Review Article

Odontoma - A brief overview

Deepali Patekar¹, Supriya Kheur², Archana A. Gupta²

¹Department of Oral Pathology and Microbiology, Saraswati Dhanwantari Dental College and Hospital, Parbhani, Maharashtra, India, ²Department of Oral Pathology and Microbiology, Dr. D. Y. Patil Dental College and Hospital, Dr. D. Y. Patil Vidyapeeth, Pune, Maharashtra, India

Abstract

Odontoma is primarily diagnosed in children and adolescents with no gender predilection. It has been reported to be the most common of all odontogenic neoplasms and tumor-like lesions. Although the odontoma's etiology remains unknown, patients have often reported a history of trauma and infection. In most cases, odontomas are asymptomatic characterized by a relatively slow growth and rarely exceeding the size of a tooth. In rare cases, odontomas exceed their usual dimensions resulting in the expansion of the cortical bone. Although odontomas may be diagnosed during any age, they are most frequently reported before the second decade of life. The present review summarizes the major clinicopathological characteristics and molecular pathogenesis of odontoma.

Keywords
Hamartomatous malformations, odontoma compound and complex, odontogenic tumor

Correspondence:
Deepali Patekar, Department of Oral Pathology and Microbiology, Saraswati Dhanwantari Dental College and Hospital, Parbhani, Maharashtra, India. E-mail: drdypatekar@gmail.com

Received: 03 October 2018; Accepted: 05 November 2018
doi: 10.15713/ins.jodm.14

Introduction

The term “odontoma” was coined in 1867 by Paul Broca. Odontoma was defined as “tumors formed by the overgrowth of transitory or complete dental tissues.”¹ As odontomas are a result of abnormalities in the proliferation of dental tissues (both epithelial and mesenchymal components), they are not regarded as true neoplasms. In addition, as odontomas do not exhibit continuous growth nor any form of infiltration into the surrounding tissues, they are regarded as hamartomatous malformations. They are often associated with impacted/retained teeth. The extraneous bud of the dental lamina is considered to be the tissue of origin for odontoma.²,³

Etiology of odontoma⁴⁻⁸

1. Trauma during primary dentition
2. Inflammatory and infectious processes
3. Family history
4. Anomalies which are hereditary in nature including Hermann’s and Gardner’s syndrome
5. Hyperactivity of the odontoblast
6. Genetic alterations causing aberrations in signaling pathways controlling tooth development.

Clinicopathological features

Most cases of odontoma are reported within the gnathic bones (intraosseous), but few rare cases have shown to occur in oral soft tissues (gingiva). Although odontoma is reported mostly in the permanent dentition, few cases have associated with deciduous teeth. The most common location for an odontoma is in the interradicular portion of erupted teeth. Most cases occur in the anterior portion of the gnathic bones, especially in the maxilla. Although relatively less frequent, odontoma has also been reported in the posteroinferior regions.⁹ The World Health Organization⁴ 2005 classification acknowledges the presence of two odontoma types, the compound and complex odontoma. The former appears as tooth-like structures (denticles) while the latter consists of a disorganized conglomerate of dental tissues (both epithelial and mesenchymal derivatives).

Complex odontomas are relatively rare than compound odontoma with the later accounting for nearly 2/3 of all odontoma cases reported.¹⁰⁻¹² As most odontomas are asymptomatic, their diagnosis is usually a chance finding in a routine panoramic and/or intraoral radiograph. Rarely, cases present with larger lesions causing swelling of the jaw. The presence of an impacted permanent or a retained deciduous tooth is a clinical sign for suspecting odontoma.⁹,¹³ Table 1 summarizes the major clinical and radiological characteristics of the compound and complex odontoma.
Table 1: Major characteristics of the compound and complex odontomas

