Review Article
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Dentin Dysplasia: A Review

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Objectives Dentin dysplasia (DD) is a rare disorder, which is not accentuated in dental practice. DD has 2 types. Type I is manifested by tooth mobility, short roots, reduced pulp space and normal crowns. Type II or coronal DD is characterized by normal crowns in permanent teeth but discolored crowns in primary teeth. Denticles are detectable in the pulp chamber of teeth in type II DD. Many uncertain aspects of this condition include its etiology, diagnosis, and treatment planning. For a long time, extraction of mobile teeth was the main possible choice, which would lead to psychosocial problems that needed psychological interventions. The present study aimed to collect and classify the recent information on DD.

Methods An electronic search of the literature was carried out in PubMed and Google Scholar from 1977 to 2018. Duplicates were eliminated and the retrieved articles and relevant textbooks were thoroughly reviewed.

Results Although DD has an unknown etiology, it is known for a fact that it has a hereditary trait. The present article provides some information about DD, including possible etiological factors, clinical, radiographic and histological manifestations, diagnosis and current treatment options.

Conclusion Management of DD is based on preservation of teeth in the oral cavity and may vary from preventive and piecemeal care to tooth extraction and regenerative pulp therapy.

Keywords Dentin Dysplasia; Type 1; Dental Pulp Calcification; Review Literature as Topic

Introduction

In 1973, dentin defects were classified by Shields et al. into two major types: dentinogenesis imperfect (DGI) and dentin dysplasia (DD).[1] DD is a rare hereditary autosomal dominant dentin disorder first announced by Ballschmiede and Berlin in 1930, which can involve both the primary and permanent dentitions.[2] The risk of affliction is not related to gender. There is no document on the association of DD with race. Later in 1939, Rushton termed this atypical dentin as DD.[3] By increasing the number of case reports, Witkop described two phenotypes for DD in 1975, appearing with normal crowns in permanent dentition.[4] Classifications established based on clinical and radiographic features are as follows: Type I DD, also called radicular or root DD affects almost 0.01% of the world's population.[5] It is characterized by missing or rudimentary roots with conical shape and with or without periapical lesions, which leads to tooth mobility. According to de La Dure-Molla et al. the prevalence of periapical pathologies is directly related to the shortness of the roots.[6] Crowns are normal in shape and color. However, there are some reports confirming brown/blue, yellow/grey, or brown opacities in the incisal third.[6] According to Carroll et al. complete or partial pulp obliteration is related to the shortness of the roots as well.[7] They claimed that there are four possible subtypes in DD type I: DDIa, DDIb, DDIc, DDId.[6] DDId is the less severe subtype with no pulp obliteration and with normal root formation. Type II DD is due to the dentin sialophosphoprotein (DSPP) gene mutation.[8] It is known as coronal DD and is less common than type 1.[9] Type II DD is more frequent in primary dentition, characterized by brown or blue amber discoloration and total pulp obliteration. Permanent teeth represent normal crowns or little amber/translucent discoloration with intra-pulpal calcification and normal roots. Type III DD was suggested by Eastman et al. for an isolated affected tooth.[10] The aim of this study was to present various treatment plans for this disorder since DD is not commonly considered in dental practice due to its rarity and is a challenging disorder to manage. There are still numerous dilemmas to be considered and a small percentage of dentists deal with such cases during their career.[9]

Materials and Methods

An electronic search was conducted in Google Scholar and PubMed search engines using the keywords dentin dysplasia, dentin dysplasia type I, dentin dysplasia type II, pulp stone, dentils, pulp calcifications, radicular dentin dysplasia, coronal dentin dysplasia and rootless teeth. We selected 40 articles from the search results and summed up the collected information.

Discussion

DD, similar to other hereditary defects, is a rare condition requiring meticulous and careful management. DD reportedly has a hereditary dominant trait, and with parents suffering from this condition, there would be 50% possibility of inheritance to the children; however, there are some cases as first generation sufferers.[11-15]

Etiology:

Despite the information mentioned above, etiology of DD is

still not known. However, there are some hypotheses discussed in the related literature. In 1962, Logan et al.[16] suggested that multiple degenerative foci within the papillae become calcified, leading to growth retardation and pulp obliteration. In 1972, Sauk et al.[17] disclaimed that dental papillae are responsible for DD and stated that alteration in the epithelial component leads to development of calcified foci. They suggested that premature invagination of Hertwig's root sheath leaves epithelial foci, which then induces ectopic dentin formation. Nevertheless, their opinion is not accepted unanimously.[6] In 1976, Wesley et al.[18] introduced the new idea of abnormal interaction between ameloblasts and odontoblasts, which leads to abnormal differentiation and function of odontoblasts.

