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Review Article

EARLY WARNINGS OF PERMANENT MANDIBULAR CANINE ECTOPIA: CLINICAL RELEVANCE. A REVIEW

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ABSTRACT

The eruption of permanent teeth represents a complex series of events, mostly genetically based, whereby eruptive movements of the tooth germ taking place at a predetermined time and route enable the tooth to find its antagonist at a predetermined occlusal plane. As the eruption process is so complex, it is not surprising that the problems related to aberrant eruption pattern may arise. When there is a clinical absence of one or several teeth, and the history indicates that they have not been extracted, then partial anodontia or tooth impaction should be considered.

Most cases with the eruption disturbances of mandibular canines do not have any symptoms, and such canines are often discovered at the radiological examination before the orthodontic treatment. Identification of early warning signs of such disturbances shall greatly enhance the diagnosis and treatment planning for such cases. In this paper we have stressed upon the clinical relevance of these signs, so that not only for the orthodontist but also for the general dental practitioner, it becomes simpler to evaluate the tendency of mandibular canine ectopia and to take appropriate action.

Keywords: Eruption, Ectopia, Impaction, Genetics.

INTRODUCTION

Appropriately referred to as the cornerstones of the dental arches, 'permanent canines' are the units that are important structurally, functionally and esthetically[1]. This is illustrated from the fact that these teeth are rarely found to be missing[2]. Yet, canines are prone to eruption disturbances. A variety of terms are used to describe aberrant mandibular canines. Ectopic or displaced canines refer to teeth that still have the ability to erupt, albeit out of position. Impacted teeth, on the other hand refers to those teeth that have lost the ability to erupt because of their position and/or angulation in the mandible. Bluestone, as early as 1951, was the first to describe impaction of permanent mandibular canine (PMnC)[3]. The documented prevalence rates of PMnC impaction vary from 0.05% to 1.29%[4-8]. When compared to the impactions of maxillary counterpart, mandibular canine impactions occur 20 times less frequently[4]. It is even more rare phenomena when such an impacted mandibular canine migrates to the other side of the mandible, crossing the mandibular midline[9]. Tarsitano et al[10] termed this abnormal movement of a tooth as 'transmigration'. In a radiographic survey by Javid[11], the incidence of mandibular canine transmigration was reported to be 0.33% in general population. Mupparapu[12] used 5 criteria to classify the transmigrated mandibular canines as follows:

Type 1: Positioned mesioangularly across the midline within the jaw bone, labial, or lingual to the anterior teeth and with the crown portion of the tooth crossing the midline.

Type 2: Horizontally impacted near the inferior border of the mandible below the apices of the incisors.

Type 3: Erupting either mesial or distal to the opposite canine.

Type 4: Horizontally impacted near the inferior border of the mandible below the apices of either the premolars or molars on the opposite side.

Type 5: Positioned vertically in the midline (the long axis of the tooth crossing the midline) irrespective of eruption status.

Nodine[13] reported that impacted and migrated mandibular canines are often discovered without having produced any apparent symptoms suggestive of their presence. It is also possible that routine full-mouth intraoral dental radiographs may fail to reveal impacted canines[14]. Hence it makes incumbent upon the clinician to undertake thorough clinical and radiographic investigations to identify the cases with possible eruption disturbances. Early warnings of such

eruption disturbances may either carry an etiologic or causal significance and/or may be purely biologically or non-causally associated with the ectopic canines. Such associations are clinically relevant as these would surely affect the treatment decisions significantly (Figure 1).

Figure 1.

If the crown of the transmigrated tooth migrates past the opposite incisor area or if the apex is seen to have migrated past the apex of the adjacent lateral incisor, it might be mechanically impossible to bring it into place orthodontically. Hence, early observation of one or more of these characteristics is important to allow an astute clinician prevent some, intercept and/or treat yet other cases of mandibular canine eruption disturbances, more effectively and more efficiently. The purpose of this review is, therefore, to explore these early warning signs of mandibular canine ectopia (Figure 2).

