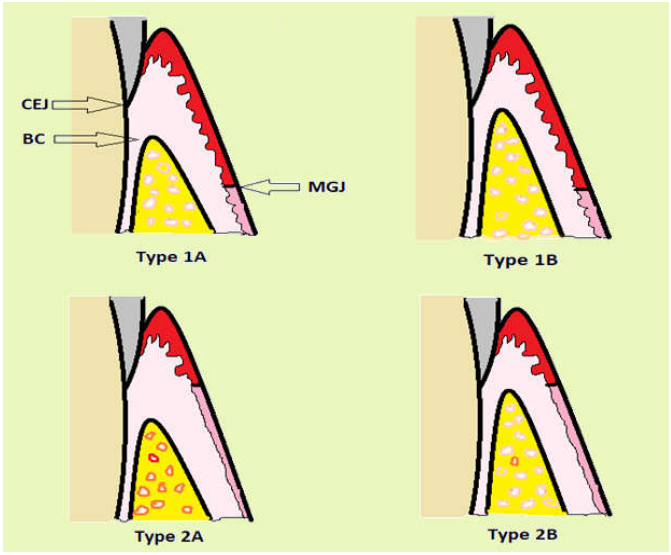

Figure 1. Coslet classification of Altered passive eruptions. CEJ- Cemento Enamel Junction, BC- Bone Crest, MGJ- Mucogingival Junction

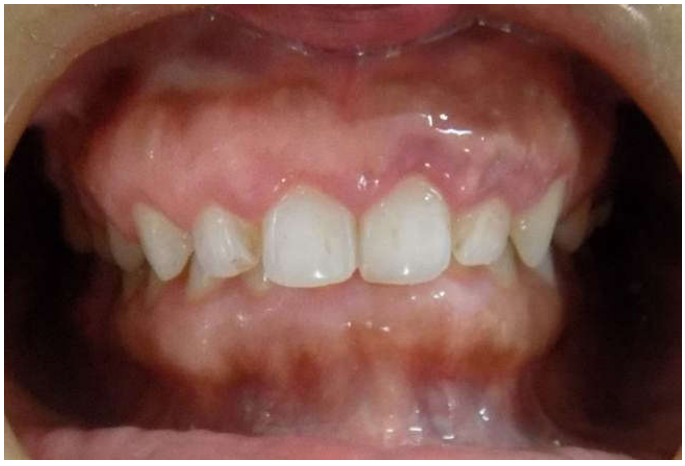

Figure 2. Pre-operative intra oral view showing wide zone of keratinized gingiva with excess overlap of gingival tissue over the limits of dental crown

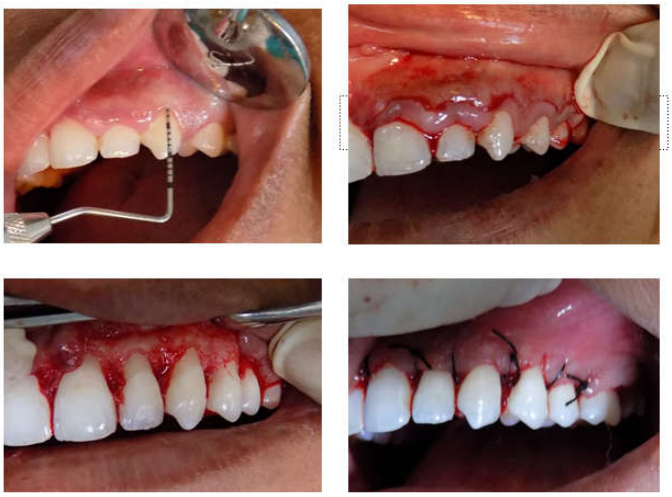

Figure 3. Initial probing of sulcus and internal bevel gingivectomy incisions performed, followed by reflection of full thickness mucoperiosteum flap and post-operative suturing

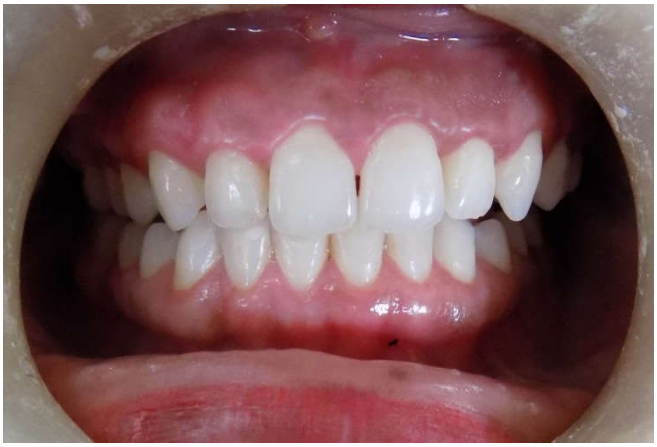

Figure 4. 3 months recall showing stable soft tissue margin with marked improvement in aesthetic outcome

