Rootless teeth: Dentin dysplasia type I

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Abstract
A rare case of hereditary disturbance of dentine, Dentin dysplasia type I is presented, which is characterized by short or total absence of roots, obliterated pulp chambers, and peri-apical radiolucencies. It affects both primary and secondary dentition. Management of patients with dentinal dysplasia is difficult and requires a multidisciplinary approach. An overview of dentin dysplasia and its management along with a case report is discussed.

Keywords: Dentin dysplasia, opalescent dentin, rootless teeth

Introduction
Dentin Dysplasia (DD), a rare anomaly is an autosomal dominant hereditary disturbance in dentin formation affecting either the primary or both the dentitions in approximately one patient in every 100,000. It was Ballschmiede in 1920 who first reported such a condition as 'rootless teeth' and in 1939 Rushton termed this condition as DD. On the basis of radiological findings, Witkop in 1972 classified DD into type I: Radicular DD and type II: Coronal dentin dysplasia.

Type I DD is characterized by crowns appearing normal or might be slightly amber colored with no or only rudimentary root development, aberrant growth of dentin in the pulp chamber leading to reduced pulp space in permanent teeth and incomplete or total obliteration of the pulp chambers, and periapical radiolucent areas or cysts which might result in premature loss of tooth. Teeth show greater resistance to caries than normal teeth do.

DD type II is characterized by yellow, brown, grey amber, translucent primary teeth with complete pulpal obliteration.

The permanent teeth have a normal appearance or might be slightly amber colored. Roots are normal in size and shape with a 'thistle tube' shaped pulp chamber with pulp stones. Obliteration of pulp chamber does not occur before eruption.

The purpose of this article is to present a case with a rare disorder, dentinal dysplasia type I, with an overview of its diagnosis and management.

Case Report
A 14-year-old girl reported with a complaint of mobile and irregularly placed teeth.

Clinical findings
General examination showed convex profile, competent lips, normally developed maxilla but slightly sagitally underdeveloped mandible. Intraoral examination showed normal appearance of crowns and gingiva with no signs of gingivitis or periodontitis. Clinical examination showed grade II mobility with maxillary and mandibular incisors and molars and had missing maxillary and mandibular right laterals. She presented with crowding in maxillary and mandibular anterior region.

Radiographic findings
Panoramic radiograph showed normal appearance of crown, rudimentary root formation with all teeth except maxillary right canine and mandibular right first premolar. Obliterated pulp chambers were found with most of the teeth.

Patient’s medical history revealed no disturbance in general health. Clinical and radiograph examination of the patient’s parents and siblings revealed no such disorder or any previous familial history of such condition, so she is considered as first generation sufferer of DD.

History, clinical and radiographic findings revealed this case as ‘DD type I’.

Considering the age of the patient, treatment was aimed in maintaining the health of existing teeth. Conservative
treatment was advised with maintaining excellent oral hygiene and dietary measures with fluoride supplements. A multidisciplinary approach with extraction of the teeth with poor prognosis i.e.; maxillary and mandibular anteriors and replacement with implant supported prosthesis was advised once her growth is complete. To receive implants, bilateral indirect sinus lifting, onlay bone grafting with autogenous grafts in maxillary and mandibular anterior region was advised, if required.

Discussion

The pathogenesis of DD is still unknown in the dental literature. Wesley et al.,[6] proposed that the condition is caused by an abnormal interaction of odontoblasts with ameloblasts leading to abnormal differentiation and/or function of these odontoblasts. Logan et al.,[7] proposed that the dentinal papilla is responsible for the abnormalities in root development. They suggested that multiple degenerative foci within the papilla become calcified, leading to reduced growth and final obliteration of the pulp space. Histopathologically, the enamel appears normal while deeper layers of dentin show atypical tubular patterns with amorphous, atubular areas, and irregular organization. On the pulpal side of the normal appearing mantle of dentin, globular or nodular masses of abnormal dentin are seen. Dysplastic areas have been shown to exhibit tubules, which are blocked and shunted from their normal course by numerous denticles.[6] DD type I should be differentiated from DD type II, dentinogenesis imperfecta, and odontodysplasia. Teeth with radiographic or histologic features of DD occur in a number of disorders such as calcinosis, osteogenesis imperfecta, Ehlers Danlos
syndrome, Goldblatt syndrome, Schimke immuno-osseous dysplasia, and Brachio-skeleto-genital syndrome.