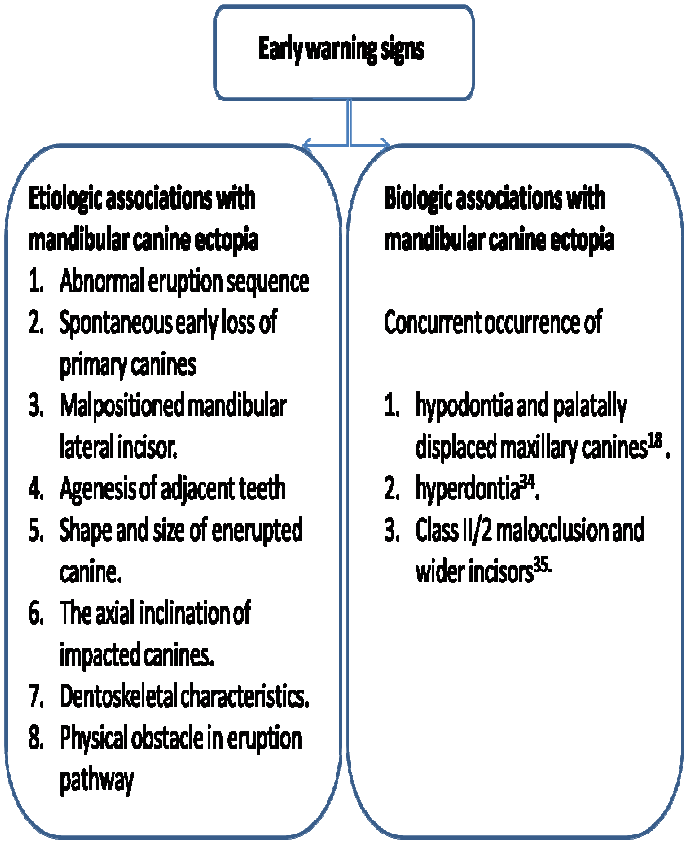

Figure 2: Early warning signs of mandibular canine ectopia.

Early Warning Signs: Etiologic Associations

Nodine[13] reported that it is not possible to put forward a definite etiologic factor responsible for this anomaly but noted that abnormal displacement of the dental lamina in the embryonic life is a commonly accepted explanation of the cause of the displacement and noneruption of such canines. Marks and Schroeder[15] attributed initiation and control of eruption to the dental follicle at the molecular level, with the coronal portion stimulating bone resorption and the apical portion stimulating deposition. They suggested that a regional disturbance in the dental follicle may lead to local defective osteoclastic function with an abnormal eruption pathway being formed. This is a plausible explanation for aberrant eruption of teeth. Further, the following warning signs are explored in an attempt to understand their etiologic relevance pertaining to ectopic mandibular canines.

Abnormal eruption sequence As mandibular lateral incisors erupt during early mixed dentition stage, they push the primary canines distally and laterally into the primate spaces. This is known as the "secondary spacing phenomenon" as it creates more space in the arch. Minor mandibular incisor crowding of 1 to 1.5mm is normal at this stage and is termed 'incisor liability'. The labial eruption of the permanent canines with respect to the primary counterparts leads to an increase in the intercanine distance, thereby, creating more space in the mandibular arch and alleviating this early incisor liability. It follows that in cases of normal eruption, a buccal bulge produced by the mandibular canine should be palpable prior to eruption at any time from the dental age of 9 years[16].

The most favourable sequence of eruption of the mandibular teeth is 6, 1, 2, 3, 4, 5, 7, 8[17]. Should the first premolar erupt prior to the permanent canine, it is not uncommon for the canine to be expected buccally or lingually blocked out (Figure 3). A similar sequaelae is also bound to occur in cases with over-retained canines, when the roots of the primary canine are not resorbing normally, thereby altering the sequence of eruption.

Figure 3. Altered sequence of eruption of teeth expected to result in ectopic canines.

Hence, it becomes clear that the absence of buccal bulges and a lack of mobility of the primary canines around the dental age of 10 years is a strong clinical indication that a developmental anomaly is underway and that there is a very real chance of mandibular canine ectopia and/or impaction[9].

