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Non-Syndromic Over Retained Primary Teeth In The Presence Of Permanent Successor Teeth. – A Rare Case Report

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Abstract
Although the delayed exfoliation of primary teeth among children is a common dental problem, but majority of the issue is associated with agenesis of permanent tooth or other finding like supernumerary teeth, odontoma, cyst, impaction of successor teeth. Not only the local condition so many systemic disease and broad spectrum of syndrome could be associated in such condition, and in very rare cases the cause of retention is not at all appreciable or may be idiopathic. Such problem may cause dental disorders, so providing a meticulous history and careful clinical examination could prevent this condition. Physician consultation to exclude any endocrinal abnormality is also helpful. The clinical implications of genetically controlled patterns of dental abnormalities are very important in the early diagnosis and appropriate orthodontic intervention. This paper reports an unusual case in which all the primary teeth are retained with no abnormality in the formation of successor teeth visible in the radiograph. Interestingly the patient family history of over retention of primary teeth is positive. This report describes questions concerning the aetiology of over retention of primary teeth.

Keywords*: Idiopathic over retention of primary teeth, persistent primary teeth, delayed eruption of Permanent teeth.*

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I. Introduction

Humans are diphyodonts, which means that they havetwo successive sets of teeth: primary and permanent. Teeth, an important part of the dentomaxillofacial complex, are an important biological marker of maturity.The chronology and sequence of eruption of primary and permanent teeth are important milestones during a child's development, as the eruption of tooth strongly influences the normal development of the craniofacial complex.

Eruption is often used to indicate the moment of emergence of the toothinto the oral cavity. This moment consists of tightly programmed series of signalling interactions between cells of the connective tissue sac surrounding the tooth (dental follicle) and surrounding alveolus. It is a dynamic process that encompasses completion of root development, establishment of the periodontium, and maintenance of a functional occlusion.Various cellular, molecular and genetic determinants are involved in this mechanism.[1]

Normal teeth eruption into oral cavity occurs over a broad chronological age rangeand can be influenced by a number of factors. Genes,hormones,and calories(nutrition) are the broad three categories on which the tooth development and eruption as well as exfoliation depends upon.[2]The timing of eruption of dentition varies between ethnic groups, gender, socioeconomic and nutritional factors, carious condition, fluorides , congenital abnormalities such as supernumerary teeth, down's syndrome, cleidocranial dysplasia and environmental secular trends.[3-5]As, the eruption is under strong genetic control, and the estimates of narrow-sense heritability are over 70% in primary tooth emergence.[6]. Studies with monozygotic twins shows a concordance rate of 0.9 [2]. Dizygotic twins and siblings show a lower concordance rate but it is still higher than

in unrelated individuals. There is also ethnicity- and sex-related differences in the timing of primary tooth eruption.[7] However, external environmental factors also make significant contributions to the timing of the primary tooth eruption. Maternal exposure to tobacco during pregnancy[8,9], infant birth weight[10,11], birth length[12], nutritional state at birth and at postnatal timepoints[13], gestational age[14], method of infant feeding[9,15] and socioeconomic situation[16] have been reported to be significant determinants of the eruption of primary teeth. Delayed tooth eruption has been reported in premature infants[1,17] with small gestational age and low birth weight and in those with systemic disorders, such as hypothyroidism[18], while accelerated tooth eruption has been observed in children whose mothers smoked during pregnancy[8,9] as well as in those with childhood obesity and diabetes mellitus[19].

Similarly,Permanent teeth eruption is a complex process that can be influenced by a number of general factors: genetics, nutrition, preterm birth, socioeconomic factors, body height and weight, craniofacial morphology, hormonal factors and various systemic diseases.[20]In the normal developmental sequence, exfoliation of primary tooth usually precedes permanent tooth eruption. Retention of primary teeth beyond their expected exfoliation date is encountered relatively frequent. Generally initial resorption begins between 1 and 3 year after the apical closure of the deciduous teeth.Exfoliation follows by approximately 3 years. Certainfactors, (e.g.; congenital absence of permanent tooth follicle) delay the root resorption of primary tooth.Dental caries in a primary molar remarkably accelerates the root resorption and eruption of permanent teeth.Along-standing periapical abscess of a primary tooth may also accelerate eruption of underlying of permanent teeth.Early extraction of primary molar delays gingival eruption of the successor.[21] Whereas,Delayed tooth eruption is the emergence of a tooth intothe oral cavity at a time that deviates from norms establishedfor different ethnicities and sexes, leading to a persistentprimary tooth.[22]Hence cases with over retained and persistent primary tooth beyond the time of normal exfoliation frequently occurs. This may lead to delayed eruption or ectopic eruption of permanent tooth. [23]This condition is not uncommon in mixed dentition stage.Onestudy reported that about 20.85% of children had overretained primary teeth with highest prevalence in children of 10 years old. In this study, 66.23% of over-retained primary tooth occurred in mandible with highestprevalence in second lower primary incisor and 67.55%of over-retained primary teeth were causing malocclusion.[24]