Premature loss of teeth and loose teeth as a result of rudimentary or absence of roots has presented dentists with many problems in management of patients with DD I. The most effective treatment is to practice excellent oral hygiene to maintain the health of teeth. The more intervention necessary, the more tooth loss is likely to occur requiring further intervention. However, treatment varies according to age of patient, severity of problem, and the presenting complaint.

Tooth wear seen in primary dentition can be maintained with stainless steel crowns to prevent tooth wear and maintain occlusal dimension. Composite facings or composite strip crowns can be added for esthetic reasons on demand.

In permanent dentition, conserving existing health of teeth may demand endodontic intervention. Obliteration of the pulp chambers and root canals in teeth that develop abscesses makes endodontic therapy difficult. If conventional endodontic therapy is not possible, periapical curettage and retrograde filling is another alternative but not recommended in teeth with too short roots.[9] Teeth with short thin roots and marked cervical constrictions, however, are often unfavorable for crowns. If abscess develops and pulp therapy is not successful, removal of the affected teeth is required and full mouth rehabilitation is considered with dentures or over dentures until growth is complete. In case of few teeth having enough root development, cast partial dentures can be a treatment option.

Dental implants should be considered when growth is complete. Ridge augmentation procedures need to be carried out prior to implant placement in maxillo-mandibular alveolar atrophy due to early loss of teeth. A combination of onlay bone grafting and a sinus lift technique to accomplish implant placement can be done.[10]

Conclusion

Oral rehabilitation of patients with DD type I require elaborate treatment planning with a multidisciplinary approach. The outcome of a diagnosis of DD largely depends upon the age at which the diagnosis was made and the quality of the treatment provided. Early diagnosis helps in guiding variable measures to prolong the retention or rehabilitation of affected teeth to maintain good esthetics and function, thereby, minimizing nutritional deficits and psychosocial distress.

References

1. Kim JW, Simmer JP. Hereditary dentin defects. J Dent Res 2007;86:392-9.
2. Ballschmiede G. Dissertation, Berlin, 1920. Quoted in Herbst E, Apfelstaedt M, editors. Malformations of the Jaws and Teeth. New York: Oxford University Press; 1930.
3. Rushton MA. A case of dentinal dysplasia. Guy’s Hosp Rep 1939;89:369-73.
4. Witkop CJ Jr. Hereditary defects of dentin. Dent Clin North Am 1975;19:25-45.
5. O’Carroll MK, Duncan WK. Dentin dysplasia type I. Radiologic and genetic perspectives in a six-generation family. Oral Surg Oral Med Oral Pathol 1994;78:375-81.
6. Wesley RK, Wysocki GP, Mintz SM, Jackson J. Dentin dysplasia type I. Clinical, morphologic, and genetic studies of a case. Oral Surg Oral Med Oral Pathol 1976;41:516-24.
7. Logan J, Becks H, Silverman S, Jr, Pindborg JJ. Dentin dysplasia Oral Surg 1962;15:317.
8. Melnick M, Levin LS, Brady J. Dentin dysplasia type I: A scanning electron microscopic analysis of the primary dentition. Oral Surg Oral Med Oral Pathol 1980;50:335-40.
9. Ravanshad S, Khayat A. Endodontic therapy on a dentition exhibiting multiple periapical radiolucencies associated with dentinal dysplasia type 1. Aust Endod J 2006;32:40-2.
10. Muñoz-Guerra MF, Naval-Gías L, Escorial V, Sastre-Pérez J. Dentin dysplasia type I treated with onlay bone grafting, sinus augmentation and osseointegrated implants. Implant Dent 2006;15:248-53.