In 2011, Bloch-Zupan et al.[19] presented two cases of DD type I identifying mutation in SMOC2 gene. SMOC2 belongs to the family of matricellular proteins that regulate interactions between cells and the extracellular matrix. To analyze the function of SMOC2 in tooth development, they planned an experiment on zebra fish, since it has a similar tooth development to mammals. The results indicated that the size and presence of the teeth were probably dependent on the level of SMOC2 depletion. In 2016, a study demonstrated that VPS4B is a disease-causing gene for DD type I, which regulates tooth development via an interaction with Wnt/β-catenin canonical signaling.[20]

In a recent study published in 2017, authors mentioned a correlation between fibroblast growth factor receptor and tooth development. Mutation in fibroblast growth factor receptor has been demonstrated in Pfeiffer syndrome, which is manifested by dental abnormalities including DD.[21] In the reported cases of type II dentin defects, there were descriptions of DSPP gene mutation that convinced the authors regarding the genesis of the defect.[8, 12]

Dentin defects show variable radiographic and clinical manifestations that should be considered in diagnosis to devise an appropriate treatment plan. Since the etiology and clinical aspects of type II DD are indistinguishable from DGI type II, the diagnosis has become intricate for practitioners. Therefore, de La Dure-Molla et al.[6] provided a revision of the classification based on the recommendations of Hart and Hart,[8] which combines all DSPP-related disorders as DGI. It is also classified into mild, moderate and severe forms, considering the mild form as Shield type II DD. In this study, Shield type I DD is considered as radicular DD.

*In one study, the periapical lesion was 3 cm and involved the sinus.[22] Besides the features mentioned in Table 1, there are other clinical or radiographic features that are not specific but have been reported in DD cases including the following:

Malocclusion,[9, 13, 21, 28-30] hypodontia,[27] microdontia,[21] oligodontia,[27] anterior open bite,[13, 21, 27] cross bite,[21] abnormal positioning of the teeth,[22, 25] and unerupted or impacted teeth; roots may or may not form.[13, 15] Extraoral examination generally reveals no abnormality.[13, 22, 27] Some cases report atrophy and decreased density of the periapical bone[11, 22] with well-defined margins.[13, 15] The potential reasons include abnormal or poor bone development and inflammatory reactions related to periapical lesions.

Table 1- Clinical and radiographic features of DD

Feature	Defect	Dentin dysplasia type I	Dentin dysplasia type II
Clinical crown shape	Primary teeth	Normal[9]	Normal[9, 22]
	Permanent teeth	Normal[9]	Normal[23]
Crown color	Primary	Generally normal[9] but brown/blue, yellow/gray or brown opacities in the incisal third have been observed.[6]	Amber[24], blue[22] or brown[25]
	Permanent		Generally normal or amber/ translucent discoloration[9]
Roots		Rootless[12] or short-rooted, pointed-shape roots in both dentition[21] Furcal region may not be present[12] or noticeable in the periapical region[22]	Normal in shape and length[22] Normal root/crown ratio[9]
Taurodontism		Detectable[9]	Detectable[23]
Pulp chamber and root canal pulp shape		Crescent shape in molars[22] Shape also depends on the shortness of the root[22]	Pulp shape depends on the amount of obliteration.[22] In the incisors, canines and premolars, the pulp canal is more bulged in the apical region. Molars have a flame or thistle-tube shape.[22]
Pulp obliteration	Primary	Complete obliteration[22, 25]	Complete obliteration[14]

Permanent	Complete or partial obliteration[22] parallel to the cementoenamel junction[9, 24]	Partial obliteration with multiple pulp stones in a large pulp chamber[9] with appearance of so-called shell teeth[22]
Time of pulp obliteration	Occurs before tooth eruption[22].	No obliteration observed before tooth eruption.[12]
Periapical pathology*	Multiple radiolucencies or cysts detected, not related to carious lesions.[22]	Reduced bone density[26]
Attrition	Attrition is reported in some cases.[27]	Permanent teeth are more susceptible to attrition.[22]
Resistance to caries	High[12]	High[22]
Mobility	Present[24] because of periapical pathologies and shortness of the roots leading to premature tooth loss.[22]	Not present[9]

Histological features:

Histological assessments of the extracted teeth affected by type I DD reveal normal gross morphology. Enamel and peripheral dentin are generally normal but Shankly et al.[25] noticed hypo-plastic enamel in their case. Deeper dentin layers display asymmetrical structure that lacks tubular pattern. In fact, numerous denticles block or deflect dentinal tubules in dysplastic areas of the underdeveloped dentin layers. The affected dentin shows waterfall appearance because of the death cycle and production of odontoblasts producing further dentin.[9, 25]
Clinical and histological evaluations interpret periapical pathologies as granulomas or odontogenic epithelial root cysts that are commonly radicular cysts.[22, 26] The cystic cavity is lined by non-keratinized epithelium covering the connective tissue containing inflammatory cells.[22]
In type II DD, diagnosis can be confirmed by detecting irregularly arranged dentinal tubules and entrapped osteodentins surrounded by a lacuna. In addition, pulp stones are detectable as abnormal calcified zones.[23]