The possible causes for this condition are1) Absences of the permanent teeth(most common cause), 2) The presence ofdense sclerotic bone around the crown of permanenttooth, 3) Failure in normal resorption of roots of the primary tooth, 4) Deviation in the eruption of the teeth and 5) Existence of pathology, such as cysts, tumours, and odontoma under the primary tooth that results in the impaction of successor teeth. Still exact reasons for the persistence of primary teeth are yet unknown. The most common over retained tooth is usually primary mandibular molar and secondly primary maxillary canine.[25,26]

This article presents the unusual case report of a non-syndromic girl with over retained primary teeth at the age of 11 yr., radiographically intact root length along with presence of all permanent teeth beneath the primary roots.

II. Case Report

A 11year old girl reported to the department of Paediatric dentistry, Dr RAhmed dental college and hospital with the chief complaint of un-aesthetic teeth.Patient's oral examination was done, Intraoral examination did not show any soft tissue abnormalities.

A clinical dental examination revealed the presence of the following teeth in hermaxilla:16,55,54,53,52,51,61,62,63,64,65 andmandible:46,85,84,83,82,81,71,72,73,74,75,36 respectively. Thus all the primary teeth were retained. However, 63,64 were carious and 54 decayed and only root stumps was visible.

In the view of this findingit was decided to perform a panoramic radiograph in order to guide the diagnosis. The radiograph revealed that all the deciduous teeth,with almost intact root length,and all the Succedaneous teeth with almost 2/3rd root formed were present beneath the roots of primary teeth. The nonerupted teeth were covered with bone and had well-circumscribed follicles. However, continued root development of the permanent teeth was noted, without progress toward the alveolar crest.No ankylosis of the deciduous teeth could be seen on clear scale view of the radiograph. Further examination showedthat the crown size of deciduous teeth was normal with generalized attrition of mandibular teeth, the discolouration of teeth was more pronounced. Bone age corresponded to chronologic age. On physical examination it was seen that patient waswell built, active and mentally alert. No abnormality was found on clinical examination of the chest, abdomen, cardiovascular and central nervous system. No cutaneous pigmentation or other congenital abnormality was present; there was no evidence of endocrinal disturbance.

Extraoral examination revealed thatthe patient had a relatively shorter lower facial height with competent lip. No medical history or dentofacial trauma was reported by the patient. the patient was referred to a medical consultant for general examination. Laboratory investigation was done mentioned in the table1:

Theseinvestigations were within normal range. Further, mineral and protein count werewithin normal limit.Thus,no significant alteration was observed with biochemical analysis.The history revealed that the patient

was born as a full-term normal delivery to a healthy mother with the birth weight of 2.5 kg.Based on the information given by patient's parents, her primary teeth started erupting between the age of 6-8 month, without any abnormality. There was no history of radiation exposure during childhood and also in the past years.

Family history showed that her elder sister who is 18 yr. old now was suffering from the same issue , on clinical examinations no soft tissue abnormalities was seen , similarly dental examination revealed the presence of following teeth in maxilla :17,16,15,14,53,12,11,21,22,63,24,25,26,27 and the mandible 47,46,45,84,83,42,41,31,32,33,74,75,36,37. She also gave the history of 73 extracted 9 months back , and the Succedaneous teeth i.e. 33 is still under the stage of eruption that has not attain the level of proper occlusion, only the occlusal tip was visible through the gingiva .

Her radiographic investigation was done, the panoramic image revealed the Succedaneous teeth i.e. 13,23 with respect to maxilla and 44,43,34,35 were present underneath the retained primary tooth root.In the view of this findings she under vent the similar laboratory investigation(T3,T4,TSH,PTH,Ca,Phosphate,Vitamin-D) and results were in normal range.In the absence of any other signs, the diagnosis was primary retention of the permanent teeth with no associated systemic or endocrinal abnormality or syndrome.

