Review Article

Hamartomas of the oral cavity

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Abstract

The majority of oral diseases present as growths and masses of varied cellular origin. Such masses may include simple hyperplasia, hamartoma, choristoma, teratoma, benign or malignant neoplasms. The distinguishing features of hamartomatous lesions are not certain, and often these non-neoplastic masses are indiscreetly denoted as neoplasms without weighing their pathology or biological behaviour. Essentially, understanding the dynamics of each of these disease processes forms an integral part of the appropriate treatment planning.

Key words: *Developmental disorders, hamartoma, oral cavity, syndrome, tumour*

INTRODUCTION

The term hamartoma is derived from the Greek word "hamartia" referring to a defect or an error.[1] It was originally coined by Albrecht in 1904 to denote developmental tumour-like malformations.[2] It can be defined as a non-neoplastic, unifocal/multifocal, developmental malformation, comprising a mixture of cytologically normal mature cells and tissues which are indigenous to the anatomic location, showing disorganized architectural pattern with predominance of one of its components.[1,3-5] The occurrence of multiple hamartomas in the same patient is often referred as hamartomatosis or pleiotropic hamartoma.[3,6]

Hamartomas are commonly observed in lung, pancreas, spleen, liver and kidney. They are rare in the head and neck region.[3] Within the oral cavity, indigenous tissues that might result in hamartomatous growths include odontogenic and non-odontogenic epithelial derivatives, smooth and skeletal muscle, bone, vasculature, nerve and fat.[5] Few hallmarks of a hamartoma based on literature include:[3,5,7,8]

- Developmental malformation may be present at birth, but manifests later
- Self-limited growth, co-ordinated with that of the surrounding tissues
- Can present as solitary or multiple masses
- May regress spontaneously
- Usually not encapsulated with ill-defined margins
- Not a true neoplasm, but a true neoplasm may develop in a hamartoma
- Microscopically, it consists of cytologically normal mature cells, native to the anatomic location
- Association with chromosomal abnormalities and syndromes.

Nevertheless, not all the lesions stated as hamartomas in the literature justify completely the above features. According to the data tabulated in Table 1, the most important features perceived are its limited growth potential after adolescence, microscopic appearance of

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Table 1: Summary of characteristic features of oral hamartomas

Oral hamartomas (possible hamartomatous lesions#)	Congenital origin	Limited growth potential after adolescence	Unencap-sulated	Cytologically normal cells & tissues, native to location	Associated chromosomal abnormalities	Malignant trans-formation	Spontaneous regression	Recurrence
Dens invaginatus	+	+	+	+	+	−	−	−
Dens evaginatus	+	+	+	+	−	−	−	−
Enamel pearl	+	+	+	+	−	−	−	−
Odontoma	−	+	−	+	+	−	−	−
Oral and labial melanotic macule	−	+	+	+	+	−	−	−
Oral melanocytic nevi	+/−	+	+	+	−	−	−	−
Hemangiomas	+/−	+	+	+	+	−	+	−
Vascular malformations	+/−	+	+	+	+	−	−	*
Glomuvenous malformations	−	+	+	+	−	−	−	−
Exostoses	−	−	+	+	−	−	−	*
Rhabdomyomatous hamartoma	+	+	+	+	+	−	−	−
Leiomyomatous hamartoma	+	+	+	+	−	−	−	−
Neurofibroma	−	+	+	+	+	*	−	−
Fibrolipomatous hamartoma of nerve	+	+	+	+	*	−	−	−
Oral neurovascular hamartoma	−	+	+	+	−	−	−	−
Oral neuromuscular hamartoma	−	+	+	+	−	−	*	−
Adenomatoid odontogenic tumour#	−	?	−	+	−	−	−	−
AF#, AFD# and AFO#	−	?	+	+	−	−	−	−
Hyperplastic dental follicle#	+	+	−	+	−	−	−	−
Adenomatoid hyperplasia of minor SG#	−	−	+	+	−	−	−	−
Fibrous dysplasia#	+	+	+	+	+	*	−	−
Familial gigantiform cementoma#	+/−	−	+	+	−	−	+	−
Mucosal neuroma#	−	+	+	+	+	−	−	−
Congenital granular cell tumour#	+	+	+	+	−	−	+	−

*+ (yes); − (no); ? (not determined); * (occasional). AF=Ameloblastic fibroma, AFD=Ameloblastic fibro-dentinoma, AFO=Ameloblastic fibro-odontoma, SG=Salivary gland*

unencapsulated admixture of mature cells native to the anatomic location and association with chromosomal aberrations.

