J Stoma 2017; 70, 1: 24-30
DOI: 10.5604/01.3001.0010.1789

ORIGINAL ARTICLE

Malocclusion and prevalence of developmental dental anomalies of form, size, number and eruption in southern Europe

Wady zgryzu i występowanie wad rozwojowych zębów w zakresie ich kształtu, rozmiaru, liczby i wyrzynania na południu Europy

Rocío Molina-Solana, Enrique Solano-Reina, Rosa-María Yáñez-Vico

Department of Dentistry, School of Dentistry, University of Seville, Spain
Katedra Stomatologii, Wydział Stomatologiczny, Uniwersytet w Sewilli, Hiszpania
Head: prof. *R. Santos*

Abstract

Aim of the study. *To analyze dental anomalies (agenesis, supernumerary teeth, microdontia, transposition, impaction, taurodontism, dilacerations, enamel hypoplasia, dens evaginatus and dens invaginatus) in orthodontic subjects.* ***Material and method.*** *Pretreatment clinical and radiographic records of 379 orthodontic subjects were studied.* ***Results.*** *22.2% of patients presented at least one dental anomaly. The prevalence in females was statistically significant (chi squared test, p<0.05) compared to males.* ***Conclusions.*** *Congenitally absent teeth and the presence of impacted teeth were the most frequently observed abnormalities. Orthodontic planning and treatment could be improved by bearing in mind the presence of dental anomalies and the relationships between them.*

Streszczenie

Cel pracy. *Analiza anomalii uzębienia (ageneza, nadliczbowość zębów, mikrodoncja, transpozycje, zęby zatrzymane, zęby pryzmatyczne, dilaceracje, niedorozwój szkliwa, dens evaginatus i dens invaginatus), u pacjentów przed leczeniem ortodontycznym.* ***Materiał i metody.*** *Zbadano kliniczną i radiologiczną dokumentację 379 pacjentów przed leczeniem ortodontycznym.* ***Wyniki.*** *U 22.2% pacjentów wykryto co najmniej jedną anomalię zębową. Przewaga kobiet była statystycznie znacząca (test chi-kwadrat, p<0.05) w porównaniu z mężczyznami.* ***Wnioski.*** *Wrodzony brak zębów i obecność zatrzymanych zębów należały do najczęściej odnotowanych anomalii. Znajomość nieprawidłowości zębowych i ich wzajemne zależności mogą mieć znaczenie dla ulepszenia planowanego leczenia ortodontycznego.*

KEYWORDS:
malocclusion, dental developmental anomalies, orthodontic treatment

HASŁA INDEKSOWE:
wada zgryzu, zaburzenia rozwojowe zębów, leczenie ortodontyczne

Introduction

The absence of one or more permanent teeth is the most common congenital, or developmental, anomaly recorded in children.[1] There are also other common developmental dental anomalies which disturb the normal form, size or eruption position and otherwise affect the presence or absence of teeth.[2-4] In the prenatal and postnatal periods, many genetic factors and etiological events in the environment can influence the morphodifferentiation stage of dental development and thus activate these dental abnormalities.[5, 6]

A proper final dental occlusion is determined to a large extent by the normal size, number and eruption of the teeth. For this reason, knowledge of dental anomalies and their identification are vital for dental and orthodontic treatment planning, since they can lead to edentulous spaces in the maxillary or mandibular arch

