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Review Article

Hamartomas and Choristomas of the Oral Cavity: A New Perspective

Hussain M. Alkhames[1] R. Venkata Subramanyam[2]

[1] Anatomy, Edinburgh Medical School: Biomedical Sciences, College of Medicine and Veterinary Sciences, University of Edinburgh, Edinburgh, United Kingdom
[2] Oral Pathology, Sumandeep Vidyapeeth K M Shah Dental College and Hospital, Vadodara, Gujarat, India

Address for correspondence R. Venkata Subramanyam, MDS, Sumandeep Vidyapeeth K M Shah Dental College and Hospital, Vadodara, Gujarat, India (e-mail: subrarv@gmail.com).

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Abstract

Hamartomas and choristomas are malformations that often manifest at birth, usually seen as a discrete, localized mass or as multiple masses that resemble a neoplasm, but not a true neoplasm with the exception in choristomas that the cells and tissues are not normally found at that location. Clear etiology and pathogenesis are not found. In addition, the distinguishing features of these masses are not certain. It is essential to differentiate between hamartomas, choristomas, and other oral cavity masses in order to provide proper management. In this study, a new perspective is provided for hamartomas and choristomas of the oral cavity.

Keywords

- hamartoma
- choristoma
- malformation
- criteria
- odontogenic
- nonodontogenic

Introduction

The word hamartoma is derived from Ancient Greek ἁμαρτία (*hamartía*, "error, failure"), from the verb ἁμαρτάνω (*hamartánō*, "to miss the mark"), and the suffix from Ancient Greek -μα (-oma) indicating tumors or masses.[1] The term hamartoma was introduced by Albrecht in 1904.[2] Although there are numerous definitions for hamartoma, all of them agree that it is a malformation, usually seen as a discrete, localized mass, or as multiple masses, and that it often manifests at birth, although it may present later in life. It resembles a neoplasm, but *not* a true neoplasm, clinically benign and often asymptomatic and histologically an abnormal mixture of cells and tissues, or an abnormal proportion of a single component, native to that location. It grows at virtually the same rate as normal components and may regress spontaneously. It may occasionally transform into a true neoplasm or compress adjacent tissues.[1,3–6]

The definition of choristoma is similar to hamartoma in all aspects, except that the cells and tissues are *not* normally found at that location. The term is derived from Ancient Greek *chōristos*, for separated. It has also been called **ectopia** or **heterotopia**.[3,7–9]

Etiopathogenesis of Hamartomas

Hamartomas result from the abnormal formation of normal tissue and sometimes occur sporadically and a few times as a part of a syndrome. Hamartoma is most likely due to a developmental error and may appear in several sites. It grows at the same rate as of the original tissue. There are also some genes involved in the pathogenesis of the development of hamartoma, including SMAD4, phosphatase and tensin homolog gene (PTEN), STK1, and BMPR1A.[10]

A broad spectrum of disorders and syndromes is associated with gene mutations, resulting in multiple hamartomas in various body parts. Loss of function of the PTEN gene by mutation results in PTEN hamartoma tumor syndrome (PHTS). Cowden's syndrome (CS) is the best-studied phenotype within PHTS. In addition to multiple hamartoma formation, patients have dermatologic manifestations such as oral fibromas, trichilemmomas, and punctate palmoplantar keratoses. These are also associated with increased malignant potential.[11]

Pathogenesis of Hamartomas

Hamartomas are fundamentally a clearly demarcated mass comprising disordered replications of normal tissue cells,

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depending on the anatomic location. Microscopically, they may have characteristics similar to any benign tumor, such as haphazard growth of the normal tissue and architectural pattern of cytologically normal cells native to the local site. However, there is usually no sign of metastasis or local invasion.[10] Some authors have described hamartomas as developmental malformations. All malformations are developmental, so the use of the term developmental as an adjective for malformation is redundant.

Is there a need for a separate term? Is there a difference between a malformation and a hamartoma? Are all malformations hamartomas? This makes it imperative to have a definite set of inclusion and exclusion criteria for hamartomas and choristomas to avoid indiscriminate use of these terms.

Inclusion/Exclusion Criteria

A hamartoma has to be an abnormal mass of tissue. In other words, malformations that are structural defects (e.g., cleft palate) should not be considered hamartomas. To state succinctly, all hamartomas are malformations, but all malformations are not hamartomas. An anomaly is a malformation that does not affect the function and should not be mistaken for a hamartoma. A hamartoma can be composed of a single type of tissue or a combination of tissues and may be classified accordingly. However, the tissue components *should be local* to that site of occurrence. If tissues present are not native to that location but otherwise all other criteria are satisfied, then the term choristoma can be employed.

