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MIDLINE DIASTEMA- A REVIEW

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ABSTRACT

The maxillary midline diastema is, indisputably, one of the dentoalveolar disorders that cause special concern to parents and patients, specifically given its position. It is most commonly seen dento-alveolar anomaly in maxillary arch than mandibular arch. Maxillary midline diastema seen in deciduous dentition is a transcient condition, while those seen in adoscelent and adult have different etiological factors. On the basis of controverting data, it may be concluded that multiple factors are involved in the etiology of maxillary midline diastema, a fact suggesting that further research is required.

KEYWORDS: Diastema, orthodontic Treatment, Retention.

INTRODUCTION

A diastema is a space or gap between two teeth or it may be defined as any spacing or gaps existing in midline of the dental arch. [1,2] Angle (1907) described the dental midline diastema as a rather common form of incomplete occlusion characterized by a space between the maxillary and – less frequent in mandibular central incisors.[2,3,4] He also recognized the functional and esthetic implications of the midline diastema. He stated that the interdental

diastema "always creates an unpleasant appearance and interferes with speech depending on its width". Broadbent (1941) described the maxillary midline diastema in growing children as non-esthetically pleasing and characterized it as the "ugly duckling" stage of dental development. [1,2,5] He considered this stage as a transitional phase for the maxillary interincisal diastema, indicating the space available for the erupting permanent dentition. Broadbent also described the closure of this diastema with complete eruption of lateral incisors and canines as a normal stage of occlusal development. Moyers (1988) studied 82 patients that presented maxillary midline diastema and reported the following causes: imperfect fusion at midline of premaxilla (32.9%), enlarged or malposed upper labial frenum (24.4%), midline diastema as part of normal growth (23.2%), congenitally missing lateral incisors (11%), e) supernumerary teeth at the midline (3.7%), f) unusually small teeth (2.4%), and g) combination of imperfect fusion and congenitally missing lateral incisors (2.4%).[1] Numerous etiological factors for the development of midline diastema are reported in the literature, many of which are environmentally related. Reports on the genetic etiology of dental features are largely anecdotal. [6-13]

Etiology and Management

Numerous etiological factors contributing to the development of midline diastema have been reported and discussed in the literature. There is no agreement on a single etiological factor. A midline diastema usually is part of normal dental development during the mixed dentition. However, several factors can cause a diastema that may require intervention. An enlarged labial frenum has been blamed for most persistent diastemas, but its etiologic role now is understood to represent only a small proportion of cases. Other etiologies associated with diastemas include oral habits, muscular imbalances, physical impediments, abnormal maxillary arch structure, and various dental anomalies. [3,13-17]

The genetic nature of midline diastema International literature includes few reports on genetics or heredity as etiological factors for the development of the maxillary midline diastema.[18-24] Gass et al. (2003) note that preliminary results from thirty families show a possible genetic basis for this diastema.[14] Incomplete palatal fusion is a possible cause of the persisting midline diastema (Stubley, 1976) and there are data supporting that these cystic formations may be related to family history, a fact indicating genetic influences.[24] Harris and Johnson (1991) also examined the role of heredity in abnormal dental occlusion. The existing gap is occupied by connective and epithelial tissue, while fibers of the maxillary labial

frenum or gingival fibers (especially interdental) are often inserted and attached in that site. Normally, the interdental (transeptal) gingival fibers, along with other types of gingival fibers, have the functional role of maintaining teeth in their position.[4] The disturbance of the gingival fiber system can lead to a distal movement of the two maxillary central incisors, sometimes accompanied by lateral incisor rotation and ectopic eruption of the canines. It must be mentioned that Popovich et al. (1977) stated that an incomplete fusion of the intermaxillary suture is only a primary cause for the development of a diastema when other predisposing factors are also present. The diagnosis is based on the radiograph. [15, 20-27] The alveolar process between the maxillary central incisors normally appears in the radiograph as a V-shaped structure, slightly bisected by the intermaxillary suture. Treatment is based on surgical excision of the fibers attached to the residual suture by proceeding with an osteotomy along the intermaxillary suture. This intervention must be performed after the orthodontic closure of the diastema, in order for tissue healing and fiber remodelling to occur in the new position of the teeth. If it is performed before orthodontic treatment, it may cause the scar formation which further will interrupt the closure of the midline diastema.