The pathogenesis of hamartomas still remains speculative.[1] They are derived from any one of the embryonic lineages, most commonly the mesoderm. This is almost never in the case of neoplasm, where the neoplastic cells are clonally derived.[9]

Clinically, majority are asymptomatic and rarely pose any complications except when situated at the base of tongue. Given the non-neoplastic nature of hamartomas, conservative surgical excision is the treatment of choice. The prognosis is excellent, with nil or minute chances of recurrence.[3]

HAMARTOMATOUS GROWTH OF ODONTOGENIC APPARATUS

Dens invaginatus

Dens invaginatus is a developmental anomaly resulting in invagination of the enamel organ into the dental papilla before the mineralization of the dental tissues begin. The prevalence is 0.3–10%. Associated syndromes include William's syndrome, Nance-Huran syndrome, cranial suture syndromes and Ekman-Westborg–Julin syndrome.[10,11]

Dens evaginatus

Dens evaginatus (DE) represents an accessory cusp and is predominantly seen in people of Asian descent with a varying incidence of 0.5–4.3%. Clinically, DE may cause malocclusion resulting in abnormal wear or fracture and is treated accordingly.[12]

Enamel pearl/Enameloma

They represent deposits of enamel located at the cemento-enamel junction or at the furcation area. Its prevalence varies between 1.1 and 9.7%. Rarely, it may be detected within the dentin, when it is known as internal enamel pearl. Clinical significance varies depending upon its topographic relation with the furcation area.[13,14]

Odontoma

According to Reichart and Philipsen and the World Health Organization (WHO) 2005, compound and complex odontomas are hamartomatous lesions.[15,16] They have a relative frequency of 4.2–73.8% and 5–30%, respectively.[15] Associated syndromes include Gardner's and Hermann's syndromes.[17]

HAMARTOMATOUS GROWTH OF EPITHELIAL DERIVATIVES

Oral and labial melanotic macule

They represent a well-circumscribed flat area of brown to black mucosal pigmentation. There is an increase in melanin production by normal mature melanocytes (without increase in their number).[18] It is associated with Peutz–Jeghers syndrome and Addison's disease.[19]

Oral melanocytic nevi

Nevi or mole represents collection of nevus cells which are derivatives of melanocytes or their precursor neural crest cells. Oral nevi are usually small and show regular symmetrical outline with no change in colour, shade or texture over time. Static nevi do not require excision and may be followed up.[19]

HAMARTOMATOUS GROWTH OF MESENCHYMAL DERIVATIVES

Congenital and infantile haemangioma

Congenital haemangioma (CH) and infantile haemangioma (IH) are present at birth or develop in the infancy period.[20,21] Majority of them involute spontaneously or gradually over the years. Microscopically, the proliferative phase of IH and CH comprises complex cellular mixtures, chiefly the endothelial cells.[20,22] Associated syndromes include PHACES and LUMBAR syndrome.[20]

Vascular malformations

Vascular malformation (VM) refers to congenital morphogenic anomalies of the various vessels.[20] Histologically, they comprise of normal vascular components.[20,22] VM may occur as primary or in association with regional or diffuse syndromes such as Sturge–Weber, Klippel–Trénaunay, proteus syndrome, Bannayan–Riley–Ruvalcaba syndrome, and Osler–Weber–Rendu, to mention a few.[20]

Glomuvenous malformations

Glomuvenous malformation (GVM) occurs more often in children.[23] Clinically, it appears as red-to-blue nodules or multifocal plaque-like lesions. Microscopically, GVM is composed of varying

proportion of blood vessels and glomus cells. The familial cases have been linked to mutations in the glomulin gene located in chromosome 1p21-22.[22,24]

Exostoses

They are described as peripheral localized overgrowth of the bone. Based on the anatomic location in the jaws, they are termed as buccal bone exostoses, torus palatinus, and torus mandibularis.[25] Surgical intervention is required only in case of tissue trauma, periodontal or prosthodontic impediment.[26]

Rhabdomyomatous mesenchymal hamartoma

It is an exceptionally rare congenital lesion of the oral cavity. It chiefly comprises of striated muscle tissue.[27] It is associated with Delleman, amniotic band and Goldenhar syndromes.[28]