Spontaneous early loss of primary canines

The spontaneous early loss of the primary mandibular canines is an early warning sign of space shortage. When it occurs bilaterally, it leads to a bilateral loss of arch perimeter and a deepening of the bite. In this situation, the incisor imbrication is often resolved by the action of the muscles of the lip and tongue which force the lateral incisors distally into the spaces of the primary canine as the incisors tip lingually. In all cases of bilateral spontaneous loss of the primary canines, the resultant buccal segment crowding will be determined by the sequence of eruption of the permanent canines and premolars, as well as by the degree of space shortage. Should the sequence of eruption be favourable, the canine has a greater chance of good alignment while the second premolar will most likely be blocked out. However, should the first premolar erupt prior to the canine, the canine will be ectopic[16].

Malpositioned mandibular lateral incisor

The permanent mandibular lateral incisor is the most frequently displaced mandibular tooth. The aetiology of this displacement is usually associated with an obstruction or crowding. In some cases however, the cause is unknown. As the alignment of the mandibular permanent canine is dependent on the position of the mandibular permanent lateral incisors, malpositioned lateral incisors cannot guide the erupting permanent canines into the correct position. The developing permanent canines therefore, tend to erupt in a position more mesial than normal, overlapping the labial aspect of the lateral incisors[16] as can be seen in Figure 4.

Figure 4. Malpositioned mandibular lateral incisors and ectopic canines.

Agenesis of adjacent teeth

Vichi and Franchi[18] suggested that agenesis of the adjacent teeth, in particular the lateral incisor, may favor retention of the primary canine and that the excess of space in the dental arch may account for the absence of a correct guide for eruption. The prolonged retention of the deciduous canine is quite often a reliable clue leading to the discovery of its impacted permanent successor[9]. Others suggested that over-retained primary canines are likely to be a result of failure of resorption of the root by the permanent canine rather than being understood as a causative factor in the eruption disturbances of its permanent successor.

Shape and size of enerupted canine

Javid[11] and Joshi and Shetye[19] suggested that the cause of transmigration may be an abnormally strong eruption force, which drives the canine through the dense symphyses. They noted that the conical shape of the tooth aids its passage through the bone. These statements are invalid because by the time the canine does erupt ectopically, the mandible has long been a single bone, the symphyses having been thoroughly remodeled. Furthermore, ectopic second premolar teeth, which are certainly not conical, have also been reported to travel quite a long way[20,21]. Excessively large crowns have been indicated as etiologic factors by Ando et al[22].

The axial inclination of impacted canines

Radiographically, the angle between the mid-sagittal plane and the long axis of a mandibular canine should be between zero and 25 degrees for the eruption of the canines to be considered normal[9]. Howard[23] observed that those canines that lie between 25^0 and 30^0 in the midsagittal plane represent a group of unerupted canines that are displaced but not migrating across the mandibular midline. Those impacted canines that are between 30^0 and 95^0 are a group that tends to cross the midline. An overlap appears to exist between 30^0 and 50^0. When this angle exceeds 50^0, crossing the midline becomes a rule.

Hence, it is advisable to always check for angulation of the mandibular canine with the midsagittal plane when screening panoramic radiograph taken for the routine purpose. If the angulation is above 30, patient should be recalled every 3 months for periodic evaluation[24].

Dentoskeletal characteristics.

Vichi and Franchi[18] observed proclination of the lower incisors and an enlarged symphyseal cross-sectional area of the chin in nearly all their cases. They suggested that these factors could play an important role in the mechanism of transmigration. They further stated that the unerupted canine has the possibility of deviating from its normal developmental site, moving to a horizontal position, and migrating through the symphyseal bone only if enough space is available in front of the lower incisors. Joshi[9] disagreed with the idea of lower incisor proclination and enlargement of the symphyses as etiologic factors. He believes that this is a consequence of canine migration, not a cause. Kerr[25] suggested that the increase in lower incisor proclination may be due to normal variations of incisor angulation during growth.