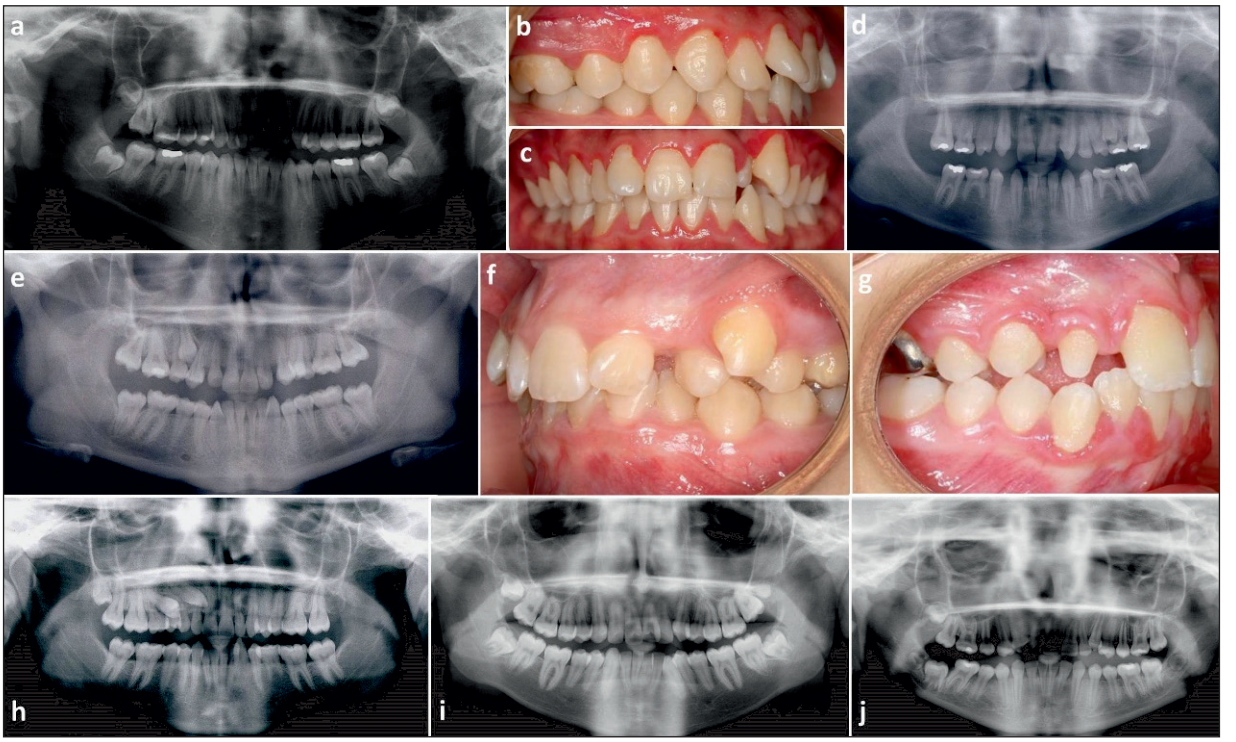

Fig. 1. *Dental anomalies found in the study; a, b, c, – hyperdontia, d – agenesis, e, f – tooth transposition, g – impaction, h – microdontia, i – taurodontism, j – dilaceration.*

that must be closed by orthodontic movement, prostheses or implants. At other times, a dental anomaly means excessive dental matter or an ectopic eruption, which makes it difficult to carry out orthodontic treatment. Planning therefore should be scheduled in the context of a multidisciplinary team and broken down into different stages in order to accomplish the specific objectives.

There have been several studies analyzing different dental anomalies in various populations, although little is known about relationships between specific abnormalities.[4,7-10] The purpose of this study was, therefore, to analyze dental anomalies in the orthodontic population and relationships between them.

Material and methods

The study was undertaken using the pretreatment records: dental history, intraoral dental photographs, dental casts, orthopantomography, lateral x-rays and periapical radiographs, where necessary, and exhaustive extraoral and intraoral analysis of 379 consecutive orthodontic patients from the Orthodontics Department of the University of Seville (Spain). The Bioethics Committee for experimentation in the University of Seville (Spain) independently approved the procedure. The present study was carried out in accordance with the ethical principles governing medical research and human subjects, as laid down in the Helsinki Declaration (2002 version, www.wma.net/e/policy/b3.htm). Patients with syndromes, severe medical histories or extractions of permanent teeth before orthodontic treatment were excluded.

The following dental anomalies were analyzed (Fig. 1):

Alterations of number:

– hyperdontia (supernumerary teeth): accessory teeth (referred to as supernumerary) which appear in addition to the regular number found in the dental arch,
– hypodontia (tooth agenesis): congenital ab-

sence of a permanent tooth or a tooth germ (excluding third molars).

Alterations of position:

– tooth transposition: a form of ectopic eruption in which a tooth develops and erupts in the position usually occupied by another or the positional interchange of two adjacent teeth,
– impaction: a tooth which is unlikely to fully erupt into its normal functional position, based on clinical and radiographic assessment.

