DATABASE ABOUT THE ANOMALIES THAT MORE DOMINANT OCCURS IN TEETH AND JAWS IN MISSAN CITY CHILDREN

REPUBLIC OF IRAQ MINISTRY OF HIGH EDUCATION SCIENTIFIC RESEARCH UNIVERSITY OF MISAN COLLEGE OF DENTISTRY FIFTH STAGE (2022-2023) A research submitted to College of Dentistry, University of Misan as a requirement for bachelor degree in dentistry

SUPERVISED BY
dr .majed hussein majed

PREPARED BY
Zahraa Raad Zuid

...DEDICATION Our study trip has reached its end after exhaustion and hardship And we are concluding the research of our graduation with all vigor and vigor .We are grateful to everyone who has been credited with our journey ,and help us ,even with ease. Parents, family , friends , and esteemed teachers. We present to you a study of our graduation.. . And do not forget the greatest credit to our research supervisor (Dr. majed hussein majed), by providing us with valuable and useful information. Thank you very much doctor...

Abstract ;The identification of specific patterns of dental anomalies would allow testing the hypothesis that certain genetic and environmental factors contribute to distinct dental anomaly subphenotypes. The genetic control of dental development represents a complex series of events, which can very schematically be divided in two pathways: specification of type, size and position of each dental organ, and specific processes for the formation of enamel and dentin Several genes linked with early tooth positioning and development

Material and methods ; This cases evaluated from Visiting Specialist Centre **In_Amarah city ,missan province/iraq Amarah city is centre of missan province**. give the more dominant anomalies
Class 111 maloclussion more dominant occurs in males,congenital missing teeth more dominant occurs in female,Mandibular anterior crowding no difference between female and male and FUSION OF THE TEETH .

INTRODUCTION

Many dental anomalies have also been reported to be associated with tooth agenesis, including small tooth size,6 peg-shaped upper lateral incisor The aetiology of dental anomalies remains largely unclear. Some investigations have already shown that different phenotypic forms of tooth agenesis are probably caused by different genes which can very schematically be divided in two pathways: specification of type, size and position of each dental organ, and specific processes for the formation of enamel and dentin. By contrast, genes involved in enamel (AMELX, ENAM, MMP20, and KLK4) and dentin (DSPP) structures are highly specific for tooth. Mutations in these genes have been identified as causes of amelogenesis imperfecta, dentinogenesis imperfecta, dentin dysplasias and anomalies of teeth number (hypo-, oligo and anodontia), mostly using mouse teeth as models, have indicated that the position, number, size and shape of different teeth are under genetic control [3]. Tooth development is

initiated by signals from the epithelial dental lamina to the mesenchyme [4]. Thereafter, the mesenchyme regulates epithelial morphogenesis. Regulation of development is mediated by complex interactions between the epithelium and mesenchyme. The centre of the epithelial bulk: the enamel knot is functioning as an important signalling centre regulating tooth shape [5]. During tooth development, the epithelium and mesenchyme interact through different families of signalling molecules and their receptors. These comprise the transforming growth factor β (TGFβ), bone morphogenetic proteins (BMP), fibroblast growth factors (FGF), epidermal growth factor (EGF), and the hedgehog (Hh) and wingless (Wnt) families [6]. In addition to these signals, the model proposed by Thesleff [7] also includes several genes, which are regulated by the signals in the responding tissues (Fig. 1). Mutations in many of these genes already have been shown to cause dental defects in mice as well as in humans.

PEG-SHAPED MAXILLARY LATERAL INCISORS:

In the morphodifferentiation stage, the formative cells are arranged to outline the form and size of the tooth. This process occurs before matrix deposition. The morphologic pattern of the tooth becomes established when the inner enamel epithelium is arranged so that the boundary between it and the odontoblasts outlines the future dentinoenamel junction. Disturbances and aberrations in morphodifferentiation lead to abnormal forms and sizes of teeth. Resulting conditions include peg teeth, other types of microdontia, and macrodontia.