Leiomyomatous hamartoma

Leiomyomatous hamartoma is another rare entity which commonly involves the midline of palate and tongue. Microscopically, it consists of an unencapsulated mass of smooth muscle.[29,30]

Neurofibroma

As the name indicates, it is an admixture of perineural fibroblasts and Schwann cells. It is associated with von Recklinghausen's neurofibromatosis syndrome. Approximately 12% of cases are associated with the syndrome tend to develop malignancy.[6]

Fibrolipomatous hamartoma of nerve

Neural fibrolipoma or fibrolipomatous hamartoma of nerve (FLHN) is a tumour-like lipomatous process. FLHN was reported in the pharyngeal mucosa by Kumar *et al*.[31] Some lesions may represent carpal tunnel syndrome as a late complication.[23]

Oral neurovascular hamartoma

The hamartomatous nature of oral neurovascular hamartoma can be supported by the characteristics such as limited growth potential, ill-defined borders and histologically consisting of closely packed groups of well-formed nerve bundles and vessels.[5]

Oral neuromuscular hamartoma

Oral neuromuscular hamartomas or triton tumours are reported to occur in the trigeminal nerve and tongue. Histologically, they show presence of mature neural and striated muscle tissue.[32,33]

SYNDROMIC HAMARTOMAS

Tuberous sclerosis

Tuberous sclerosis (TS) is a rare syndrome characterized by the classic triad of seizures, mental deficiency and angiofibromas, affecting about 1 in 6000 people.[34] Oral hamartomas in TS were reported by Celenk *et al*. (2005) and Amin and O'Callaghan (2012).[34,35]

Cowden syndrome/multiple hamartoma syndrome

Cowden syndrome represents the principal *PTEN* (phosphatase and tensin homolog) gene-related disorder which occurs in 1 in 200,000 people.[34] The oral manifestations include multiple papules involving the gingivae, buccal mucosa and dorsum of tongue.

Proteus syndrome

Proteus syndrome is a rare congenital hamartomatous condition with an incidence of less than 1 in a population of 1 million.[34] Reported associated oral hamartomas include exostoses of facial bones and lymphangiomas.[36]

Oral-facial-digital syndrome

Oral-facial-digital syndrome (OFDS) comprises a group of heterogeneous disorders with an incidence of 1 in 50,000–250,000 newborns.[34] The oral hamartomatous findings include lingual hamartomas (in 70% cases of OFDS I). Microscopically, they are composed of muscles, adipose tissues and salivary glands.[37]

CONTROVERSIAL ORAL HAMARTOMATOUS LESIONS

Adenomatoid odontogenic tumour

The hamartomatous nature of adenomatoid odontogenic tumour is supported by its limited growth potential and lack of recurrence.[38] Nevertheless, its biological behaviour has been a topic of long debate and the related research is depicted in Table 2.

Ameloblastic fibroma, ameloblastic fibro-dentinoma and fibro-odontoma

Reichart and Philipsen proposed a neoplastic and hamartomatous line of development for the mixed odontogenic tumours. But currently, there is no substantial evidence to prove either of the above hypothesis.[15]

Table 2: Literature relevant to the biological behaviour of AOT

Research supporting hamartomatous nature	Research supporting neoplastic nature
Similar immuno-histochemical composition of AOT w.r.t. reduce enamel epithelium[39]	Most odontogenic tumours are monoclonal[47]
Ameloblastoma has greater proliferative potential than the AOT[40]	Marked expression of cyclin D1 in AOT[48]
Low Ki-67 expression[41]	The spindle-shaped tumour cells of AOT show close associations with extracellular matrix signalling as well as cell proliferation[49]
Neither p16 expression nor Ki-67 expression[42]	Strong immunolocalization of HGF (Hepatocyte growth factor) and c-met in squamous cells present in AOTs[50]
More invasive behaviour of ameloblastomas compared to AOTs[43]	Strong cytoplasmic expression of β-catenin[51]
Stronger expression of p53 in ameloblastoma compared to AOT[44]	Similar proliferative potential between ameloblastoma and AOT[52]
Ameloblastoma has more aggressive behaviour when compared to AOT[45]	
Higher percentage of Ki-67 and Bcl-2 in solid ameloblastoma compared to AOT[46]	

AOT=Adenomatoid odontogenic tumour

Hyperplastic dental follicle

It has been referred as an odontogenic hamartomatous lesion associated with single/multiple unerupted teeth. It commonly involves permanent first and second molars. Microscopically, it comprises of odontogenic epithelium and calcifications.[53,54]