III. Discussion

Eruption is a developmental process, in which a toothmoves from its crypt position through thealveolar process intothe oral cavity to its final position of occlusion with its antagonist.[1] The delay in the eruption and emergence of a tooth is commonlyassociated with a primary tooth retained beyond the time ofnormal exfoliation, known as a "persistent primary tooth."Both delayed tooth emergence and persistent primary teeth arecorrelated with the complex mechanism of tooth eruption.[23]

The most accepted mechanism of primary tooth exfoliation involves the pressure resorption of primary tooth root evoked by the pressure exerted by Succedaneous tooth through its peri-coronal follicle or differentiation of monocyte from periodontal ligament into odontoclast. The odontoclast then resorbs the root of primary tooth in a similar manner as osteoclasts during bone remodelling or resorption without inflammatory response. The main difference between the bone and tooth biology is that the bone undergoes constant physiological turn over whereas teeth only in the case of primary dentition undergo normal resorption.[1]

Similar to bone physiology the receptor activator of NF-kappaB (RANK), its ligandRANKL, and the decoy receptor osteoprotegerin (OPG) areessential central regulators of osteoclast development andfunction. Studies with animal models demonstrated that thegenes that code for RANK/RANKL/OPG are differentially expressed at specific sites during the eruption process.[27] RANKL activation stimulate the resorptive cell and cause the resorption of primary tooth root and bony crypt coronal to the permanent tooth eruption and RANK/OPG coding the positive effect over the bone formation below the erupting permanent tooth.[28,29]

Studies with animal models have highlighted the important role ofRANKL in tooth emergence, since RANKL-null mice exhibitno tooth eruption.[30] ARID et Al. conducted a study to find the correlation between gene expression of RANK/RANKL/OPG in children with delayed tooth emergence and persistent primary teeth, periapical tissue from 15 children with persistent primary teeth were taken , through quantitative PCR analysis it was observed that reduced relative gene expression of RANKL in periapical tissue is associated with persistent primary teeth.Thus, the problem in expressivity of this gene could be one reason of persistent of primary tooth.[22]

Other relevant findings in the case of persistent primary tooth could be the absence of permanent tooth that has proved to be most common cause followed by impaction, abnormal position, and late eruptionof successor teeth.[23]

In normal dentition, the primary tooth roots undergogradual resorption concurrently with the eruption of thesuccessors. The normal interrelationship between the eruption of permanent teeth and the resorption of theprimary teeth is well described by Haavikko, 1973.[31]Who said that the primary root resorption starts when the permanent root achieve ¼ th of its length .Whereas,Matasumoto et al [32]reported that root resorption of primary teeth rapidly progresses up to the labial surface when ¾ of the root length of the successive permanent teeth is formed,but theresorption of the primary tooth root is also generally viewedas a process that can occur when the underlying permanenttooth is absent (Rune and Sarnas, 1984).[33] Another study done by Aktan et al suggested that persistent teeth were related to the congenital absence of successor teeth, less resorption of the primary teeth roots were encountered. On the other hand, if the reason for the persistence of primary teeth was the impaction of the successor teeth, more resorption of their root was encountered.[23]Several studies have been done to observe root resorption in subjectswith agenesis of the successors.[34,35]

Pathological cause of over retention of primary teeth can be rampant caries, the presence of a calcifying odontogenic cyst, intraluminal adenomatoid odontogenic tumour, monostotic fibrous dysplasia, and chronic malnutrition.Also, accumulative and quantitative effect of rampant caries may delay the beginning of the root resorption process. Chronic malnutrition reflected by a stunted growth pattern has been related to delayed exfoliation of primary teeth.[36]

The vital role of nutrition is precisely defined by Garn et al,some of his important findingswith respect to tooth eruption are, 'the taller and heavier children are slightly advanced dentally while it is apparent that stunting (retarded linear growth) is more strongly associated with delayed tooth eruption.Also,positive correlation between body height andweight and teeth emergence has been establishedin the earlier studies.[2]

