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REVIEW ARTICLE

Mandibular canine transmigration: aethio-pathogenetic aspects and six new reported cases

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Abstract

Mandibular canine transmigration is a rare dental ectopia that creates surgical, orthodontic, restorative and interceptive problems. Aethiologic and pathogenetic aspects of mandibular canine transmigration are examined through an international literature review.

Six new cases are then presented. All of them involve the left canine in women aged between 7 and 32 years.

Finally, a pathogenetic theory is elaborated to explain such long and lasting tooth migration. Two main factors contribute to this movement: a pericoronal osteolitic area and a strong and lasting 'vis a tergo' because of the root formation.

An early interceptive treatment of mandibular canine migration can avoid tooth extraction as well as complex orthodontic and restorative therapies.

Introduction

Intraosseous tooth migration is a very rare dental ectopia, that occurs only in the mandible[1], involving lateral incisors, second premolars and especially canines[1,2]. Such teeth normally remain in the same side of the mandibular arch. Canine is the only tooth that migrates across the midline[3] and this anomalous movement has been defined as 'transmigration'[4]. Canine transmigration creates surgical, orthodontic, restorative and interceptive problems.

The aim of this study is to present six new clinical cases of canine transmigration, concerning its aethiologic and pathogenetic aspects according to the recent literature.

Aethiologic and pathogenetic aspects

Many pathologic conditions have been listed as aethiologic factors of the canine transmigration (Table 1), but it is difficult to say if they are responsible for the transmigration or they occur after the canine is migrated. Anyhow, it should be considered that any reason that can cause the canine impaction and its eruptive delay can be able to prepare the canine to migrate.

Nevertheless, pathogenetic theories are not so numerous. Some authors[5,6] deemed the canine germ been originated far from the normal site of eruption; however, all the available evidences demonstrate that the germ develops in the typical side and then migrates in an ectopic position[3,7]. Sutton[8] suggested that the canine, horizontally positioned for unknown causes, undergoes to an extremely strong eruptive thrust that leads the crown across the midline through the dense mandibular symphysis. Howard[9] suggested that when canine inclination ranges between 25° and 30° in the midsagittal plane, the tooth can move but does not migrate across the midline; while if the inclination ranges between 30° and 95°, the canine tends to cross the midline. Tooth behaviour seems to be variable between 30° and 50° but transmigration becomes a rule when the inclination exceeds 50°. Joshi[10] (values between 45° and 90°) and Aydin *et al.*[11] (values between 40° and 93°) confirmed such observations.

Table 1 Aetiologic factors of transmigration

Heredity and genetic factors
Obstacles to canine eruption (root fragments of the deciduous canine, odontomas, tumours)
Cysts
Early loss of the deciduous canine
Over-retention of the deciduous canine
Crowding and inadequate available space to eruption
Hypodontia and extreme available space to eruption
Supernumerary teeth
Extreme length of the mandibular canine crown
Buccal inclination of the lower incisors
Increased axial inclination of the un-erupted canine
Increased symphyseal area

Thoma[12], Pippi and Sfasciotti[7], Greenberg and Orlian[13], Wertz[14] and Fiedler and Alling[15] reported cases in which a radio-transparent cystic area surrounded the crown of the transmigrated canines. Pippi and Sfasciotti[7] suggested that the appearance of a pericoronal osteolitic area, because of an increased follicular space, could be related with the intraosseous migration of the canine. This relationship was presented in all cases and in all the radiographs in the follow-up. Also, Al-Wahedi[16] noticed that the transmigrated canines were often associated to cystic lesions, and he suggested that they could facilitate the migration. Many other authors noted the association between cysts and transmigration. Others, instead, believe that the migration is due only to a strong root eruptive thrust. On the contrary, Camilleri and Scerri[3] affirmed that, as the cyst is an expanding lesion, it positions the tooth to the back rather than facilitates its motion ahead. They also described a development pattern of transmigration as follows:

1. Development and eruption initially appear normal.
2. The tooth deviates from its path for no apparent reason.
3. Transmigration. The greatest amount of the movement occurs during the pubertal age, where alveolar growth is at its maximum. The direction of the movement is usually mesial, even though a distal movement[1] was also described.
4. Occlusal movement of the tooth ceases. A mesial and apical path of movement is established, which worsens with time. As alveolar growth continues, the tooth becomes progressively buried.