| Major characteristics | Compound odontoma | Complex odontoma |
|-----------------------|-------------------|------------------|
| Age                   | Most cases appear before the age of 20 years, making it a lesion of childhood/adolescence | Most cases occur before the age of 30 years with a peak in the second decade of life |
| Gender                | Male and female subjects are equally affected | Male and female subjects are equally affected |
| Sites                 | The most frequent site is the maxillary anterior region | Posterior mandibular followed by anterior maxilla is the most frequent sites |
| Frequency             | The relative frequency among odontogenic tumors varied between 9% and 37%. It is considered the most common odontogenic malformation | The relative frequency among odontogenic tumors varies between 5% and 30% |
| Clinical presentation | Painless, non-aggressive lesion with a more limited potential growth than the complex odontoma. Often associated with an unerupted permanent tooth | Painless, slow-growing, and expanding lesion. Often associated with an unerupted permanent tooth |
| Radiological features | Radio-opaque calcified structures which are small and multiple in numbers. They are anatomically similar to normal teeth. A narrow radiolucent zone usually surrounds the radio-opacity in most cases | An amorphous mass of calcified material with the radiodensity of tooth structure, which bears no anatomical resemblance to the tooth, surrounded by a narrow radiolucent rim |
| Treatment             | Conservative surgical enucleation | Conservative surgical enucleation |

Odontoma. Histopathologically, compound odontoma consists of tooth-like structures embedded in a matrix which is loose fibrous in nature. Complex odontoma histopathologically largely shows dentin of the mature tubular form enclosing enamel represented by hollow structures due to decalcification.

Molecular alterations in odontoma formation

Recent studies have delineated multiple molecules aiding in the signaling and transcription of tooth morphogenesis. These include paired box gene 9, msh homeobox 1/2, runt-related transcription factor 2, bone morphogenetic proteins, fibroblast growth factors activin, lymphoid enhancer-binding factor 1 (Lef1), distal-less homeobox 1, Bar H-like homeobox 1, LIM homeodomains, and glioma-associated oncogene homolog L1/2/3. These factors are vital for tooth morphogenesis and are spatiotemporally expressed in the tooth germs during development.\[9,13,16\]

Odontoma is an analog of a developing tooth germ whose differentiation is incomplete either during the pre-ameloblastic or ameloblastic period, causing aberrant enamel organ mineralization. According to Crivelini et al. dysregulation in tooth morphogenesis and mineralization results in halts, the progression of normal tooth development culminating in the formation of odontoma.\[17,18\]

Together with transforming growth factor-β, β-catenins induce morphogenic changes in epithelial cells. β-catenins act as an intracellular signal transducing agent in the Wnt signaling pathway. Wnt signaling, in turn, inhibits degradation of β-catenins. Thus, mutant β-catenin through Shh and Bmp 4 pathway causes increased mesenchymal condensation which leads to excessive ectopic dental hard tissue formation. This could be one of the hypotheses for odontoma formation.\[19\] The stimulation of the Wnt pathway leads to abundant de novo tooth formation which is the possible etiology for supernumerary teeth and teeth-like structures.\[20\]

The Wnt-β-catenin-Tcf/Lef activated pathway is closely related to ghost cell formation in odontomas.\[21\] The cytokeratin (CK) profile of odontoma consists of CK-14 and CK-7 positivity and CK-19 negativity.\[22\] CK-19 is positive in normal dental tissue and can be used to differentiate from odontomas.

Conclusion

Despite odontoma being one of the most common forms of odontogenic tumors, its molecular biology remains relatively unexplored. Being a relatively non-aggressive odontogenic tumor, it is often not pursued in research. Since odontoma is a proliferating tumor with a close relationship to signaling pathways involved in normal tooth development, exploring its molecular pathogenesis could provide vital information which, in turn, could be exploited in research regenerating dental tissues.