O'Connell *et al*. found that tooth eruption disorder is a part of the hyper IgE syndrome. This disorder occursbecause of delayed primary tooth exfoliation rather than a developmental delay in the formation of the permanent dentition. [37]The persistence of Hertwig epithelial root sheath is unusual and may be related to the lack ofresorption of the primary teeth. They found that 75% of patients older than 7 years reported problems with permanent tooth eruption, as evidenced by retained primary teeth or the need for optional extractions of primaryteeth to allow eruption of their successors.It is the proven fact that pressure of eruption permanent tooth plays a contributory role in setting resorption but the present of a permanent successor is not a prerequisite for this to occur.[1]The eruption process of permanent teeth is regulated by various factors and therefore, these factors have an indirect effect on resorption courses of the primary tooth root.[38,39]

Enlightening some of the factor associated with permanent tooth emergence failure could be divided into local or systemic.

Local factor includes;

-Present of supernumerary teeth that is the condition can be associated with one or few numbers of unerupted teeth[40]

-Odontoma or other tumour associated with primary or permanent teeth.[41-43]

- Injuries to deciduous teeth have also been implicated as a cause of DTE of the permanent teeth. Smith and Rapp[44,45]

-Cystic transformation of a nonvital deciduous incisor might also cause delay in the eruption of the permanentsuccessor.[46]

-The traumatized deciduous incisor might become ankylosed or delayed in its root resorption this also leads to the over retention of the deciduous tooth and disruption in the eruption of its successor.[44]

- Succedaneous teeth isoften delayed after the premature loss of deciduousteeth before the beginning of their root resorption. Thiscan be explained by the abnormal changes that might occur in the connective tissue overlying the permanenttooth and the formation of thick, fibrous gingiva.[47]

- X-radiation has also been shown to impair tootheruption.Root formationimpairment, periodontal cell damage, and insufficientmandibular growth also are thought to be linked to tootheruption disturbances due to x-radiation.[48]

Systemic factors that play vital role in the emergence phenomena are:

- As stated by Garn et al [2], nutrition plays an important role in dentition development and eruption but according to Alvarez JO[49] the influence of nutrition on calcification anderuption is less significant compared with other factors,because it is only at the extremes of nutritive deprivation that the effects on tooth eruption have beenshown.
- Disturbances of the endocrine glands usually has a profoundeffect on the entire body, including the dentition.Hypothyroidism, hypopituitarism, hypoparathyroidism, and pseudohypoparathyroidism are the most commonendocrine disorders associated with Delay tooth eruption.[50,51]
- Retardation of dental growth and development inpreterm babies has been reviewed by Seow. The permanent teeth showed asignificant mean delay in dental maturation of approximately 3 months in very low birth-weight babies (birthweight of 1500 g).[52,53]
- Other systemic conditions associated with impairment of growth, such as anaemia (hypoxic hypoxia,histotoxic hypoxia, and anaemic hypoxia)[54]and renal failure,[55] have also been correlated with Delay tooth eruption and otherabnormalities in dentofacial development
- As we already know tooth eruption is a complex phenomenon which is associated with multiple gene expression and their respective functions e.g. the various eruption regulatory cytokines are epidermal growth factor, transforminggrowth factor-", interleukin-1, and colony stimulatingfactor-1.[56]Lack of appropriateinflammatory response, an inadequate expression of some cytokines,and increased bone density that impedes resorptionhave been noted to be factors for DTE in some syndromese.g.,osteopetrosis,[57] sclerosteosis,[58]Carpenter syndrome,[59]Apert syndrome,[60]cleidocranialdysplasia,[61]pyknodysostosis,[62]and others, underlyingdefects in bone resorption and other operating mechanisms might be responsible for DTE.

As the patient in the present case shows no significant the medical history and related physiological findings, other than delayed tooth eruption.Based onseveral studies, the only possible explanation is geneticand hereditary predisposing factors as the aetiology ofdental anomalies. Hence, delayed onset of gene expressivity may lead to delay in tooth eruption, such situation may be called 'inherited retarded eruption'.[38]