A syndrome should not be considered a hamartoma unless it is a syndrome of hamartomas. A good example is multiple hamartoma syndrome or CS. In a similar vein, a malformation, and subsequently derived developmental defects (sequence), should not be considered a hamartoma.[12] The criteria are listed in ►**Table 1**.

Malformations That Are Not Hamartomas

Traditionally, dens invaginatus, dens evaginatus, and enamel pearls have been listed as hamartomas.[13] Dens invaginatus (also called dens in dente or tooth within a tooth) results from invagination of the enamel organ before mineralization of the tooth and forms a deep pit, lined by enamel. It can be seen in the crown (coronal dens invaginatus) or the root (radicular dens invaginatus). Dens evaginatus is like a supernumerary cusp, due to excessive, localized proliferation of the enamel organ, commonly seen on the occlusal surface of premolars as a tubercle. Enamel pearls are blebs of enamel deposited on the root surfaces.[14–16] All these are not abnormal masses of growth, but malformations occurring during development resulting in a dental defect. Therefore, these are better considered developmental anomalies rather than hamartomas.

Table 1 Criteria for hamartomas

Major criteria
1. It should be developmental in origin
2. The lesion should a mass of tissue
3. Histologically, the tissue components must be native to the site
4. The growth rate should be normal, similar to adjacent normal tissues
5. It should be benign with no malignant transformation
Minor criteria
1. The lesion should stop growing after a certain period
2. The lesion should undergo spontaneous regression
3. It should be seen at birth or shortly after
4. It should be asymptomatic (other than its appearance)
Exclusion criteria
1. Obvious structural defects of developmental origin
2. Reactive lesions that undergo spontaneous regression
3. True benign neoplasms

Note: To be considered a hamartoma, a lesion should satisfy all the major criteria and exclusion criteria. Minor criteria are optional.
All the above criteria can be used for choristomas, except[3] the major criteria. In the case of choristomas, the tissue components must NOT be native to the site.

Hamartomas of Odontogenic Origin

Odontomas have traditionally been described as benign odontogenic tumors, derived from both epithelial and mesenchymal tissues and their prevalence exceeds all other odontogenic tumors.[16] They have been classified as compound composite odontoma or complex composite odontoma based on the arrangement of their tissues. They are typically asymptomatic and revealed in routine radiographs, although clinical signs such as delayed eruption of permanent teeth and, in severe cases, infection and lymphadenopathy might occur.[17] The compound type has a regular arrangement of dental tissues resembling many small teeth (denticles), whereas the complex odontomas are composed of haphazardly arranged dental hard tissues.[14–16] But these masses seen in the jaws are known to be the result of malformation and therefore better labeled as hamartomas rather than benign neoplasms. According to the "maturation concept," odontomas are the end stage in the continuum of differentiation of ameloblastic fibroma (AF) to ameloblastic fibro-odontoma (AFO).[18] In other words, the lesion begins as AF, differentiates into AFO, and finally matures into odontoma (►**Fig. 1**).

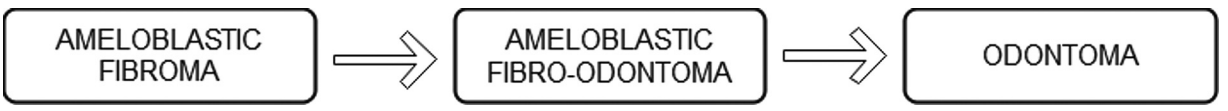

Fig. 1 Sequence of pathogenesis of odontoma.

However, all do not agree with this theory. Some authors believe that AFO is a hamartoma and an immature complex odontoma and that AF is a neoplasm and not a precursor of AFO.[19,20]

Some authors are of the view that both AF and AFO are two distinct entities and both are neoplasms.[21] Trodahl suggested that odontomas have a noncalcified stage of development, which histologically resembles AF, which is a true neoplasm.[22] Similarly, odontoma and AFO also have similar histopathological features and it is known that some of the latter do show aggressive behavior and bone destruction. Hence, it is highly likely that there are two variants of AFO—one a hamartoma and the other a true neoplasm.[23] Recently, in the 2017 WHO Classification of Head and Neck Tumors, primordial odontogenic tumor has been included.[24] Some authors feel that it represents a stage in the development of AF rather than a true neoplasm.[25] Generally, the management of odontoma is surgical excision. However, AF and AFO are usually treated with an enucleation and curettage surgery.[16]