Alterations of morphology:

– microdontia: a dental anomaly that produces one or more disproportionately small teeth,
– taurodontism: a dental anomaly in which the body of the tooth and pulp chamber are enlarged, with apically displaced furcation,
– enamel hypoplasia: the result of defective enamel matrix formation, leading to altered development of the organic matrix or calcification,
– dilaceration: an abnormal angulation or curve in the linear relationship between the crown of a tooth and its root,
– dens evaginatus (talon cusp): a developmental anomaly with the characteristic presence of a supernumerary tubercle extending from the cingulum area or cemento-enamel junction of the maxillary or mandibular anterior teeth,
– dens invaginatus: a dental anomaly characterized by a developmental malformation involving an invagination of the crown or root surface.

The data were analyzed using IBM SPSS software for Windows (version 20.0, New York, United States). A descriptive analysis of the presence and types of anomaly according to gender was carried out. The chi square test was used to determine statistical significance in the occurrence of dental anomalies by gender. The relationship between the frequencies of different dental anomalies in the same subject was studied using the phi coefficient. To determine possible relationships between the numbers of teeth affected by each type of dental anomaly, Spearman's rho coefficient was calculated. Significance was set at $p<0.05$.

Table 1. Incidence and distribution by gender of developmental dental anomalies

Dental anomaly	Male (n=156)		Female (n=223)		p
	n	%	n	%	
Any dental anomaly	26	16.7	58	26	0.031*
Agenesis	9	5.8	23	10.3	0.117
Hyperdontia	2	1.3	3	1.3	0.958
Microdontia	6	3.8	5	2.2	0.360
Transposition	0	0	5	2.2	0.60
Impaction	8	5.1	23	10.3	0.70
Taurodontism	2	1.3	1	0.4	0.367
Dilaceration	3	1.9	7	3.1	0.467
Enamel hypoplasia	0	0	0	0	
Dens evaginatus	0	0	0	0	
Dens invaginatus	0	0	0	0	

* statistically significant with chi-square test.

Results

From a total sample of 379 subjects, 58.8% were female (20.32± 8.42 years) and 41.2% were male (17.45±5.17). The incidence of dental anomalies in the sample was 22.2%. Agenesis (16.1%) and impaction (15.4%) were the most frequently found anomalies (Table 1). There was a significant gender difference between males and females in terms of those affected by any dental anomaly at all ($p<0.05$) (Table 1). Agenesis, supernumerary teeth, microdontia, transposition, impaction, taurodontism and dilacerations were not statistically significant by gender using the chi square test. No subjects were found with enamel hypoplasia, dens evaginatus or dens invaginatus (Table 1).

There were statistically significant differences in the incidence of associations between different kinds of developmental anomaly (Table 2). Abnormal tooth morphologies (microdontia with taurodontism) and alterations of eruption position (transposition with impaction) were found to correlate. Alterations of number were variously associated with an abnormal morphology (agenesis and supernumerary teeth with microdontia) and alterations of eruption (agenesis with transposition) (Table 2). There was also a correlation between the numbers of teeth affected (Table 3).

Discussion

Developmental dental anomalies have a significant influence on orthodontic treatment. A dental abnormality can increase or occupy available space in the dental arch, resulting in crowding or sagittal, transverse and vertical malocclusions. Moreover, alterations in the eruption position

Table 2. Incidence of relationships between dental anomalies in the study sample (phi coefficient)

	Dilaceration	Taurodontism	Impaction	Transposition	Microdontia	Supernumerary
Agenesis	0.858	0.597	0.108	**0.011***	**0.001***	0.494
Supernumerary	0.711	0.000	0.502	0.795	**0.022***	
Microdontia	0.580	**0.002***	0.219	0.697		
Transposition	0.711	0.841	**0.009***			
Impaction	0.339	0.604				
Taurodontism	0.775					

The table shows p-value, * in bold: statistically significant.

Table 3. Relationship between the number of dental anomalies found in the study using the Spearman rho coefficient

	Agenesis	Hyperdontia	Microdontia	Transposition	Impaction	Taurodontism
Hyperdontia	-0.036					
Microdontia	**0.157****	**0.117***				
Transposition	**0.113***	-0.015	-0.022			
Impaction	0.074	-0.034	0.065	**0.113***		
Taurodontism	-0.028	**0.249****	**0.161****	-0.011	-0.027	
Dilaceration	0.006	-0.019	-0.028	-0.021	-0.049	-0.015

* in bold: statistically significant at p<0.05, ** in bold: statistically significant at p<0.01.

make orthodontic treatment difficult. In this study, developmental dental anomalies in orthodontic patients were examined before orthodontic treatment took place. The literature reports significant differences in the incidence of dental anomalies in orthodontic patients,[7,9-11] which are the result of racial and ethnic differences or the influence of local environmental factors.[12-14] The sample involved the Spanish population and, as far as can be ascertained, there are no similar studies about this population. The prevalence of dental anomalies in orthodontic patients ranged between 5.64%[10] and 74.7%.[7] In our study, 22.2% of patients had dental anomalies.