of teeth. Resulting conditions include peg teeth, other types of microdontia, and macrodontia. Anomalies of maxillary lateral incisors including shape, size or even agenesis are quite common, with a prevalence varies from 1.6% to 4.9% with higher prevalence in women than men. 1 , 8 They can be either unilateral or bilateral touching the left or the right side with a higher incidence on the left-side dental arch. 7 Peg-shaped anomaly of lateral incisors is one of the most common form of localized microdontia that affects the shape of permanent maxillary lateral incisors (peg lateral). It is characterized by the reduction of the incisal mesiodistal width compared with the cervical region. 6 This shape anomaly leads to anterior diastemas, which causes functional and esthetic major concerns for the affected patients. 4 , 5 Many treatment options of peg-shaped lateral incisors are available including one or many of these clinical procedures: no treatment, orthodontic treatment first, direct or indirect composite restorations, bonded ceramic crowns or veneers, 3 and finally, extractions and implant placement. PMCID: PMC8888921 [

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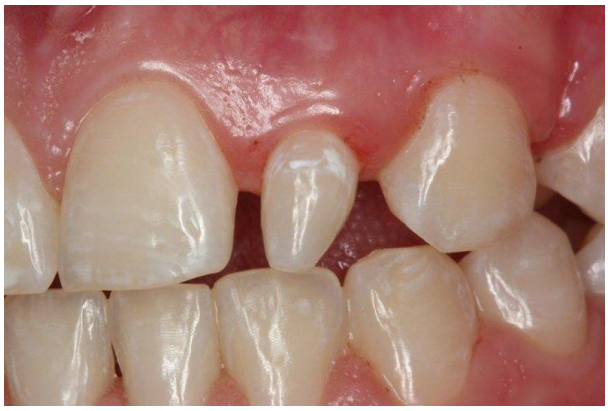


Direct composite resin restoration (right lateral incisor)

CONGENITALLY MISSING TEETH

Congenitally missing teeth (CMT), or as usually called hypodontia, is a highly prevalent and costly dental anomaly. Besides an unfavorable appearance, patients with missing teeth may suffer from malocclusion, periodontal damage, insuffi cient alveolar bone growth, reduced chewing ability, inarticulate pronunciation and other problems. Treatment might be usually expensive and multidisciplinary. This highly frequent and yet expensive anomaly is of interest to numerous clinical, basic science and public health fi elds such as orthodontics, pediatric dentistry, prosthodontics, periodontics, maxillofacial surgery, anatomy, anthropology and even the insurance industry

ETIOLOGY

is a result of disturbances during the early stages of development[15] and is suggested as a mild dysplastic expression of the ectoderm.[20-23] When a primary tooth is congenitally absent, its permanent counterpart might also be missing.[22,24] Genetics plays a crucial role in congenital dental aplasia,[4] as confi rmed by studies on monozygotic twins Interestingly, the pattern of CMT can differ between monozygotic twins, possibly pointing to additional underlying mechanisms,[25] such as epigenetic factors which might be implied by simultaneous occurrence of two anomalies.[4] This multifactorial etiology can include environmental factors as well, since a combination of environmental and genetic factors might contribute to the occurrence of dental agenesis.[4,8,14,19,28] These include infection, trauma and drugs, as well as genes associated with about 120 syndromes,[2,3,6,8,19,22,29-35] such as cleft lip, cleft palate or both,[36] ectodermal dysplasia[9,27,37] and Down, Rieger and Book syndromes.[9,22] A possible general explanation is that except in hereditary cases, CMT has greater occurrence likelihood when the dental germ is developing after the surrounding tissues have closed the space needed for the tooth development.[3,38] Other investigations demonstrated that delays in tooth development and reductions in tooth size correlate with advanced CMT.[3,39-41] Both of these might accord with the terminal reduction theory.[3,42] Furthermore, it is suggested that anterior agenesis may depend more on genes while posterior missing might be sporadic.[23] CMT can form in isolation as well. Isolated cases are more common than syndromic type[17] and might be familiar or sporadic.[22] The isolated condition can follow autosomal dominant,[45-47] autosomal recessive[48,49] or X-linked[50] patterns of inheritance, with remarkable variation in both penetrance and expressivity.[17,20,22,51] Different subphenotypes of dental agenesis might be probably caused by various genes.[52-57] Mutations in genes such as MSX, PAX9 or TGFA might cause CMT in different racial groups.[9,14,30,31,47,56,58-60] Among the homeobox genes, MSX1 and MSX2 play an important role in mediating direct epithelial-mesenchymal interactions during craniofacial bone and tooth development.[14,17,61] The autosomal-dominant CMT might be correlated with a mutation in the MSX1 and PAX9 genes.[9,17,31,47,58,59,62] MSX1 mutations

affect predominantly the second premolars and third molars, sometimes in combination with other types of teeth like the fi rst molars.[17] On the other hand, in more common cases of incisorpremolar type of dental agenesis, MSX1 is less likely to play a role as the causative locus for this type of CMT.[17,53] In addition, PAX9 and TGFA are associated with congenital missing by interacting between MSX1 and PAX9.[14,56] A recent study showed a novel mutation in MSX1 gene responsible for CMT of the second premolars and third molars only.[63]