Physical obstacle in eruption pathway

Bennett[26] emphasized that a very small obstacle, such as a small root fragment, would be sufficient to divert a tooth from its normal path of eruption. Tumors, cysts, and odontomas may cause malposition of teeth if they lie in the path of eruption of teeth[27]. However, it is difficult to say that these pathological conditions were responsible for the transmigration process or the pathological condition occurred after the migration of the canine.

EARLY WARNING SIGNS: BIOLOGIC ASSOCIATIONS

Since eruption process is common to all teeth, a genetic defect in the eruption process of one tooth may reasonably be expected to affect all teeth to some degree. This is validated by the growing body of evidence identifying a complex of genetically controlled dental disturbances that often occur in combination[28-31]. Such associated dental anomalies or dental anomaly patterns (DAP)[32] have become a prime focus of a

number of biologically enlightened clinicians. DAP refers to associated dental abnormalities that are observed together much more frequently than can be explained by chance alone. Peck[33] cited the role of genetics in the etiology of ectopic mandibular canines. He noted bilateral occurrences and the elevated occurrence of hypodontia and palatally displaced canines in the 12 cases of Vichi and Franchi[18] (Figure 5). Retained deciduous teeth and supernumerary teeth, both of which have a genetic etiology are associated with transmigrated mandibular canines and hence, may be biologically or non-causally linked[34]. According to a recent investigation by Shikha et al[35], subjects with mandibular canine(s) impaction appear to be characterized with wider incisors and a remarkably high rate of class II/2 malocclusion. Possible linkage between tooth-size alterations, malocclusion category, and the mandibular canine impaction is therefore likely. Tooth-size alterations and malocclusion category which can be identified early during mixed dention may therefore serve as useful predictors for any possible impending canine impaction. It shall be of great interest in view of above reports to examine whether genes associated with mandibular canine eruption/impaction are also involved in controlling associated disturbances. Such biological associations may help us better understand the concepts on the origin and development of this eruption anomaly and the possible mechanisms of its examination and treatment[32].

Figure 5: Impacted left permanent mandibular canine associated with bilateral maxillary canine impaction.

CONCLUSION

Monitoring for early indications of permanent mandibular canine eruption disturbances is useful in following ways:

1. It provides an insight into the various etiological factors related to this important orthodontic problem. Any biological associations may help us modernize the century old mechanical view of malocclusion.
2. It helps in prediction and early diagnosis of such disturbances thereby allowing identification of candidates who would benefit from early treatment and minimizing potential complications.

REFERENCES

1. Ash, Nelson. Wheeler's Dental Anatomy, Physiology, and Occlusion. 8th edition, 2003, Elsevier.
2. Fukuta Y, Totsuka M, Takeda Y, Yamamoto H. Congenital absence of the permanent canines: J Oral Sci. 2004; 46(4): 247-252.
3. Bluestone LI. The impacted mandibular bicuspid and canine: indications for removal and surgical considerations. Dent Items Interest. 1951; 73: 341–355.
4. Rohrer A. Displaced and impacted canines. International Journal of Orthodontics and Oral Surgery. 1929;15:1002–1004.
5. Celikoglu M, Kamak H, Oktay H. Investigation of transmigrated and impacted maxillary and mandibular canine Teeth in an Orthodontic patient population. Oral Maxillofac surg. 2010;68:1001-1006.
6. Aydin U, Yilmaz HH, Yildirim D. Incidence of canine impaction and transmigration in a patient population. Dentomaxillofac Radiol. 2004;33:164-9.
7. Yavuz MS, Aras MH, Büyükkurt MC, Tozoglu S. Impacted Mandibular Canines. J Contemp Dent Pract. 2007;(8)7:078-085.
8. Mead SV. Incidence of impacted teeth. International Journal of Orthodontia,Oral surgery and Radiography. 1929;15:1003-1020.
9. Joshi MR. Transmigrant mandibular canines: a record of 28 cases and a retrospective review of the literature.Angle Orthod.2001; 71:12–22.
10. Tarsitano JJ, Wooten JW, Burditt JT. Transmigration of nonerupted mandibular canines: report of cases.J Am Dent Assoc.1971; 82:1395–1397.
11. Javid B. Transmigration of impacted mandibular cuspids. Int J Oral Surg 1985;14:547-9.
12. Mupparapu M. Patterns of intra-osseous transmigration and ectopic eruption of mandibular canines: Review of literature and report of nine additional cases. Dentomaxillofac Radiol 2002;31:355.
13. Nodine AM. Aberrant teeth, their history, causes and treatment. Dent Items of Interest.1943; 65: 440–451.
14. Howard RD. The anomalous mandibular canine.Br J Orthod. 1976; 3: 117–119.
15. Marks SCJ, Schroeder HE. Tooth eruption: theories and facts.Anat Rec.1996; 245: 374–393.
16. Hudson APJ, Harris AMP, Mohamed N. Early identification and management of mandibular canine ectopia. SADJ 2011; 66 (10): 462-7.
17. Moyers RE. Handbook of Orthodontics. 4th edition, Chicago, 1988, Mosby.
18. Vichi M, Franchi L. The transmigration of the permanent lower canine. Minerva Stomatol.1991; 40:579–589.
19. Joshi MR, Shetye SB. Transmigration of mandibular canines: areview of the literature and report of two cases.Quintessence Int. 1994; 25: 291–294.
20. Sutton PR. Migrating nonerupted mandibular premolars: a case of migration into the coronoid process.Oral Surg Oral Med Oral Pathol.1968; 25: 87–98.