Diagnosis:

Since the signs and characteristics of DD are specific, when some clinical abnormalities are suspected, diagnosis is conducted based on dental history, and clinical and radiographic examinations. Genetic tests, which are confirmed by the Genetic and Rare Diseases Information Center, are helpful in some cases. Cone-beam computed tomography can be requested to localize denticles or ascertain root canal length. Histological assessments are not needed due to the characteristic clinical and radiographic features.
Early tooth loss is predictable not only in DD but also in some syndromes like Papillon-Lefevere,[31] Chediak-Higashi,[32, 33] Kostman disease,[33] hypophosphatasia[34] and vitamin-D-resistant rickets.[33] Hence, we need to become familiar with these disorders and distinguish them from DD. Most of these disorders are genetically inherited; therefore, performing extended diagnosis in family members will be beneficial for detecting these syndromes before they get worse and hard to treat. Genetic and Rare Diseases Information Center recommends genetic counseling as well. However, the general expectation is that primary dentition will exfoliate and patients will not look for treatment unless permanent teeth are affected as well.
DD and associated radiographic or histological features appear in association with a number of syndromes such as calcinosis,[35] Ehlers-Danlos syndrome,[35] brachio-skeleto-genital syndrome,[35] Goldblatt syndrome, and Schimke immune-osseous dysplasia,[12] which may bear a resemblance to DD. DD type I can be a part of Pfeiffer syndrome[21] and Seckel syndrome as well.[36] Genetic consultation can help in diagnosis of these conditions.

Differential diagnosis:

DD should be differentiated from other dentin disorders. Dentinogenesis imperfecta (DI) is a different pathology affecting both deciduous and permanent teeth and causing bulbous crowns and narrow roots with blue gray crowns. [(8)] Type I DI is a syndromic pathology that occurs with osteogenesis imperfecta.[37] DI type II shows similar manifestations and the same etiology to DD type II affecting both dentitions, while DD type II almost affects the primary teeth. DD type II and DGI can only be differentiated by their clinical aspects in permanent teeth. In DD, permanent teeth are generally normal in color and shape.[(6)] DI type III has shell teeth appearance on the radiographs because of the significant pulpal enlargement and the same clinical appearance as type II DI. Amelogenesis imperfecta[22] that is caused by abnormal formation of the enamel and odontodysplasia[13] should be differentiated from DD as well.
Root shortening is a characteristic feature of DD type I. That is why other conditions with root dwarfism such as orthodontic treatment,[38] abnormal hemoglobin production in thalassemia,[39] Steven-Johnson syndrome,[40] infection with Herpes Zoster virus[41] and also hypoparathyroidism[42] should be considered in differential diagnosis. Each of these conditions has specific manifestations that can be differentiated with precise examination. Thus, we suggest the practitioners to be aware of these conditions and distinguish them from one another.

Chief complaint:
Early diagnosis of DD is crucial to maintain the existing

teeth as long as possible. Being familiar with the manifestations of DD can enhance and accelerate the diagnosis. Patients may refer to dentists complaining of tooth mobility due to short roots, pain, tooth fracture, or recurrent abscess, or may require rehabilitation of the missing teeth.

Oral hygiene is often below the optimal level because DD comes with disabling syndromes preventing oral hygiene[21] or because sufferers are afraid of falling off of their teeth by brushing.[9] Poor oral hygiene can confuse dentists, mistaking DD with rampant caries. Thus, rampant caries can be considered as a differential diagnosis.

Poor oral hygiene leads to gingivitis, attachment loss, periodontitis, and several carious lesions.[15] Caries can cause pain, urging patients to see a dentist. In view of the fact that in DD the teeth may not respond to vitality tests, root canal treatment may be performed and since root canal treatment is difficult in such cases, it may result in accidental diagnosis of DD.

Psychosocial considerations:

Discoloration and premature tooth loss can cause psychosocial problems for patients and anxiety for the families in case of risky treatment plans such as major bone grafting for implantation. Numerous extractions can distance young sufferers from the society and make them feel less confident about themselves. Also, the patients may experience some emotions after tooth extraction, like feeling old and sad. Facial changes following tooth loss can result in social seclusion of the patient. Thus, consultation with psychologists before execution of such treatment plans is recommended. Some patients tend to keep tooth loss and prosthetic treatments private to avoid being considered as facing premature aging. Also, they may not let their friends and partners see them without their dentures. Thus, patients should be prepared for the effect of tooth loss; an explanation from the dentist or talking to someone with the same experience would be helpful. It is wise to present information about tooth loss in an appointment prior to the extraction date since patients may not pay attention to this because of their high stress level.[43]

Treatment plans:

Before any therapeutic intervention, preventive care and improving oral hygiene approaches should be applied since they cost less and can improve prognosis, as it is the case in normal individuals.

