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Case Report

DENTIN DYSPLASIA WITH TAURODONTISM: A CASEREPORT OF MULTIPLE TEETH INVOLVEMENT

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ABSTRCAT:

Dentin dysplasia is a rare disturbance of dentin formation either in crown and root with normal enamel formation. It is divided in two types: type I (radicular) and type II (coronal). Type I by far is more common and both types include single or multiple teeth in primary and permanent teeth dentition. Combinations of both types also have been described in literature. Four distinct forms of Type I and one form of Type II were identified. Taurodontism is a rare dental anomaly affecting primarily the molars and occasionally premolars and are usually found in association with other anomalies. In this article we are reporting a rare case of a 16 year old female patient who presented with dentin dysplasia (rootless teeth) with taurodontism involving multiple teeth, with crown of normal enamel and normal dimentions, associated with excessively mobile teeth which appeared radiographically as rootless teeth, atop the alveolar bone.

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خلل التنسج العاجي و أسنان الثور: تقرير لحالة متعددة الاسنان المتضررة.

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خلل التنسج العاجي هو اضطراب نادر في تشكي لعاج الاسنان إما في التاج والجذر مع تشكيل المينا اعتياديا. وهي مقسمة في نوعين: النوع الأول(الجذر) والنوع الثاني (التاجي). النوع الاول الأكثر شيوعا وتشمل كلا النوعين واحد أو عدة أسنان لبنية أو ودائمة. أيضاوقد وصفت عدة حالات تظهر كلا النوعين في الدوريات. تم تحديد أربعة أشكال متميزة من النوع الأول وشكل واحد من النوع الثاني. أسنان الثورهوشذوذ الأسنان النادرة التي تؤثرفي المقام الأول على الأضراس والضواحك أحيانا وعادة ما تكون موجودة في بالاشتراك مع غيرها من الحالات الشاذة. هذا المقال يبلغ عن حالة نادرة لمريضة،16سنة تحوي خلل التنسج العاجي (أسنان بلا جذور) مع أسنان الثورلأسنان متعددة أخرى،مع تاج من المينا العادية وبأبعادها العادية، ويرتبط مع الأسنان حركة بشكل مفرطوالتي ظهرت الأشعة السينية وبلا جذور الأسنان فوق العظم السنخي.

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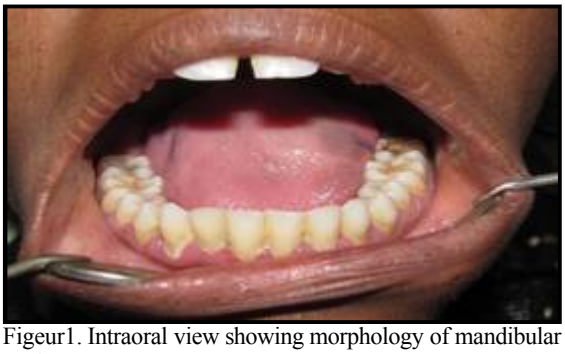
INTRODUCTION:

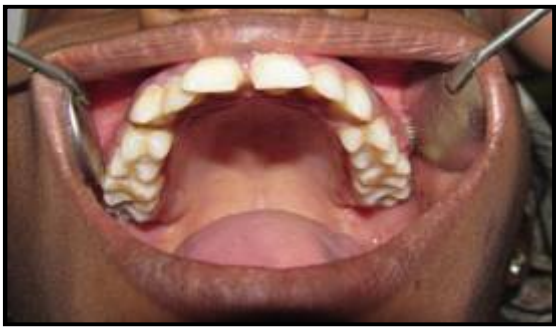
Dentinal dysplasia, so named by Rushton, is an autosomal dominant hereditary condition affecting dentin formation that is rarely encountered in dental practice. Its prevalence has been recorded as 1 in 100, 000[1]. Dentin dysplasia is characterized by normal enamel but atypical dentin formation with abnormal pulpal morphology. In 1920 Balchsmiede first reported 8 cases and described as "root less teeth"[2]. Rushton in 1939 described it as "Dentinal Dysplasia" (DD), since disturbance of dentine development was recognized such as highly atypical dentine, sometimes with pulpal obliteration and defective root formations [3]. Carrol et al proposed a sub classification based on the radiographic findings. They proposed 2 basic types. Type I was classified into 4 sub-types; 1a, 1b, 1c, and 1d. In type 1a, showed complete obliteration of the pulp chamber and usually there is little or no root development. Type 1b variation has a horizontal, crescent shaped, radiolucent line, which separates normal coronal dentine from abnormal radicular dentin. The roots are short, conical and rudimentary. Type1c variation shows 2 crescent–shaped horizontal radiolucent lines with their concavities toward each other at the cement- enamel junction and the roots one half the normal length. Type 1d is characterized by normal root formation, which sometimes may be bulbous in the coronal 3rd. Within the pulpal canal "a stone" may be found. In this type of DD, the pulp chamber is usually not obliterated and normal root formation occurs[4]. In type II the pulp chamber and root canal are shaped like thistle tube with an accumulation of pulp stones. There is no periapical radiolucency[5].Clinical findings in DD1 have shown affected teeth in both dentitions to be normal in shape, size, color, and enamel formation. The literature describes increased mobility of teeth, usually followed by early exfoliation [6]. Affected teeth may be prematurely exfoliated owing to their short roots and periapical lesions or lost as a result of minor trauma[7]. Some authors have suggested that affected teeth are more resistant to dental caries. Teeth eruption is usually normal, but a delayed dental eruption pattern has also been reported[8]. Histologically, normal dentinal tubule formation appears to be blocked and new dentin forms around the obstacles, which takes on lava flowing around boulders[5]. The dentin defect is confined mainly to the root, with the underlying enamel, a layer of mantle dentin and most of the remainder of the coronal dentin typically reported as normal. Dysplastic areas have been shown to exhibit tubules which are blocked and shunted from their normal course by numerous denticles[9, 10]. Taurodontism was first reported in the remnants of prehistoric hominids by Gorjanovic-Kramberger and Aldoff in 1907[11]. In the modern man this anomaly was first reported by Pickerill in 1909[12]. Sir Arthur Keith[13] coined the term "taurodontism" meaning bull-teeth in 1913 to describe molar teeth in which the body tends to enlarge at the expense of the roots. Modern man's teeth have pulp chambers that are relatively small, set low in the crown, and have a constriction of pulp chambers at approximately the cementoenamel junction, and are called cynodont. Taurodontism has diverse possible causes, [14] including failure of invagination of epithelial root sheath sufficiently early to form the cynodont or as a variant of the pulp chamber form which may or may not arise as a result of abnormal dentine development. Taurodontism can appear either as an isolated trait or in combination with other anomalies such as hypodontia, amelogenesis imperfecta, Down's syndrome, Klinefelter's Syndrome, or tricho-dento-osseous (TDO) syndrome. [15]

CASE REPORT:

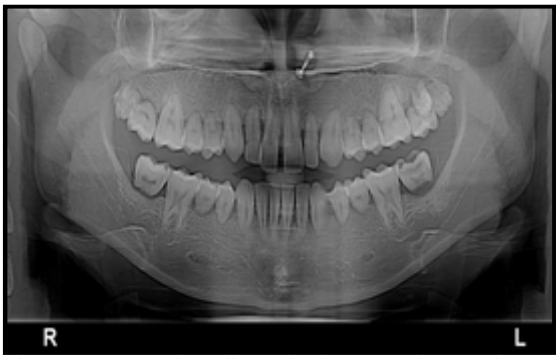
A16-year-old girl reported in our institution with the complaint of mobility in maxillary and mandibular posterior teeth and wanted the teeth to be fixed. According to her previous dental records her primary teeth were of normal appearance and exfoliation times were normal. Oral examination revealed a permanent dentition of normal size and appearance with some plaque and calculus deposition, Figeur1, 2. Gingiva was normal but there was grade 3 mobility of all posterior teeth in both arches except the first molars. Medical and family history was not contributory. Panoramic radiographi canalys showed that manibular second molars and maxillary second and third molars had no roots at all. It appeared as though the crowns of the teeth were placed directly on the bone without any deeper anchorage and the premolars having short rudimantary roots, Figeur3, 4. The roots of incisors and canines are also defective they are short with round apex. Mandibular first molars showed short, conical, misshapen roots with enlarged pulp chambers, which are classified also as taurodontism (mesodont) by crown root ratio, Fig. 3, 4. A buccal paramolar present in maxillary arch on left side.