Adenomatoid hyperplasia of minor salivary gland

It is a rare lesion of the minor salivary glands. Clinically it presents as a solitary, painless mass or nodule.[6] Microscopically, it comprises of lobular aggregates of normal mucus acini.[6,25] Recurrence and malignancy are not reported.[6]

Fibrous dysplasia

Fibrous dysplasia is a developmental tumour-like condition that becomes relatively static after skeletal maturation.[6,25] It is associated with Jaffe–Lichtenstein syndrome, McCune Albright syndrome, and Mazabraud's syndrome.[25] Malignant transformation is reported in 0.5% (1 in 200) of cases.[6]

Familial gigantiform cementoma

It is an extremely rare cemento-osseous disease restricted to the jaws.[55,56] Its growing potential can be correlated with skeletal growth and maturation.[55] If left untreated, the enlargement eventually ceases during the fifth decade.[25]

Mucosal neuroma

It is commonly associated with multiple endocrine neoplasia (MEN) IIB syndrome. Clinically, it occurs in multiple small masses. Microscopically, it is partially encapsulated and contains aggregation or proliferation of histologically normal nerves.[6]

Congenital granular cell tumour

Congenital granular cell tumour is thought to be a variant of granular cell tumour, but the exact nature of the lesion is unclear. It exclusively occurs in infants or immediately after birth. Most of the lesions cease to grow or regress spontaneously without intervention.[19,23]

CONCLUSION

Oral hamartomas are unique presentations of the head and neck region. Nevertheless, the criteria to delineate hamartomas from other similar masses are ambiguous. To conclude, hamartomas should promptly be included in the differential diagnosis of the tumours of oral cavity, essentially the paediatric tumours, to avoid aggressive treatment and morbidity.

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Conflicts of interest

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REFERENCES

1. Pilch BZ. Larynx and hypopharynx. In: Pilch BZ, editor. Head and Neck Surgical Pathology. Philadelphia, USA: Lippincott Williams and Wilkins; 2001. p. 230-83.
2. Craig O. Hamartoma. Postgrad Med J 1965;41:636-8.
3. Barnes L. Surgical Pathology of the Head and Neck. Vol. 2. 2nd ed. New York, USA: Marcel Dekker; 2001. p. 1649-72.
4. Kumar V, Abbas AK, Aster JC, editors. Robbins Basic Pathology. 9th ed. Philadelphia, USA: Saunders, Elsevier; 2013. p. 257.
5. Allon I, Allon DM, Hirshberg A, Shlomi B, Lifschitz-Mercer B, Kaplan I. Oral neurovascular hamartoma: A lesion searching for a name. J Oral Pathol Med 2012;41:348-53.
6. Gnepp DR. Intraosseous mucoepidermoid carcinoma. In: Gnepp DR, editor. Diagnostic Surgical Pathology of the Head and Neck. 2nd ed. Philadelphia, USA: Saunders, Elsevier; 2009. p. 816-8.