Case

A 33 years old female patient presented with complaint of excess gum display while smiling (Figure 2). Medical history was taken but was of insignificant value to the diagnosis Lip examination shows competent lip and no sign of hypeactivity.Intraoral examination revealswide zone of keratinized gingiva superimposed with slight marginal inflammation. Clinical crown length appears short with no sign of incisal wear. Initial therapy including oral hygiene instructions and scaling was performed, and re-evaluation of the gingiva showed improved gingival condition. However the gingiva was covering about one third of the total crown length and the lower incisors were slightly visible when she occlude her teeth. Probing of gingival sulcus revealed depth of about 3-3.5 mm throughout the dentition. Attempts were made to locate the CEJ in the sulcus using an explorer, but we were unable to locate it.Bone sounding revealed a probing depth of 5.5 mm from the free gingival margin to the osseous crest, which indicated that there was about 2-2.5 mm of soft tissue attachment. Based on the above examination, a diagnosis of APE Type 1 subtype A was made. Esthetic crown lengthening was suggested and discussed with the patient. Quadrant wise surgery was panned and consent was signed. The surgical procedure includes administration of local anesthetic and marking the sulcus depth on the external surface and giving an internal bevel gingivectomy incision followed by reflection of full thickness mucoperiosteum flap (Figure 3).An internal bevel gingivectomy incisionwas preferred over an external bevel gingivectomy incision as it may preserve the pigmentation of the gingiva and preventing an un-esthetic gingival outline after healing.Diagnosis was confirmed after reflection of the mucoperiosteum flap, as a subtype A since there was enough distance between the CEJ and the bone margin for the soft tissue attachment. Although a simple gingivectomy was suffice for the treatment of APE type1A, in our case, osteoplasty was performed as a part of the surgery to improve the surgical outcome since we noticed a slight bulge of the alveolar cortical bone. Simple interrupted sutures were placed and recalled after 7 days for removal. At 3 months recall (Figure 4), the soft tissue margin appears stable with marked improvement in aesthetic outcome.

DISCUSSION

Even though Coslet and co-workers (Coslet *et al*., 1977) had classified and explained the mechanism for the failure of the apical migration of the dentogingival junction, little is known about the specific developing cause of it and little investigation have done for the prevalence of various type of APE and also lack of proper diagnostic criteria (Alpiste-Illueca, 2011). Some authors (Piattelli and Eleuterio, 1991) have even investigated the cause and mechanism which may lead to the failure of tooth eruption, however few studies have tried to relate such mechanism to explain APE. Factors which have been proposed to explain for the cause of the altered eruption includes the interocclusal interference on the part of soft tissue during eruption phase, presence of thick and fibrotic gingival tissue which may impede or slow down the apical migration of the soft tissue. Others have suggested that there might be certain hereditary tendency inpatients presenting with APE. Apreliminary investigation study have found out that a positive correlation can be seen between family members and APE (Rossi *et al*., 2014). Treatment of APE can be a challenge to the clinician because aproper treatment strategy is needed for a successful outcome. If excess bone is removed, chances of gingival recession to a more apical position may be anticipated. If the removal of bone is less than optimal, then there is a high chance of partial resolution of the problem. In some instances, the gingival tissue might even go back to its initial position. The application of the biological width concept finds its applicability in the successful treatment of APE.The preservation of biological width provide adequate physiological dimension for both epithelium and connective tissue attachment. This dimension is relatively constant at approximately 2mm (±30%) (Gargiulo *et al*., 1961). However, it had also been reported in some literature (Vacek *et al*., 1994; Perez *et al*., 2008) that variations in biological widths exists, with biological width as narrow as 0.75mm in some individuals, whereas others had biological width as tall as 4.3mm (Vacek *et al*., 1994). This variation may also be seen in the same patient at different sites and also on different surface of the same tooth (Perez *et al*., 2008). Restoration of crown or class II or III restoration in subject with APE also presents as a risk for the periodontium (Dello Russo, 1984). Three reasons were given for this: the presence of a short clinical crown forces the clinician to make intra sulcular margin restorations; the difficulty of hygiene in this zone; and the absence of connective tissue attachment to the radicular cement that can pose problems for the periodontal defenses (Dello Russo, 1984). Thus the maintenance of biological width is essential when one contemplate on surgical procedure or even restorative procedure.