Statistically significant differences according to gender were observed, with a significantly higher occurrence of any dental anomaly in females. At the same time, there were also more female patients in the sample. This coincides with other authors who also found significant differences in the general population[15,16] or subjects with malocclusions.[9,17]

The congenital absence of a permanent tooth (agenesis) was the most frequent anomaly found in the non-syndromic orthodontic sample (16.1%) which has been described to be notably higher in certain syndromes.[18,19] In fact, the literature reports a higher incidence of agenesis in orthodontic subjects than in the general population.[20,21] Excluding third molars, hypodontia ranged between 0.3% and 11.3% in the orthodontic population.[1,22] There are many available treatment alternatives associated with orthodontic procedures for managing tooth space in relation to hypodontia, such as orthodontic space closure, creating space for prosthodontic treatment or tooth transplantation.[23] Planning and decision making for orthodontic treatment is based on an exhaustive diagnosis, as determined by intraoral and extraoral photographs, study models, radiographs, the skeletal pattern, dental malocclusion and tooth size-arch length discrepancies.[24-26] Using orthodontics to open up a space for a future prosthetic replacement or implant is one of the treatment possibilities since a minimum of bone thickness is absolutely necessary for the successful placement of the implant. So, delayed orthodontic space opening is one of the commonest orthodontic strategies used to preserve and increase the implant site.[27]

The incidence of dental impaction (15.4%) in our population study was higher than that found in the literature.[9,17] Dental impaction has a genetic cause, with insufficient arch space interfering with the eruption path.[28] Differences in prevalence can be explained in terms of ethnic differences or sampling techniques, given that *Basdra* et al.[17] only studied the presence of impacted canines with Class II division 2 malocclusion. Dental impaction has been related to other genetically linked dental abnormalities in the same subject.[29,30] Subjects with impacted teeth in our study sample also presented agenesis (16.1%), microdontia (6.5%) and transposed teeth (6.5%). In the clinical practice this is an important consideration since the presence of such disturbances and other clinical signs – such as midline deviations – may alert the clinician to an early diagnosis of an impacted tooth.[31]

In our study, the presence of microdontia was observed in 6.0% of the subjects. Its prevalence has previously been reported as between 0.33% and 7.5%.[10,17,32] This morphological abnormality could be highly influential with regard to other dental anomalies such as taurodontism, agenesis and supernumerary teeth, since there is a statistically significant relationship between them. At the same time, the incidence of microdontia can vary in subjects with malocclusions and between different ethnic groups and environmental factors. This could explain the different data found in the literature.

Dilaceration was observed in 5.0% of pretreatment patients analyzed in this study. The presence and diagnosis of a dilaceration affects root canal treatment, extraction, and orthodontic movement.[7] This abnormal tooth root has been related to significant root shortening during orthodontic treatment,[33] although other factors have recently been associated with this pathology.[34-40] At the same time, the presence of a dental anomaly has been considered a risk factor for root resorption.[41] Considering that 22.2% of our pretreatment orthodontic subjects presented at least one dental anomaly, full and specific consideration should be given to other factors related to external root resorption.[42]

Summarizing the data from our analysis of developmental dental anomalies in subjects with a malocclusion, the following have been concluded:

22.2% of orthodontic patients presented at least one dental anomaly in Southern Europe,

Agenesis was the most common dental anomaly, followed by dental impaction,

There were significant differences in the distribution of dental abnormalities between males and females.