7. Das S. A Concise Textbook of Surgery. 3rd ed. Calcutta, India: Dr. S. Das; 2001. p. 457.
8. Walter JB, Israel MS. Wound healing. In: Walter JB, Israel MS, editor. General Pathology. 6th ed. USA: Churchill Livingstone; 1987. p. 117-2.
9. Berman JJ. Neoplasms: Principles of Development and Diversity. USA: Jones and Bartlett Publishers; 2009. p. 429.
10. Alani A, Bishop K. Dens invaginatus. Part 1: Classification, prevalence and aetiology. Int Endod J 2008;41:1123-36.
11. Bishop K, Alani A. Dens invaginatus. Part 2: Clinical, radiographic features and management options. Int Endod J 2008;41:1137-54.
12. Levitan ME, Himel VT. Dens Evaginatus: Literature review, pathophysiology and comprehensive treatment regimen. J Endod 2006;32:1-9.
13. Chrcanovic BR, Abreu MH, Custódio AL. Prevalence of enamel pearls in teeth from a human teeth bank. J Oral Sci 2010;52:257-60.
14. Akgül N, Caglayan F, Durna N, Sümbüllü MA, Akgül HM, Durna D. Evaluation of enamel pearls by cone-beam computed tomography (CBCT). Med Oral Patol Oral Cir Bucal 2012;17:e218-22.
15. Reichart PA, Philipsen HP. Odontogenic Tumors and Allied Lesions. London, UK: Quintessence Publishing Co. Ltd; 2004. p. 41-115.
16. Barnes L, Eveson JW, Reichart P, Sidransky D, editors. World Health Organization Classification of Tumours: Pathology and Genetics Head and Neck Tumours. USA: WHO Publications Center; 2005. Available from: www.iarc.fr/IARCPress/pdfs/index1.php. [Last accessed on 2015 Feb 28].
17. Satish V, Prabhadevi MC, Sharma R. Odontome: A brief review. Intl J Clinical Ped Dentistry 2011;4:177-85.
18. Greenberg MS, Glick M, Ship JA. Burket's Oral Medicine. 11th ed. Ontario: BC Decker Inc; 2008. p. 131-2.
19. Marx RE, Stern D. Oral and Maxillofacial Pathology: A Rationale for Diagnosis and Treatment. 1st ed. Illinois, USA: Quintessence Publishing Co, Inc; 2003. p. 707-36, 427-28.
20. Lowe LH, Marchant TC, Rivard DC, Scherbel AJ. Vascular malformations: Classification and terminology the radiologist needs to know. Semin Roentgenol 2012;47:106-17.
21. Ethunandan M, Mellor TK. Haemangiomas and vascular malformations of the maxillofacial region: A review. Br J Oral Maxillofac Surg 2006;44:263-72.
22. North PE. Pediatric vascular tumors and malformations. Surg Pathol 2010;3:455-94.
23. Weiss SW, Goldblum JR, Folpe AL. Enzinger and Weiss's Soft Tissue Tumors. 4th ed.. Philadelphia, USA: Mosby, Elsevier; 2008. P.617-18, 1187-88.
24. Marler JJ, Mulliken JB. Current management of hemangiomas and vascular malformations. Clin Plast Surg 2005;32:99-116, ix.
25. Neville BW, Damm DD, Allen CM, Bouquot JE. Oral and Maxillofacial Pathology. 3rd ed. India: Saunders, Elsevier; 2009. p. 163-6.
26. Smitha K, Smitha GP. Alveolar exostosis revisited: A narrative review of the literature. Saudi J Dent Res 2015;6:67-72.
27. Dal Vechio A, Nakajima E, Pinto D Jr, Azevedo LH, Migliari DA. Rhabdomyomatous (mesenchymal) hamartoma presenting as haemangioma on the upper lip: A case report with immunohistochemical analysis and treatment with high-power lasers. Case Rep Dent 2013;2013:943953.
28. Rodrigues FA, Cysneiros MA, Rodrigues Jr R, Sugita DM. Rhabdomyomatous mesenchymal hamartoma: A case report. J Bras Patol Med Lab 2014;50:165-8.
29. Nava-Villalba M, Ocampo-Acosta F, Seamanduras-Pacheco A, Aldape-Barrios BC. Leiomyomatous hamartoma: Report of two cases and review of the literature. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2008;105:e39-45.
30. Iida S, Kishino M, Senoo H, Okura M, Morisaki I, Kogo M. Multiple leiomyomatous hamartoma in the oral cavity. J Oral Pathol Med 2007;36:241-4.
31. Kumar N, Mittal M, Sinha M, Thukral B. Neural fibrolipoma in pharyngeal mucosal space: A rare occurrence. Indian J Radiol Imaging 2012;22:358-60.
32. Castro DE, Raghuram K, Phillips CD. Benign triton tumor of the trigeminal nerve. AJNR Am J Neuroradiol 2005;26:967-9.
33. Daley TD, Darling MR, Wehrli B. Benign Triton tumor of the tongue. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2008;105:763-6.
34. Genetic Home Reference. Available from: http://www.ncbi.nlm.nih.gov/omim. [Last accessed on 2015 Feb 27].
35. Celenk P, Alkan A, Canger EM, Günhan O. Fibrolipomatous hamartoma in a patient with tuberous sclerosis: Report of a case. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005;99:202-6.
36. Becktor KB, Becktor JP, Karnes PS, Keller EE. Craniofacial and dental manifestations of proteus syndrome: A case report. Cleft Palate Craniofac J 2002;39:233-45.
37. Mihci E, Tacoy S, Ozbilim G, Franco B. Oral-facial-digital syndrome type 1. Indian Pediatr 2007;44:854-6.
38. Tegginamani A, Kudva S, Shruthi D K, Karthik B, Hargavannar VC. Adenomatoid odontogenic tumor-hamartoma/cyst or true neoplasm; a Bcl-2 immunohistochemical analysis. Indian J Dent Adv 2012;4:730-5.
39. Crivelini MM, Soubhia AMP, Felipini RC. Study on the origin and nature of the adenomatoid odontogenic tumor by immunohistochemistry. J Appl Oral Sci 2005;13:406-12.
40. Barboza CA, Pereira Pinto L, Freitas Rde A, Costa Ade L, Souza LB. Proliferating cell nuclear antigen (PCNA) and p53 protein expression in ameloblastoma and adenomatoid adontogenic tumor. Braz Dent J 2005;16:56-61.
41. Leon JE, Mata GM, Fregnani ER, Carlos-Bregni R, de Almeida OP, Mosqueda-Taylor A, *et al.* Clinicopathological and immunohistochemical study of 39 cases of adenomatoid odontogenic tumour: A multicentric study. Oral Oncol 2005;41:835-42.
42. Suzuki H, Hashimoto K. Adenomatoid odontogenic tumour of the maxilla: Immunohistochemical study. Asian J Oral Maxillofac Surg 2005;17:267-72.
43. de Medeiros AM, Nonaka CF, Galvão HC, de Souza LB, Freitas Rde A. Expression of extracellular matrix proteins in ameloblastoma and adenomatoid odontogenic tumors. Eur Arch Otorhinolaryngol 2010;267:303-10.
44. Salehinejad J, Zare-Mahmoodabadi R, Saghafi S, Jafarian AH, Ghazi N, Rajaei AR, *et al.* Immunohistochemical detection of p53 and PCNA in ameloblastoma and adenomatoid odontogenic tumor. J Oral Sci 2011;53:213-7.
45. Krishna A, Kaveri H, Naveen Kumar RK, Kumaraswamy KL, Shylaja S, Murthy S. Overexpression of MDM2 protein in ameloblastomas as compared to adenomatoid odontogenic tumor. J Cancer Res Ther 2012;8:232-7.
46. Razavi SM, Tabatabaie SH, Hoseini AT, Hoseini ET, Khabazian A. A comparative immunohistochemical study of Ki-67 and Bcl-2 expression in solid ameloblastoma and adenomatoid odontogenic tumor. Dent Res J (Isfahan) 2012;9:192-7.