A similar pattern of movement has been proposed by other authors[1,4,13,14,17–19]. A radiographic control of this pattern can be also observed in a case reported by Pippi and Sfasciotti[7], in which the correlation between dental movement, pericoronal radiolucency and progressive inclination of the tooth axis can be noted. The vast majority of ectopic teeth follow this typical pattern of movement[3]. This seems to suggest that a wrong area of the dental follicle is activated[20], leading the tooth to 'erupt' in a wrong direction. In the case of transmigrated canines, the unobstructed direction of movement allows them to travel as far as the aberrant eruption pathway will carry them[3,21].

The speed of migratory movement could not be investigated in the most of cases because of lack of radiographic follow-up. Tooth migration, anyhow, is very slow[13,22]. Ando *et al.*[4] reported the first long-term study with a radiographic follow-up. These authors followed up one case of canine transmigration over a 6-year period, in which the tooth progressively moved from its original position near to the contralateral mental foramen. They noted that the canine movement was more rapid before the root formation was completed. Stafne[23] and Sutton[8] also noted that the greater part of migration occurs before the complete root formation and they pointed out that the tooth always moves in the crown direction. Only Dhooria *et al.*[24] observed that the tooth movement was more rapid after the complete root formation (3–4 mm per year). Pippi and Sfasciotti[7] published the panoramic radiographs of two cases followed up for a long time. In the first case, the right canine moved as far as the mesial root of the opposite first molar over a 3-year period of time. In the second case, the right canine reached the mandibular lower margin below the contralateral premolars. Howard[9] suggested that the elder is the patient, the more distant is the canine far from the midline, because the age increases the available time for the migration; even if in the elderly patients transmigrated canines seem to be closer to midline than those in the younger patients[7,11,22,25].

Case report

Up to 2007, six new cases of mandibular canine transmigration were found and treated at the Oral Surgery Unit of the Odontostomatological Sciences Department of the University 'La Sapienza' of Rome. Table 2 resumes clinical and radiographic features of all these six cases. The Figures 1, 2, 4, 5 and 6 show the OPT of the patients and Figure 3 shows the clinical aspect of the case 3.

All patients are women and the age at the discovery time was comprised between 17 and 21 years old. In all cases, the lower left canine was involved. Five patients presented the over-retained deciduous canine, and in five of the six cases, the transmigrated canine was in a vestibular position while the fifth one was in a lingual

Table 2 Present transmigrated canines by clinical features

Name	Age	Sex	Canine	Mandibular position	Inclination	Position	Crown location	Pericoronal radiotransparency	Observations	Type (Mupparapu's classification)[25]
A.I.	21	female	3.3	Lower border	Horizontal	4.3–4.4	Vestibular	+	73 over-retained; 18-28-38 missed; 48 bud not erupted	4
A.B.	17	female	3.3	Lower border	Horizontal	4.3	Vestibular	+	73 over-retained; 42 missed	2
G.P.	16	Female	3.3	Under the tooth apex	Oblique	4.2–4.3	Vestibular	+	73 over-retained	1
V.C.	14	female	3.3	Mucogingival line	Oblique	4.1–4.2	Vestibularly erupted	NN		1
F.U.	32	female	3.3	Mucogingival line	Oblique	42–43	Vestibularly erupted	NN	73 over-retained; multiples caries; remained root fragment	1
M.N.	19	female	3.3	Lower border	Horizontal	4.4–4.5	Vestibular	+	7.3 over-retained	4

NN, Not noticeable.

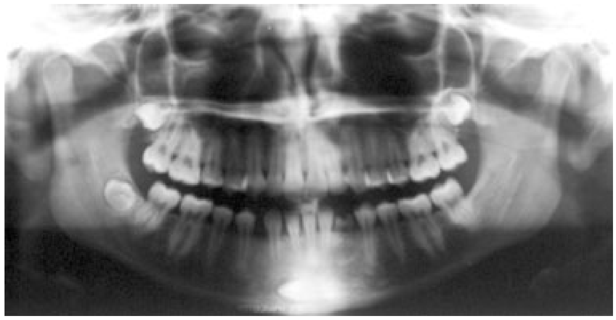

Figure 1 Case 1.

Figure 2 Case 2.

Figure 3 Case 3.

position. All the impacted canines (four cases) presented a pericoronal radiotransparency. In all cases, the canine was extracted.