Although the persistence primary tooth may provide the sufficient function up to some limit, but long-time retention of primary teeth may lead to periodontitis, profound caries, and ankylosis. Infra-occlusion is frequently caused by ankylosis of the retained primary teeth and by tipping of the adjacent permanent teeth. The timely eruption of tooth is responsible for the proper craniofacial development, as in the present case due to long time retention of the deciduous teeth the subjects diagnosed with apparently reduced lower facial height and also crossbite. Hence such situation can be challenging for orthodontic treatment. Aestheticscan be the major concern in such cases if it comes to anterior teeth. The appropriate treatment should be planned to meet the patient's requirement.[23]Information in the literature issparse on this topic, either because the condition is underreported or because defective tooth developmentis primarily diagnosed in the permanent dentition. No systematic approach to accelerate the eruption of malformed retained teeth could be foundin the literature. However, Andreasen[63] suggests that the patients in whom the defect is not in the supporting apparatus of the tooth, exposure of the affected teeth might bring about the eruption. If the tooth is lagging in its eruption status, active treatmentis recommended when more than 2/3 of the root has developed. Exposure accompanied by orthodontic tractionhas been shown to be successful. The decision to use orthodontic traction inmost case reports seems to be a judgment call for theclinician. No conclusive guidelines could be derivedfrom the literature regarding when active force shouldbe used to aid eruption of the exposed tooth.As the root development of permanent tooth was normal, patient was kept on periodic observation. The required restorative treatment was done with respect to upper left first and second molars and the patient was advisedfor the extraction of the upper first molar which her parents didn't agree to.Hence the patient and parents were guided to maintain the proper oral hygiene, relative instruction was given and was kept on monthly oral check-up.

IV. Conclusion

Variation in the normal eruption of teeth is acommon finding, but significant deviations from established norms should alert the clinician to further investigate the patient's health and development. Delayedtooth eruption might be a harbinger of a systemiccondition or an indication of altered physiology of thecraniofacial complex. Whereas, some cases are idiopathic or due to genetic polymorphism. So, theclinical implications of genetically controlled patternsof dental abnormalities are very important in the earlydiagnosis and appropriate orthodontic intervention.

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Laboratory Investigation	Results
T3	3.9pg/ml
T4	1.05 ng/dl
TSH	1.2uIU/mL
Serum Calcium	10.3mg/dl
Serum Phosphorous Inorganic	5.1mg/dl
PTH	36.6pg/ml
Vitamin D total 25 Hydroxy	29.45 ng/ml
Alkaline phosphate	181 U/L

Table1: Laboratory Investigation

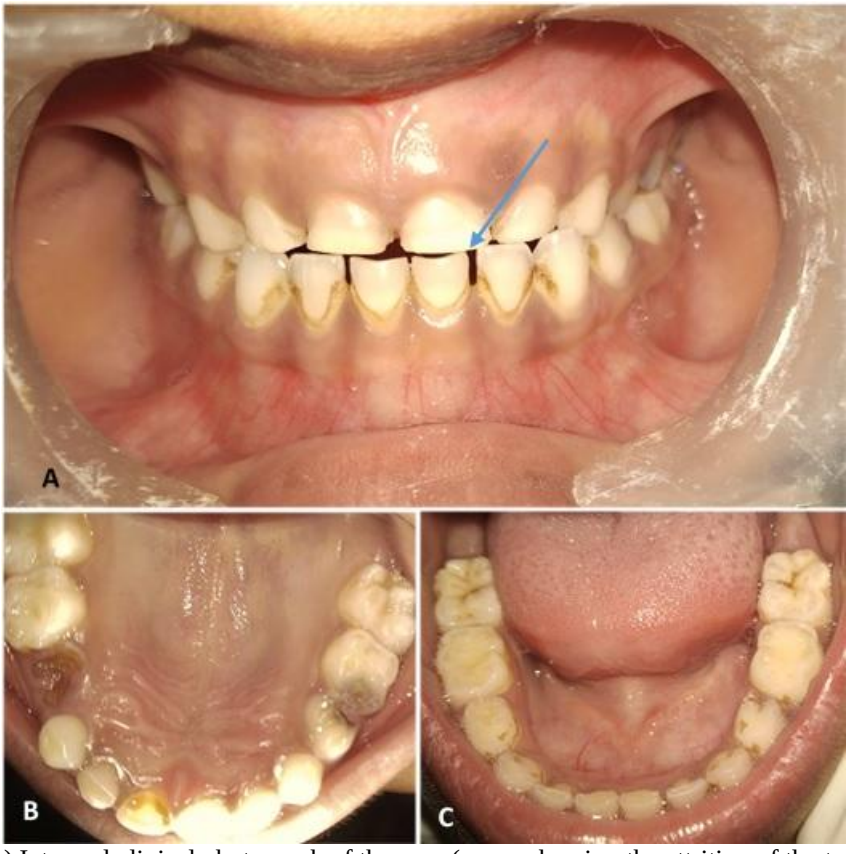

FIGURE 1: A) Intraoral clinical photograph of the case (arrow showing the attrition of the teeth due to long time retention in mouth). B) Intraoral clinical photograph of maxillary occlusal view. C) Intraoral clinical photograph of mandible occlusal view.