References

1. *Fekonja A:* Hypodontia in orthodontically treated children. Eur J Orthod 2005; 27: 457-460.
2. *Gupta SK, Saxena P, Jain S, Jain D:* Prevalence and distribution of selected developmental dental anomalies in an Indian population. J Oral Sci 2011; 53: 231-238.
3. *Ezoddini AF, Sheikhha MH, Ahmadi H:* Prevalence of dental developmental anomalies: a radiographic study. Community Dent Health 2007; 24: 140-144.
4. *Montasser MA, Taha M:* Prevalence and distribution of dental anomalies in orthodontic patients. Orthodontics (Chicago) 2012; 13: 52-59.
5. *Kotsomitis N, Dunne MP, Freer TJ:* A genetic aetiology for some common dental anomalies: a pilot twin study. Aust Orthod J. 1996; 14: 172-178.
6. *Baydas B, Oktay H, Metin Dagsuyu I:* The effect of heritability on Bolton tooth-size discrepancy. Eur J Orthod 2005; 27: 98-102.
7. *Thongudomporn U, Freer TJ:* Anomalous dental morphology and root resorption during orthodontic treatment: a pilot study. Aust Orthod J 1998; 15: 162-167.
8. *Hamasha AA, Al-Khateeb T:* Prevalence of fused and geminated teeth in Jordanian adults. Quintessence Int 2004; 35: 556-559.
9. *Uslu O, Akcam MO, Evirgen S, Cebeci I:* Prevalence of dental anomalies in various malocclusions. Am J Orthod Dentofac Orthop 2009; 135: 328-335.
10. *Altug-Atac A T, Erdem D:* Prevalence and distribution of dental anomalies in orthodontic patients. Am J Orthod Dentofac Orthop 2007; 131: 510-514.
11. *Cadenas-Perula M, Yañez-Vico RM, Solano-Reina E, Iglesias-Linares A:* Effectiveness of biologic methods of inhibiting orthodontic tooth movement in animal studies. Am J Orthod Dentofacial Orthop 2016; 150: 33-48.
12. *Mendoza-Mendoza A, Iglesias-Linares A, Yañez-Vico RM, Abalos-Labruzzi C:* Prevalence and complications of trauma to the primary dentition in a subpopulation of Spanish children in southern Europe. Dent Traumatol 2015; 31: 144-149.
13. *Mendoza-Mendoza A, González-Mallea E, Iglesias-Linares A:* Intrusive luxation in primary teeth: a case report. J Clin Pediatr Dent 2015; 39: 215-218.
14. *Caleza C, Yañez-Vico RM, Mendoza A, Iglesias-Linares A:* Childhood obesity and delayed gratification behavior: a systematic review of experimental studies. J Pediatr 2016; 169: 201-7.e1.
15. *Brook AH:* Dental anomalies of number, form and size: their prevalence in British schoolchildren. J Int Assoc Dent Child 1974; 5: 37-53.
16. *Bergstrom K:* An orthopantomographic study of hypodontia, supernumeraries and other anomalies in school children between the ages of 8–9 years. An epidemiological study. Swedish Dent J 1977; 1: 145-157.
17. *Basdra EK, Kiokpasoglou M, Stellzig A:* The Class II Division 2 craniofacial type is associated with numerous congenital tooth anomalies. Eur J Orthod 2000; 22: 529-535.
18. *Molina-Solana R, Yáñez-Vico RM, Iglesias-Linares A, Mendoza-Mendoza A, Solano-Reina E:* Current concepts on the effect of environmental factors on cleft lip and palate. Int J Oral Maxillofac Surg 2013; 42: 177-184.
19. *Yáñez-Vico RM, Iglesias-Linares A, Gómez-Mendo I, Torres-Lagares D, González-Moles MÁ, Gutierrez-Pérez JL,* et al.: A descriptive epidemiologic study of cleft lip and palate in Spain.Oral Surg Oral Med Oral Pathol Oral Radiol 2012; 114: S1-4.
20. *Horowitz JM:* Aplasia and malocclusion: a survey and appraisal. Am J Orthod Dentofac Orthop 1966; 52: 440-453.
21. *Thilander B, Myrberg N:* The prevalence of malocclusion in Swedish schoolchildren. Scand J Dent Res 1973; 81: 12-21.
22. *Endo T, Ozoe R, Kubota M, Akiyama M, Shimooka*

S: A survey of hypodontia in Japanese orthodontic patients. Am J Orthod Dentofac Orthop 2006; 129: 29-35.