Discussion

Much has been already written about mandibular canine transmigration but much will be yet written. Some clinical and pathogenetic features, indeed, are

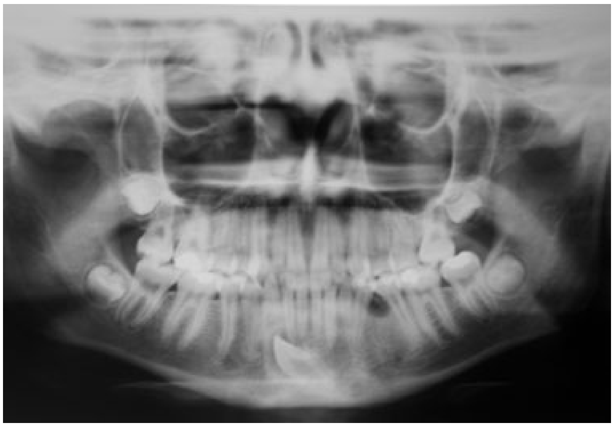

Figure 4 Case 4.

Figure 5 Case 5.

Figure 6 Case 6.

not still explained. Why and how the canine transmigration that once was delayed in eruption or impacted is still unclear. Why no cases of real maxillary canine transmigration are reported is likewise unexplained.

First of all, most authors consider transmigration of the canine, which has crossed the midline with just only the tip of the cusp; this relies on the canine tendency to cross the midline and not on the real amount of the movement. We prefer to consider transmigrated, as Javid[26] already did; the impacted mandibular canine, which crosses the midline for, at least, half of its length although it can depends on the stage in which the impacted tooth, is discovered.

In this regard, only 65 out of the 179 mandibular canines, published as transmigrated, have crossed the midline for, at least, half of their length, and therefore, they solely can be really considered transmigrated. On the other hand, all the upper canines reported as transmigrated were not really transmigrated, because they cross the midline for less the half of their entire length.

Then, it is noteworthy that all the present cases (six) involve the left canine, although our previously reported five cases[7] involved the right one more than the left one (4:1). The higher frequency of the left canine transmigration agrees with the international data but it cannot be explained.

On the basis of the present series, in addition to the cases already published[7] and reviewed, it is possible to elaborate a pathogenetic theory about the canine intraosseous migration. Two different and contemporary factors are necessary to justify tooth movement:

1. A strong and extended 'vis a tergo', caused by a lasting root formation.
2. A pericoronal osteolitic area, because of a widening follicular space.

Once the canine impaction has taken place, these factors can cause its anomalous movement inside the mandible. Why the inclination of tooth axis takes place is unclear, but an abnormal activation of the dental follicle is a sure cause for the pericoronal osteolitic area, because of an anomalous secretion of signal molecules (EGF, CSF, IL, RANKL, MCP-1, OPG, Cbfa1), in terms of timing, localisation and/or amount. This relates to traumatic/inflammatory stimuli or otherwise genetically induced, and leads to a localised modification of the osteoclastic/osteoblastic activity[27–29]. Therefore, at the beginning, a pericoronal enlargement takes place and then it becomes asymmetrically wider. This space represents a 'locus minoris resistentiae', towards which the tooth moves when the root formation is still in action, so that the canine walks inside the pericoronal enlargement. The simultaneous occurrence of these two events creates the movement and the progression of the canine inside the mandible, in the opposite direction to the root. This movement stops when the tooth finds a mechanical obstacle (e.g. jaw cortical bone), when it exhausts the 'vis a tergo' of the root or when the pericoronal osteolisis doesn't form itself anymore because of the end of the follicular osteoclastic activity. Such movement seems to be also facilitated by several factors that are a voluminous mandibular symphysis[30], an emphasised buccal inclination of the lower incisors, as well as the typical conic shape of the canine root and crown[26]. The association of the osteolitic area, that facilitates the progression inside the jaw[7,16], with the strong eruptive trust, that allows the tooth[4,7,8,23] movement forward, probably explains the lasting and, therefore, long canine migration. Finally, the particular mandibular structure, with two cortical plates and a very spongious bone tissue in between, can explain why the lower canine transmigrates while the upper one doesn't transmigrate through the dense palatine bone.

Conclusions

Transmigration of the mandibular canine is a rare event. Why the canine intraosseous migration happens is already unknown, but all events that determine canine impaction or a delay in its eruptive process are able to predispose to its migration. The dental follicle plays an important role for this migration movement causing an abnormal pericoronal ostelitic area.

A missing canine in the dental arch is an extremely important problem from a functional and an aesthetic point of view. It is, therefore, necessary to early intercept all the situations that can predispose, such as pathology by routine radiographic exams, so that the patient can be treated before the canine transmigrates, in order to avoid its surgical extraction as well as complex orthodontic and restorative therapies.

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