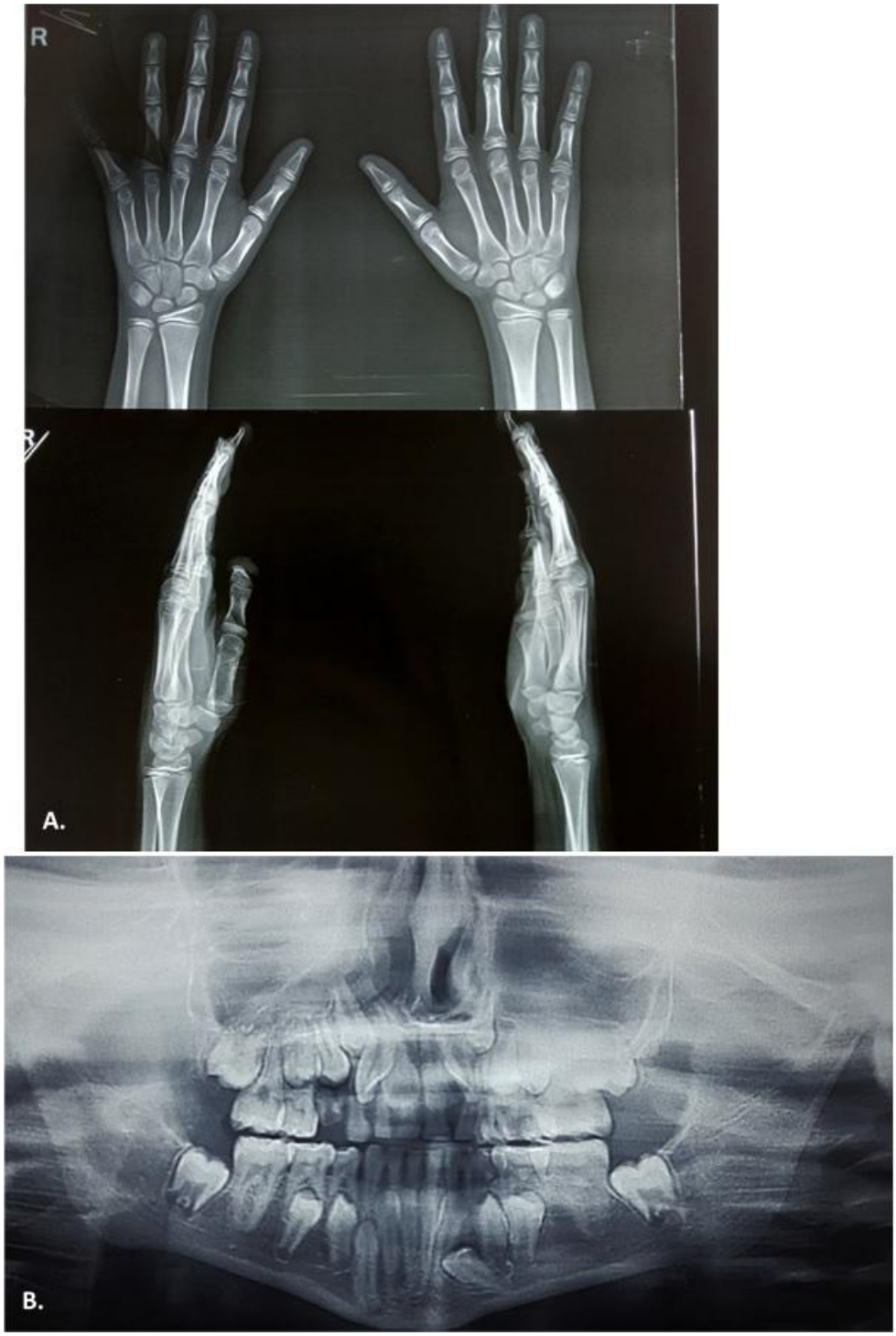

FIGURE 2: A) Hand Wrist Radiograph to depict bone age of the child. B) Radiographic evaluation showed unerupted successor teeth with normal root forming and retained deciduous teeth with their almost intact roots.