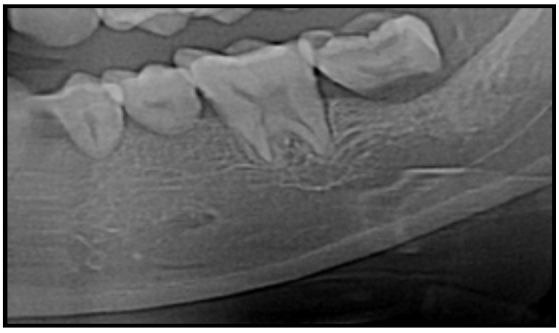
Figeur1. Intraoral view showing morphology of mandibular teeth.



Figeur2. Intraoral view showing morphology of maxillary teeth.



Figeur3. Panoramic radiograph shows rootless teeth number UR-7,8, UL-7,8, LL-5,7, LR-5 and 7, Mandibular first molars showed short, conical, misshapen roots with enlarged pulp chambers, which are classified as taurodontism (mesodont) by crown root ratio, Short rudimentary roots with blunt apex teeth number UR-3,4,5, UL-3,4,5,LL-3,4,LR-3,4.



Figeur4. A close-up of the panoramic radiograph concentrating on the lower left quadrant depicts rootless teeth number LL-5 and 7, and tooth number 6 having short, conical, and misshapen roots with enlarged pulp chamber classified as taurodontism (mesodont) by crown root ratio

DISCUSSION:

This case exhibited defined features of DD1 with taurodontism. Little is known about etiology of DD and taurodontism in healthy individuals. Several etiological factors have been implicated as possible causes, but the precise nature of the defect has not yet been determined. Scanning electron microscope (SEM) study by Sauk et al[16] postulated that the DD1 is the defect in the epithelial component of the developing tooth germ in which the invagination of the root seath occurs too soon resulted in stunted root form. Melnick et al suggested that the abnormal root morphology is due to abnormal differentiation of odontoblastic cells[10]. According to Witkop taurodontism is more likely an anomaly arising from a failure of sufficient invagination of epithelial root sheath and not an intrinsic defect in dentin formation[14]. The hereditary tendency of taurodontism is well established. According to Shaw (1928)[17] the trait is inherited as an autosomal recessive disorder, and Dominant inheritance was suggested by Goldstein and Gottlieb (1973) and Gramer and Zusman (1967) [18, 19]. In this case report, the patient was in permanent dentition with normal size and shape. As illustrated in the panoramic radiograph, Figs 3. Teeth UR-6, UL-6, LL-6 and LR-6 were diagnosed as taurodontism (mesodont). It might be postulated that abnormal dentin formation in DDI caused different teeth morphology, teeth UR- 3,4,5, UL-3,4,5, LL-3,and LR-3 having short roots, and teeth UR-7,8,UL-7,8,LL-5,7 and LR-5,7 being rootless. Identification of familial history in this case was unsuccessful because little information about the patient's father and the mother teeth. The patient reported that her mother had become edentulous at a very early age, but the etiology of tooth loss was unknown. Although the patient's information did not reach to any conclusive diagnosis. In conclusion, it can be seen that dentin dysplasia and taurodontism has until now received insufficient attention from dentists. There are still some clinical and theoretical issues that remain inconclusive. More detailed documentation of clinical presentation in cases of DD1with taurodontism is needed in order to establish relevant clinical signs that will aid in the diagnosis of this disorder.

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