Apart from the consequence of aesthetic problem in APE, the discussion on whether it presents a risk for further periodontal problem is not clear. Even though APE cases presents with little or no inflammation, an incisally placed gingival margins are more prone to gingival pathosis than those which are located at the CEJ as proposed by Perichard (Perichard, 1979). He also stressed that in this position, the marginal gingiva is not protected from the excursion of food during mastication, which may contribute to the pathosis of the gingiva. Volchansky and Cleaton-Jones in one of their study have reported a statistically significant relationship between APE and acute necrotizing ulcerative gingivitis. They argue that a deep gingival sulcus creates the necessary anaerobic conditions for the development of acute necrotizing ulcerative gingivitis (Volchansky and Cleaton-Jones, 1974). Hence it is prudent to suggest the patient for the correction of APE, not only for aesthetic reasons, but as well for preventing future periodontal complications.

Conclusion

With greater awareness in facial esthetics, the importance of gingiva in its relation to tooth and smile line has drawn considerable attention and interest in the field of esthetic dentistry. The success of treatment outcome of such problems needs a thorough knowledge about the dentogingival complex, biological width considerationand a proper treatment planning. Each specific type of APE must be identified and clear to the treating clinician as the treatment planning and management differs from type to type.Periodontal surgery plays a central role in the correction of APE by placing the marginal gingiva in a more stable position and also maintaining a proper tooth crown proportion, thereby ensuring an optimal aesthetic result.

REFERENCES

Ainamo J. and Löe H. 1966. Anatomical characteristics of gingiva. A clinical and microscopic study of the free and attached gingiva. *J Periodontol*., 37:5-13.

Alexander RE. 1998. Eleven myths of dentoalveolar surgery. *J Am Dent Assoc*., 129: 1271–1279.

Alpiste-Illueca F. 2011. Altered passive eruption (APE): A little -known clinical situation. *Med Oral Patol Oral Cir Bucal*., 1;16 (1):e100-4.

Coslet JG, Vanarsdall R, Weisgold A. 1977. Diagnosis and classification of delayed passive eruption of the dentogingival junction in the adult. *Alpha Omegan*., Dec;7(37):24-8.

Dello Russo NM. 1984. Placement of crown margins in patients with altered passive eruption. *Int J Periodontics Restorative Dent*., 4:58-65.

Dolt AH 3rd, Robbins JW. 1997. Altered passive eruption: an etiology of short clinical crowns. *Quintessence Int*., 28(6):363-72.

Gargiulo AW, Wentz FM, Orban B. 1961. Dimensions and Relations of the Dentogingival Junction in Humans. *Journal of Periodontology*, Vol. 32, No. 3, Pages 261-267.

Gottlieb B, Orhan B. 1933. Active and passive continuous eruption of teeth, *J Dent Res*., 13:214.

Perez, J. R., H. Smukler, M. E. Nunn, 2008. "Clinical dimensions of the supraosseous gingivae in healthy periodontium," *Journal of Periodontology*, Vol. 79, no. 12, pp. 2267–2272.

Perichard JF. 1979. Advanced periodontal disease, ed 2. Philadelphia: Saunders, 420.

Piattelli A. and Eleuterio A. 1991. Primary failure of eruption. *ActaStomatol Belg*., 88:127-30.

Robbins JW. 1999. Differential diagnosis and treatment of excess gingival display. *Pract Periodontics Aesthet Dent*., 11:265-72.

Rossi R, Brunelli G, Piras V, Pilloni A. 2014. Altered Passive Eruption and Familial Trait: A Preliminary Investigation. *International Journal of Dentistry*, Article ID 874092, 5 pages.

Vacek JS, Gher ME, Assad DA, *et al*. 1994. The dimensions of the human dentogingival junction, *Int J Periodont Rector Dent.,* 14:155.

Volchansky A, Cleaton-Jones PE. 1974. Delayed passive eruption. A predisposing factor to Vincent´s infection? *J Dent Asso S Africa*, 29:291-294.

****