There is remarkable consensus among scientists concerning the existence of a cause-effect relationship between the presence of hypertrophic or malposed maxillary labial frenum and the maxillary midline diastema. Shashua and Artun (1999) found that there is a correlation between the width of the diastema and the presence of an abnormal frenum. [13, 28-30] Angle (1907) supposed that the maxillary midline diastema is caused by the superior labial frenum; however, he supported that the stability of space closure does not depend on frenum excision. Edwards (1977) found that there was a high, but not absolute correlation of the pretreatment relationship between clinically hypertrophic superior labial frenum and midline diastema.[31,32] Sicher (1952) and Gardiner (1967) also supported this view.[33] Furthermore, individuals with hypertrophic frenum before treatment had higher relapse risk following space closure with orthodontic treatment. James (1967) surgically removed the hypertrophic superior labial frenum in ten individuals, all of whom had midline diastema and complete adult dentition before surgery. [1-3] After a one-year postoperative period and without any orthodontic treatment, 80% of them presented some degree of midline space closure. On the other hand, Popovich et al. (1977a,b) argued that in cases with diastema, the hypertrophic frenum continues to develop more coronally as the alveolar process growths with teeth eruption, because the dentition exercises minimal or no pressure on the frenum. [15,20,28] Tait (1924) supported that the frenum has no effect on the maxillary incisors. [1,2] Ceremelo (1953)

concluded that the presence of the frenum is not related with the presence or the width of the midline diastema.[2,3] Finally, Bergstrom et al. (1973) noted that the probability of long-term spontaneous diastema closure in patients with an abnormal frenum is the same, regardless of whether or not the frenum had been surgically excised.[34] Consequently, further research is needed to determine the cause-effect relationship between the abnormal labial frenum and the maxillary midline diastema. The management involves the fixed orthodontic treatment and surgical removal of frenum after the diastema closure and the orthodontic appliances must be retained in place during healing. By choosing this approach, the tissue expected to form in the new position will help retain the result of treatment. Nevertheless, occasionally, when the frenum is particularly hypertrophic and inhibits the orthodontic closure of the diastema, it is necessary to surgically reposition the frenum nasally before the end of orthodontic treatment.

Bolton discrepancy (tooth size discrepancy) is another cause of midline diastema often reported in the literature.[1] Bishara (1972), Becker (1978) and, Oesterle and Shellhart (1999) described tooth size discrepancy as one of the main causes for maxillary midline diastema.[2,5] They claimed that the most common discrepancy concerning tooth size is the presence of peg shaped lateral incisors; the small shape and size of lateral incisors allows for the distal tipping of central incisors, thus creating the midline diastema. Approximately 5% of the population have some degree of disproportion among the size of individual teeth. The small size or conical shape of the crown of upper lateral incisors is the commonest tooth size abnormality. Sometimes, this abnormality is responsible for the development of a local diastema between the maxillary central incisors, since the space created mesially to the lateral incisors allows the central incisors to move distally. Tooth size discrepancy is a manifestation of what could be considered a generalized dentoalveolar discrepancy. The management involves the aesthetic and restorative treatment, if necessary, the space is opened up mesial and distal to peg shaped lateral for restorative purpose with orthodontic appliance.

Congenital missing lateral incisor or canine is also cause for generalised spacing in upper front teeth and midline diastema between the maxillary central incisors. The diagnosis is made radiographically. The management part includes proper diagnosis and Boltans analysis. Treatment options are one, to close the space with fixed orthodontic treatment if proper overjet and overbite is achievable. Selective grinding of the incisive and palatal canine cusps and of the palatal cusps of the first premolars and restorations with resin composite must be

performed in order to transform canines and first premolars into lateral incisors and canines, respectively. Second option includes the space is opened up at place of missing lateral incisor or canine and prosthetic substitution of the missing teeth. Another less frequently observed reason for midline diastema is the mesiodens. A mesiodens is usually interposed between the roots of the maxillary central incisors and does not allow them to move to the midline and close the diastema. Tay et al. (1984) stated that when supernumerary teeth are normally orientated, they are more likely to cause a delayed eruption of permanent teeth, while when they are inverted, they usually cause bodily displacement of the permanent incisors, torsi version and midline diastema.[2,11,16] Sometimes the incisors are severely rotated and it has effect on mandibular anterior teeth. Diagnosis is exclusively based on the radiographic examination, unless the mesiodens has erupted. Treatment involves the removal of the supernumerary tooth as soon as diagnosed, without causing injury to the adjacent teeth.Then the diastema is usually corrected by using orthodontic forces.