References

1. Zoremchhingp, Joseph TB, Varma BC, Mungara JD. A compound composite odontoma associated with unerupted permanent incisor-a case report. J Indian Soc Ped Prev Dent 2004;22:144-7.
2. Jhamb AV, Dolas R, Panditwar P. Odontoma: Case series and report of cases complicated by infection and multiple denticles. Open J Stomatol 2012;2:44-9.
3. Chaudhari NT, Sharma NK, Kanodia DR, Sethy SK. Compound composite odontoma: Two case reports and review. J Oral Med Oral Surg Oral Pathol Oral Radiol 2015;1:83-8.
4. Morgan P. Odontogenic tumors: A review. Periodontology 2011;57:160-76.
5. Salgado H, Mesquita P. Compound odontoma-case report. Rev Port Estomatol Cir Maxilofac 2013;54:161-5.
6. Singh S, Singh M, Singh I, Khandelwal D. Compound composite odontome associated with an unerupted deciduous incisor-a
Odontoma

Patekar, et al.

rarity. J Indian Soc Pedod Prev Dent 2005;23:146-50.
7. Cuesta SA, Albiol JG, Aytes LB, Escoda CG. Revisión de 61 casos de odontoma. Presentación de un odontoma complejo erupcionado. Med Oral 2003;8:366-73.
8. Koussoulakou DS, Margaritis LH, Koussoulakos SL. A curriculum vitae of teeth: Evolution, generation, regeneration. Int J Biol Sci 2009;5:226-43.
9. Nelson BL, Thompson LD. Compound odontoma. Head Neck Pathol 2010;4:290-1.
10. Vengal M, Arora H, Ghosh S, Pai K. Large erupting complex odontoma: A case report. J Can Dent Assoc 2007;73:169-723.
11. Serra-Serra G, Berini-Aytés L, Gay-Escoda C. Erupted odontomas: A report of three cases and review of the literature. Med Oral Pathol Oral Cir Bucal 2009;14:E299-303.
12. Baldawa R, Khante K, Kalburge J, Kasat V. Orthodontic management of an impacted maxillary incisor due to odontoma. Contemp Clin Dent 2011;2:37-40.
13. An S, An C, Choi K. Odontoma: A retrospective study of 73 cases. Imaging Sci Dent 2012;42:77-81.
14. de Oliveira BH, Campos V, Marçal S. Compound odontoma—diagnosis and treatment: Three case reports. Pediatr Dent 2001;23:151-7.
15. Thesleff I, Sharpe P. Signalling networks regulating dental development. Mech Dev 1997;67:111-23.
16. Tucker A, Sharpe P. The cutting-edge of mammalian development; How the embryo makes teeth. Nat Rev Genet 2004;5:499-508.
17. Crivelini MM, de Araujo VC, de Sousa SO, de Araujo NS. Cytokeratins in epithelia of odontogenic neoplasms. Oral Dis 2003;9:1-6.
18. Kim JY, Jeon SH, Park JY, Suh JD, Choung PH. Comparative study of LHX8 expression between odontoma and dental tissue-derived stem cells. Oral Pathol Med 2011;40:250-6.
19. Xavier GM, Patist AL, Healy C, Pagrut A, Carreno G, Sharpe PT, et al. Activated WNT signaling in post-natal SOX2-positive dental stem cells can drive odontome formation. Sci Rep 2015;5:14479.
20. Tummers M, Thesleff I. The importance of signal pathway modulation in all aspects of tooth development. J Exp Zool B Mol Dev Envol 2009;312B:309-19.
21. Tanaka OA, Okamoto M, Yoshizawa D, Ito S, Alva PG, Ide F, et al. Presence of ghost cells and the Wnt signaling pathway. J Oral Pathol Med 2007;36:400-4.
22. Jeon SH, Yoon JY, Park YN, Jeong WJ, Kim S, Jho EH, et al. Axin inhibits extracellular signal-regulated kinase pathway by ras degradation via beta-catenin. J Biol Chem 2007;282:14482-92.

How to cite this article: Patekar D, Kheur S, Gupta AA. Odontoma - A brief overview. J Oral Dis Markers 2018;2:23-25.