47. Gomes CC, Oliveira Cda S, Castro WH, de Lacerda JC, Gomez RS. Clonal nature of odontogenic tumor. J Oral Pathol Med 2009;38:397-400.
48. Kumar H, Vandana R, Kumar G. Immunohistochemical expression of cyclin D1 in ameloblastomas and adenomatoid odontogenic tumors. J Oral Maxillofac Pathol 2011;15:283-7.
49. Tsuneki M, Maruyama S, Yamazaki M, Cheng J, Saku T. Podoplanin expression profiles characteristic of odontogenic tumor-specific tissue architectures. Pathol Res Pract 2012;208:140-6.
50. Poomsawat S, Punyasingh J, Vejchapipat P, Larbcharoensub N. Co-expression of hepatocyte growth factor and c-met in epithelial odontogenic tumors. Acta Histochem 2012;114:400-5.
51. Harnet JC, Pedeutour F, Raybaud H, Ambrosetti D, Fabas T, Lombardi T. Immunohistological features in adenomatoid odontogenic tumor: Review of the literature and first expression and mutational analysis of β-catenin in this unusual lesion of the jaws. J Oral Maxillofac Surg 2013;71:706-13.
52. Moosvi Z, Rekha K. c-Myc oncogene expression in selected odontogenic cysts and tumors: An immunohistochemical study. J Oral Maxillofac Pathol 2013;17:51-6.
53. Cho YA, Yoon HJ, Hong SP, Lee JI, Hong SD. Multiple calcifying hyperplastic dental follicles: Comparison with hyperplastic dental follicles. J Oral Pathol Med 2011;40:243-9.
54. Schmitd LB, Bravo-Calderón DM, Soares CT, Oliveira DT. Hyperplastic dental follicle: A case report and literature review. Case Rep Dent 2014;2014:251892.
55. Abdelsayed RA, Eversole LR, Singh BS, Scarbrough FE. Gigantiform cementoma: Clinicopathologic presentation of 3 cases. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001;91:438-44.
56. Eversole R, Su L, ElMofty S. Benign fibro-osseous lesions of the craniofacial complex. A review. Head Neck Pathol 2008;2:177-202.