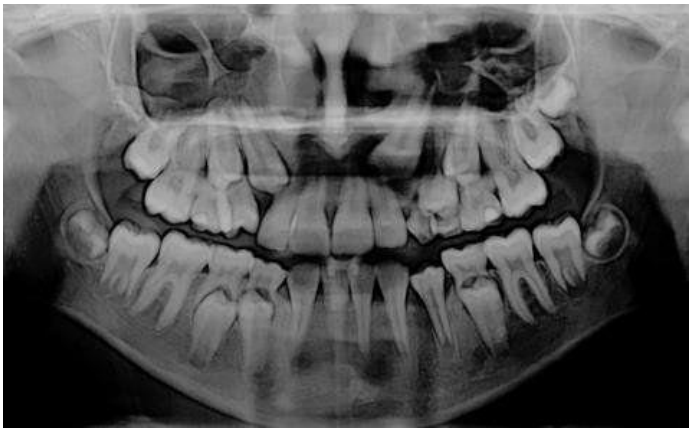
THE PREVALENCE OF DENTAL AGENESIS

In the primary dentition, the CMT is not frequent, being between 0.1% and 2.4However, primary dental aplasia is usually followed by permanent tooth missing.[8,19,34] The prevalence of CMT in the permanent dentition excluding the third molars ranges between 0.15% and 16.2% [Table 1] in studies varying in size from about 200 subjects to] Japanese people showed the highest rates both in deciduous and permanent dentitions The CMT prevalence was found to differ between continents and races, but unlikely over time.[34] The CMT prevalence in third molars has been reported over a rather broad range, between 5% and 37%.[22] For example, Ghaznawi et al. [134] reported 5.5% of wisdom tooth missing in Saudi Arabia, while Varela et al. [29] observed that 11.5% of a population from Spain had missing of third molars. Other rates might be much greater. For instance, Afi fy and Zawawi[155] and Silva Meza[141] reported 24% third molar absence rates in Saudi Arabia and Mexicans, respectively. Sheikhi et al. [60] have reported 34.8% missing prevalence of Iranians' third molars. Australian aborigines and perhaps African Blacks might have a low chance of dental agenesis.[8,19] Indians have shown very small prevalence rates, as two out of three studies in India had rates less than 1% and the other one had about 4% prevalenceThe different rates reported could be explained by different measurement approaches or other methodologiesand ethnic backgroundsIn contrast, X-ray is a carcinogen factor and cannot be prescribed without any treatment needsThus, researchers need to use previously taken radiographic images. In very rare cases, such images have been taken from randomly selected subjects (epidemiological samples such as patients attending mandatory public health

TREATMENT OF CONGENITALLY MISSING TEETH

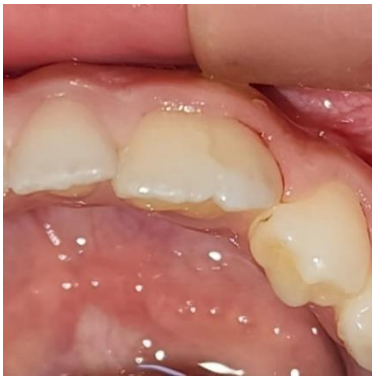
treatment would be usually diffi cult.[137] It might represent an interdisciplinary challenge for specialists in oral and maxillofacial surgery, operative dentistry, pediatric dentistry, orthodontics and prosthodontics.[9,22,65,171,178-181] General or pediatric dentists can facilitate multidisciplinary treatments by diagnosing congenital absence of primary teeth and then through early referrals of patients; as the absence of primary teeth highly associates with missing of permanent successors.[8,19,34] They might also ensure the retention of reduced number of teeth,[8,19] in cases such as palatal impaction of the maxillary canines caused by the missing laterals, in which early extraction of deciduous canines might guide the eruption of the permanent ones into the correct position.[8,182] This necessitates the early evaluation of the number of missing teeth and the consideration of the CMT risk factors, as