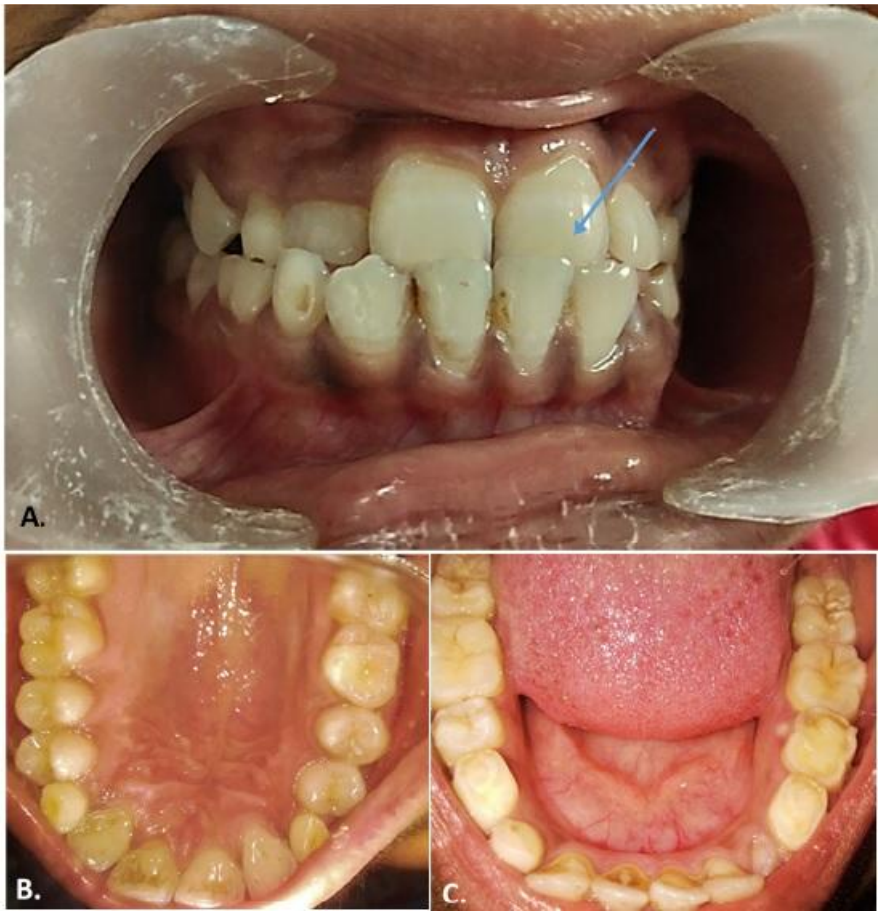

FIGURE 3: A) Intraoral clinical photograph of patient's elder sister (18 yr. old) (arrow showing development of anterior cross bite due to long time retention of deciduous teeth). B) Intraoral clinical photograph of maxillary occlusal view. C). Intraoral clinical photograph of mandibular occlusal view.

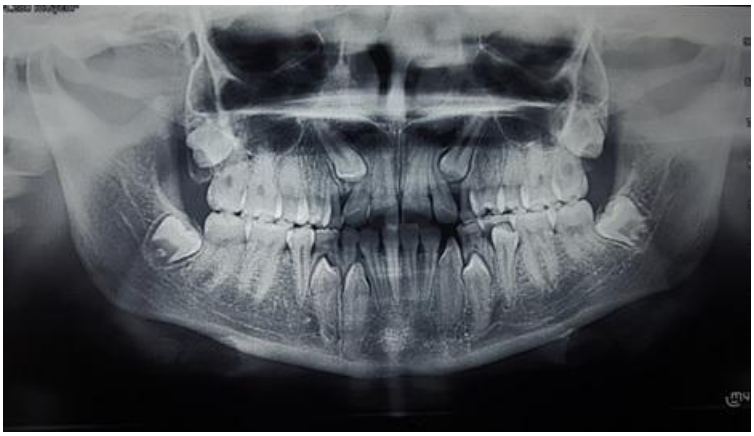

FIGURE 4: Radiographic evaluation of 18 yr. old girl showed retention of 53,63 in maxilla and 74,75,83,84 in mandible respectively