Other factors contributing to maxillary midline diastema include teeth missing due to trauma or disease (congenital tooth absence), displaced teeth in the anterior maxillary segment (Bishara, 1972) and presence of odontomas (Follin, 1985).[2] Oesterle and Shellhart (1999) lay out the consequences of dentoalveolar trauma stating that in any case it may result in generalized spacing and, more specifically, in maxillary midline diastema. The authors report that endocrine disorders resulting in increased growth hormone, such as in acromegaly, and weak tone of orofacial muscles may also contribute to diastema problems. People with acromegaly may also have large jaws for their tooth size, a situation that creates diastemas. Moreover, individuals with weak orofacial muscles and hypotonic lips may present external dentoalveolar migration with labial or buccal tooth movements. These patients may have broad oval arches with no tooth contacts. Excessive anterior overbite has also been suggested by Oesterle and Shellhart (1999) as a factor contributing to midline diastema. When there is no Bolton discrepancy and the patient has an Angle Class I occlusion, increase of the anterior overbite results in either increase of upper arch circumference leading to diastema or anterior mandibular crowding. This phenomenon is due to the wedge-shaped lingual surface of upper central incisors. Oral habits are another etiological factor (Bishara, 1972). Gardiner (1969) noted that finger sucking and/or tongue movement or size may result in interincisal spacing. Proffit (1993) rejected the view that tongue pressure is a possible etiological factor, because of the brief tongue contacts with the lingual surface of anterior teeth; however, he suggested that tongue position at rest may have a greater impact on tooth position. Many other

clinicians have "blamed" habits, such as thumb or finger sucking as etiological factors for maxillary midline diastema. Oesterle and Shellhart (1999) suggested that any habit that results in a long-term force leading to upper anterior teeth separation should be considered as a possible etiological factor.[12] Finally, specific pathological conditions, such as extensive interdental caries, may create the impression of diastema. According to Bishara (1972), periodontitis, cysts and tumors may cause tooth separation. [9]

PREVALENCE OF DIASTEMA

Numerous studies have investigated the frequency/prevalence of diastema. Consequently, there was a wide range of findings from 1.6% to 25.4% in adults and an even greater range in groups of young people (Keene, 1963; Richardson, 1973; McVay and Latta, 1984; Steigaman and Weissberg, 1985; Nainar and Gnanasundaram, 1997). Differences in epidemiological study findings may be attributed to the increased number of factors contributing to midline diastema, to the definitions used to explain its presence and to gender and race differences in the distribution of the hereditary feature in question (Sullivan et al., 1996). [29]

RETENTION AND STABILITY

It is clear that the midline diastema does not always close as a consequence of dental development. Persisting midline diastemas are often seen by dentists in people seeking esthetic improvement. Relapse of the maxillary midline diastema appears, according to Sullivan et al. (1996), in almost 34% of cases, while, according to Shashua and Artun (1999) this rate rises to 50%. [13] Rosenstiel and Rashid (2002), in an Internet study concerning the opinion of lay people about anterior teeth esthetics, showed that conditions such as diastema and midline deviation received the worst ratings.

Treatment of midline diastema is as much important as its retention. Fixed orthodontic treatment is instituted after eliminating the etiology of diastema such as mesiodens. If the highly attached frenum is the reason for maxillary midline diastema, then frenectomy is performed after the diastema closure.

The retention includes the lingually permanent bonded retainer across the diastema for a considerable period of time or for a life, is essential in almost in every case. This long term retention after orthodontic treatment allow the teeth to maintain their physiologic mobility. In cases where the retainer interferes in functional movements of the mandible, it can be bonded cervically or within a shallow rim constructed in the enamel of teeth.

CONCLUSIONS

Considering the different views related to normal dental development, it is concluded that an initial presence of midline diastema in deciduous dentition is not a matter of concern, because this is transcient phase and get self corrected with the eruption of the permanent canine. However, when the diastema is greater than 2.7 mm even after the eruption of lateral incisors, orthodontic intervention is necessary. Its presence has been attributed by most authors to environmental factors, but according to recent studies, genetic predisposition seems to have great importance. According to the literature, the etiology of midline diastema may include a variety of factors. Greater emphasis has been given to environmental factors, whereas possible genetic influences have received minimal attention. The careful evaluation and diagnosis should be made before adoption of any treatment and should be evaluated if there is actual need of treatment. After completion of orthodontic treatment the retention and stability plays an important role in part. In almost all cases, permanent retention of the result of treatment is inevitable for satisfying long-term results. If restorative treatment is performed then avoidance of hard food products and proper oral hygiene should be maintained.

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