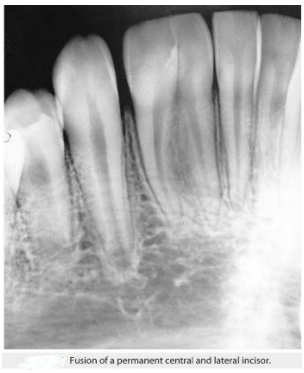
well as the size and number of teeth remaining in both arches in planning and managing treatment.[3,19,22] The type of malocclusion, severity of crowding and facial profi le are of major concern in determining the fi nal treatment plan.[22] Bone volume is related to facial esthetics such as smile, and should be considered in treatment planning as well.[65,183] During treatment planning, possible changes in the craniofacial morphology associated with CMT should be as well borne in mind.[14] Another therapeutic challenge is the need to carry out treatment in the growing young patient.[9,179] While treatment should be initiated during adolescence,[9] interim treatment should begin in around 7-9 years of age before the affected children realize they are different from other children.[9,19,179] The edentulous space can be either left open for prosthetic restoration, or closed by orthodontic means.[13,14,164] Other treatment modalities might include autotransplantation[14,184] or protraction[14,185] of the third molars, which are otherwise extracted, in order to substitute for the edentulous region or to increase the number of occluding teeth.[14] In prosthodontic treatments, transplantation is a better choice than implanting, since osseointegrated implants are contraindicated in the growing alveolar bone.[22] Successful autotransplantation of teeth ensures the stability of alveolar bone volume due to physiological stimulation of the periodontal ligament.[22] Implant treatment is postponed until the jaws have stopped growing in adolescence.[22,186-188] It is also possible to close the lateral space in crowded maxillae and recontour the canine into the lateral's shape. In an aligned maxillary arch, the distributed excess space can be localized and then restored using prosthetic approaches.[22] Absent lower incisors need esthetic and functional camoufl age regarding the relationship between the maxillary and mandibular anterior teeth.[65] In crowded jaws, the missing premolar spaces can be used as one of the extraction spaces for arch alignment.[22] In uncrowded jaws with missing permanent premolars, the primary second molar might be left in situ. However, since, there is the risk of infra-occlusion or progressive root resorption, it might be eventually extracted and replaced with an implant or and autotransplanted tooth.[22] The treatment of severe cases is complex and should be performed in centers such as "Hypodontia Clinics"[65,189] with access to pediatric dentistry, orthodontics, prosthodontics and oral and maxillofacial surgery.[9,22,171,180,181] It should be noted that orthodontic/prosthodontic treatments might compromise esthetics and periodontal health.[22,190,191]



FUSION OF THE TEETH

Fusion describes the joining of two (or more) independently developing primary or permanent teeth that began from their own unique tooth germs. Fusion leads to the formation of a single large tooth and reduces the total number of visible teeth in the affected arch by one (or more). At the radiographic level, fusion usually affects the length of the teeth from the crowns (enamel/dentin) to the roots (cementum) in such a manner that the fused teeth maintain independent pulp chambers and root canals . When a joining affects only the roots (cementum) of neighboring teeth, the condition is specifically termed concrescence. Fusions can occur in both the primary and permanent dentitions. Dental fusion is usually localized to the anterior of the mouth, with the maxillary central and lateral incisors being the most frequently affected teeth. Fusions can occur within families, suggesting a hereditary pattern of occurrence. In rare circumstances, bilateral fusions and triplicate fusions of primary teeth have been observed necessitating the placement of a restoration. Furthermore, a frequent finding when two primary teeth fuse is the developmental absence of one of the corresponding permanent teeth. Consequently, patients with fused teeth often require a multidisciplinary approach for their dental care involving pediatric dentistry, endodontics, surgery, restorative dentistry, and orthodontics. Surgical sectioning and separation of fused teeth may be possible, and although dentin is exposed, such teeth are easily moved orthodontically without the risk of ankylosis. Although diagnosis may be difficult using traditional radiographic techniques due superimposition of adjacent structures or other teeth, cone beam computed tomography is useful in determining the extent and exact location of the fusion Gemination or fusion is a rare to the occurrence in the mandibular posterior teeth. Fusion of permanent and supernumerary teeth usually occurs in the anterior region of the Maxilla. HowEve, fusions involving molars are rarely reported. Fig Fusion of a permanent lateral incisor and canine(case : Dr. Akram Alhuwaizi)





Fusion of a permanent central and lateral incisor.

Table l : more dominant anomalies in missan city children

peg-shaped maxillary lateral incisors

congenital missing teeth more dominant occurs in female

FUSION OF THE TEETH

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