In DD treatment, we have to preserve the teeth in the oral cavity as long as possible to avoid bone loss. Preventive care includes nutritional instructions and advice for reducing the frequency and amount of sugar intake. Providing instructions for improving oral hygiene such as frequency and methods of toothbrushing can be advantageous. There is useful advice about taking fluoride supplements that can be taken into account.[12, 13, 15, 21, 22, 25, 44] The patients should be followed up by their dentists in order to prevent caries and periodontal complications to decrease the need for advanced interventions and preserve the teeth for a longer period of time.[12] Most dentists believe that maintenance and improvement of oral hygiene is the most effective approach for DD.[12]

Non-carious periapical radiolucencies are one of the diagnostic characteristics of DD. Teeth with necrotic pulp will not respond to vitality tests and have discolored crowns. Also, pulpal necrosis can be a consequence of caries that can lead to apical periodontitis. If apical periodontitis is not treated, it converts to apical granuloma. Periapical radiolucency caused by cysts is treated by enucleation.[15, 22] Root canal therapy for apical radiolucencies is considered in teeth without complete pulp obliteration.[14, 22, 27, 45, 46] Delay in treating necrotic teeth or PA radiolucency may lead to bone loss but there is a possibility of root canal perforation because of obliteration.[15] Extraction of these teeth is another aggressive approach. Short or blunt roots or periapical radiolucency can induce tooth mobility. Mobile teeth can be stabilized with splints.[9, 21] Carious mobile teeth with poor prognosis are better to be extracted.[12, 15, 21, 25, 44] Restorative procedures should be applied in the most conservative way to avoid pulp exposure because of thin dentin layer. Various restorative materials have been applied.[13, 15, 21, 22, 25] Stainless steel crowns can be used to prevent posterior tooth wear.[12, 27] Crown lengthening can be performed for teeth with attrition.[22]

In DD, teeth are often displaced or trans-positioned. Orthodontic care is suggested with minor forces to prevent bone and root resorption and tooth mobility.[13, 22]

Premature loss of teeth is the main problem in DD. Rehabilitation of the oral cavity with different denture types and implants can return function and esthetics. Denture has a lower cost and risk in comparison with implants; additionally, the patients should have adequate bone for implant placement. Ridge augmentation may be required for dental implant placement, which is physically and financially costly and patients may not accept it.[12, 15] However, there is a general belief that implant placement is the ultimate treatment for DD. Sinus lifting and bone grafting are successfully performed to restore the lost alveolar ridge for implant treatment or implant-supported dentures.[15] Malocclusion can be modified with dentures while improving patient's chewing function.[21] For young patients, dentures will soon be useless and tooth eruption can interfere with placement of the denture and cause pain. Hassona et al.[21] suggested using flexible denture bases for young patients.

Periodontitis has to be managed by pocket debridement or scaling and root planing to improve oral hygiene before attachment loss and gingivitis. Chlorhexidine rinse is also recommended.[21]

DSPP is the most abundant dentin extracellular matrix non-collagenous protein. Using bioactive extracellular matrix molecules such as DSPP can induce pulp mineralization. As

mentioned in the literature, mutation in dentin sialoprotein (DSP) section of DSPP gene is known as the common cause of DD type II and DI. Since DSP stimulates the differentiation of mesenchymal stem cells of the pulp into odontoblasts, researchers suggest implantation of recombinant DSP into carious areas to induce regeneration of periodontal ligament and pulp tissue, and dentin formation. The differentiated cells produce tertiary dentin and induce demineralization of periodontal ligament. Having no side effects and migration ability of DSP-induced odontoblasts into microleakage zone to seal the gap between the cavity wall and restoration are the advantages of this suggested approach.[47]

Regenerative therapy by using dental stem cells is being widely investigated. Although there is no certain outcome, it is an emerging field that will lead to significant useful data that can change the whole dental profession.[5] Since there is no evidence and case reports of these regenerative treatments on DD patients, the prognosis cannot be detected.

Conclusion

DD is an unusual disorder causing tooth mobility, periapical radiolucencies, and premature tooth loss, in which crowns have normal appearance. Dentists should consider DD in their differential diagnosis when encountering such symptoms. Although treatment plan is specific for each patient, early diagnosis and implementing practices to improve oral hygiene would help preserve the teeth for a longer period of time. Proper esthetic and functional rehabilitation of the lost teeth can help reduce psychosocial misery.

Conflict of Interest

Non Declared ■

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