23. *Kokich Jr VO, Kinzer GA:* Managing congenitally missing lateral incisors. Part I: canine substitution. J Esthet Rest Dent 2005; 17: 5-10.
24. *Turpin DL:* Treatment of missing lateral incisors. Am J Orthod Dentofac Orthop 2004; 125: 129.
25. *Iglesias-Linares A, Yáñez-Vico RM, Moreno-Manteca B, Moreno-Fernández AM, Mendoza-Mendoza A, Solano-Reina E:* Common standards in facial esthetics: craniofacial analysis of most attractive black and white subjects according to People magazine during previous 10 years. J Oral Maxillofac Surg 2011; 69: e216-24.
26. *Orce-Romero A, Iglesias-Linares A, Cantillo-Galindo M, Yañez-Vico RM, Mendoza-Mendoza A, Solano-Reina E:* Do the smiles of the world's most influential individuals have common parameters? J Oral Rehabil 2013; 40: 159-170.
27. *Borzabadi-Farahani A:* Orthodontic considerations in restorative management of hypodontia patients with endosseous implants. J Oral Implantol 2012; 38: 779-791.
28. *Sambataro S, Baccetti T, Franchi L, Antonini F:* Early predictive variables for upper canine impaction as derived from posteroanterior cephalograms. Angle Orthod 2004; 75: 28-34.
29. *Baccetti T:* A controlled study of associated dental anomalies. Angle Orthod 1998; 68: 267-274.
30. *Peck S, Peck L, Kataja M:* The palatally displaced canine as a dental anomaly of genetic origin. Angle Orthod 1994; 64: 249-256.
31. *Jiménez-Castellanos E, Orozco-Varo A, Arroyo-Cruz G, Iglesias-Linares A:* Prevalence of alterations in the characteristics of smile symmetry in an adult population from southern Europe. J Prosthet Dent 2016; 115: 736-740.
32. *Clayton JM:* Congenital dental anomalies occurring in 3557 children. J Dent Child 1956; 23: 206-208.
33. *Iglesias-Linares A, Hartsfield JK Jr:* Cellular and molecular pathways leading to external root resorption. J Dent Res 2017; 96: 145-152.
34. *Iglesias-Linares A, Yáñez-Vico RM, Ballesta-Mudarra S,* et al.: Postorthodontic external root resorption is associated with IL1 receptor antagonist gene variations. Oral Dis 2012; 12: 198-205.
35. *Iglesias-Linares A, Sonnenberg B, Solano B, Yañez-Vico RM, Solano E, Lindauer SJ,* et al.: Orthodontically induced external apical root resorption in patients treated with fixed appliances vs removable aligners. Angle Orthod 2017; 87: 3-10.
36. *Iglesias-Linares A, Morford LA, Hartsfield JK Jr:* Bone density and dental external apical root resorption. Curr Osteoporos Rep 2016; 14: 292-309.
37. *Iglesias-Linares A, Yañez-Vico RM, Moreno-Fernández AM, Mendoza-Mendoza A, Orce-Romero A, Solano-Reina E:* Osteopontin gene SNPs (rs9138, rs11730582) mediate susceptibility to external root resorption in orthodontic patients. Oral Dis 2014; 20: 307-312.
38. *Iglesias-Linares A, Yañez-Vico RM, Ortiz-Ariza E, Ballesta S, Mendoza-Mendoza A, Perea E,* et al.: Postorthodontic external root resorption in root-filled teeth is influenced by interleukin-1β polymorphism. J Endod 2012; 38: 283-287.
39. *Iglesias-Linares A, Yañez-Vico RM, Ballesta S, Ortiz-Ariza E, Mendoza-Mendoza A, Perea E,* et al.: Interleukin 1 gene cluster SNPs (rs1800587, rs1143634) influences post-orthodontic root resorption in endodontic and their contralateral vital control teeth differently. Int Endod J 2012; 45: 1018-1126.
40. *Iglesias-Linares A, Yañez-Vico RM, Ballesta-Mudarra S, Ortiz-Ariza E, Mendoza-Mendoza A, Perea-Pérez E,* et al.: Interleukin 1 receptor antagonist (IL1RN) genetic variations condition post-orthodontic external root resorption in endodontically-treated teeth. Histol Histopathol 2013; 28: 767-773.
41. *Thongudomporn U, Freer TJ:* Anomalous dental morphology and root resorption during orthodontic treatment: a pilot study. Aust Orthod J 1998; 15: 162-167.
42. *Topkara A, Karaman AI, Kau CH:* Apical root resorption caused by orthodontic forces: A brief review and a long-term observation. Eur J Dent 2012; 6: 445-453.

Address: School of Dentistry, University of Seville
C/Avicena s/n 41009 Seville, Spain
Tel.: +34 639563909
e-mail: rosayanezvico@gmail.com

Received: 26th February 2017
Accepted: 23rd April 2017