21. Orton HS, McDonald F. The eruptive potential of teeth: a case report of a wandering lower second premolar. Eur J Orthod.1986; 8:242–246.
22. Ando S, Aizawa K, Nakashima T, Sanka Y, Shimbo K, Kiyokawa K. Transmigration process of the impacted mandibular cuspid. J Nihon Univ Sch Dent.1964; 6: 66–71.
23. Howard RD. The anomalous mandibular canine.Br J Orthod. 1976; 3: 117–119.
24. Sharma G, Nagpal A. Transmigration of Mandibular Canine: Report of Four Cases and Review of Literature. Case Reports in Dentistry. Volume 2011, Article ID 381382, 5pages.
25. Kerr WJ. A migratory mandibular canine.Br J Orthod.1982; 9: 111–112.
26. Bennett S. Prehistoric specimen-inverted canine in mandible of a child. In: The Dental Record.1931:138. Cited by: Nodine AM. Aberrant teeth, their history, causes and treatment. Dent Items of Interest.1944; 66: 256–265.
27. Camilleri S and Scerri E. Transmigration of Mandibular Canines—A Review of the Literature and a Report of Five Cases. Angle Orthod., 2003; 73: 753–762.
28. Becker A, Smith P, Behar R. The incidence of anomalous maxillary lateral incisors in relation to palatally-displaced cuspids. Angle Orthod. 1981; 51: 24–29.
29. Peck S, Peck L, Kataja M. Prevalence of tooth agenesis and pegshaped maxillary lateral incisor associated with palatally displaced canine (PDC) anomaly. Am J Orthod Dentofacial Orthop. 1996; 110: 441–443.
30. Pirinen S, Arte S, Apajalahti S. Palatal displacement of canine is genetic and related to congenital absence of teeth. J Dent Res.1996; 75: 1742–1746.
31. Zilberman Y, Cohen B, Becker A. Familial trends in palatal canines, anomalous lateral incisors, and related phenomena. Eur J Orthod. 1990;12:135–139.
32. Peck S. Dental Anomaly Patterns (DAP). A new way to look at malocclusion. Angle Orthod. 2009; 79(5): 1015-1016.
33. Peck S. On the phenomenon of intraosseous migration of nonerupting teeth. Am J Orthod Dentofacial Orthop. 1998; 113: 515–517.
34. Camilleri S. Double transmigration and hyperdontia. Angle Orthodontist, 2007; Vol 77, No 4, 2007.
35. Jain S, Shetty KS, Prakash AT, Agrawal M, Jain S. Permanent mandibular canine(s) impaction: expansion of our understanding. Australian Journal of Orthodontics. 2014; 30(1): 39-44.

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