AF and AFO are not the only odontogenic tumors that are considered hamartomas. Adenomatoid odontogenic tumors (AOTs), peripheral odontogenic tumors (POTs), peripheral ameloblastomas (PAs), squamous odontogenic tumors (SOTs), and adenoid ameloblastoma with dentinoid are other odontogenic lesions that are also considered hamartomas.[26,27]

AOT has been considered by many as a hamartoma because of its clinical behavioral pattern, radiographic appearance, and essentially no recurrences after surgical removal. Reichart et al did an immunohistochemical profile of the AOT and concluded that its biologic behavior is more consistent with a hamartoma than neoplasm. This has been substantiated by lower or negligible levels of Ki-67 and Bcl-2 (cell proliferation and anti-apoptosis markers), metallothionein (marker for recurrence potential), matrix metalloproteinase (marker for local aggressiveness), MDM2 (proliferative marker), and integrin types a2b1, a3b1, and a5b1 (markers of invasion) when compared to ameloblastoma. In addition, higher levels of β-catenin confer a greater level of cell adhesion properties in AOT.[28]

A soft-tissue variant of AOT is known to exist, which is also considered a hamartoma.[29] In fact, most odontogenic tumors have a peripheral counterpart, which vary in behavior from their central, intraosseous variants. All these can be clubbed under the umbrella of POTs, regardless of their central counterpart.[30] These may arise from the remnants of dental lamina (rests of Serres) or from the basal layers of surface epithelium, which undergo a hamartomatous transformation.[31] The authors are of the view that all POTs should be labeled as peripheral odontogenic hamartomas (POHs). A mind map of common hamartomas of the oral cavity is shown in ►**Fig. 2**.

Hamartomas of Nonodontogenic Origin

Although hamartomas of odontogenic origin are more likely to occur in the oral and maxillofacial region, hamartomas of nonodontogenic origin are not uncommon. The most common one is hemangioma, an abnormal proliferation of blood vessels, which can develop in any vascularized tissue. Globally, the reported overall range is 2 to 10%; this is due to the differences in infant age, study methodologies, countries, and ethnicities.[32] Among all complications of hemangiomas, ulceration appears to be the most common type.[32]

Some authors are of the opinion that hemangiomas are true neoplasms,[33] whereas there are others who favor the concept that these are hamartomas.[34] To add to the confusion, we have the term *vascular malformation*. Both hemangioma and vascular malformations are congenital vascular anomalies; the former is considered as a neoplasm of endothelial cells, whereas the latter caused by abnormal blood vessel morphogenesis.[35] Characteristically, hemangioma is well circumscribed, with a rapid growth phase initially, which usually undergoes spontaneous involution and is unlikely to recur after removal.[16,35] These features are more consistent with a hamartoma than a true neoplasm. Unlike hemangiomas, there is not much debate about lymphangioma, which is considered a hamartoma of lymphatic vessels. These lymphatic malformations arise from lymphatic tissue that have separated from the rest of the lymphatic system.[16] In the management of lymphangioma, surgical excision is commonly utilized. However, sclerotherapy also appears to be effective.[36]

There are developmental tumor-like conditions of bone, which can be considered hamartomas. Fibrous dysplasia is one such example, which belongs to the category of fibro-osseous lesions, which are characterized by replacement of normal bone with cellular fibrous connective tissue and in which irregular woven bone trabeculae are formed.[16] Fibrous dysplasia is basically of two types: monostotic (involving only one bone) or polyostotic (affecting many bones). The latter can be further classified as McCune–Albright type, Jaffe–Lichtenstein type, and craniofacial fibrous dysplasia.[37] Fibrous dysplasia is of developmental origin and its growth stabilizes after puberty and occasionally undergoes spontaneous involution. Nevertheless, in case of large extensive lesions, surgical countering might be required.[16] In fact, some authors are of the opinion that some osteomas could represent the end stage of fibrous dysplasia and therefore should be regarded as hamartomas rather true neoplasms. Tori (palatal and mandibular) and exostoses are also developmental in origin and should likewise be viewed as hamartomas.[16]

Another fibro-osseous lesion, cherubism, erroneously labeled as familial fibrous dysplasia, can also be regarded as a hamartoma. It is a developmental condition, inherited as an autosomal dominant trait, usually seen as bilateral symmetrical expansion of the jaws before 5 years of age, and it typically progresses until puberty, stabilizes, and then later slowly regresses.[16]

Hamartomas of Salivary Gland Origin

Hamartomas of the salivary gland are quite uncommon. These are usually seen as asymptomatic masses, without any particular age or gender predilection and are generally classified as vascular and nonvascular types. These are histologically composed of normally appearing acini and ductal elements, admixed with mature lipocytes and variable

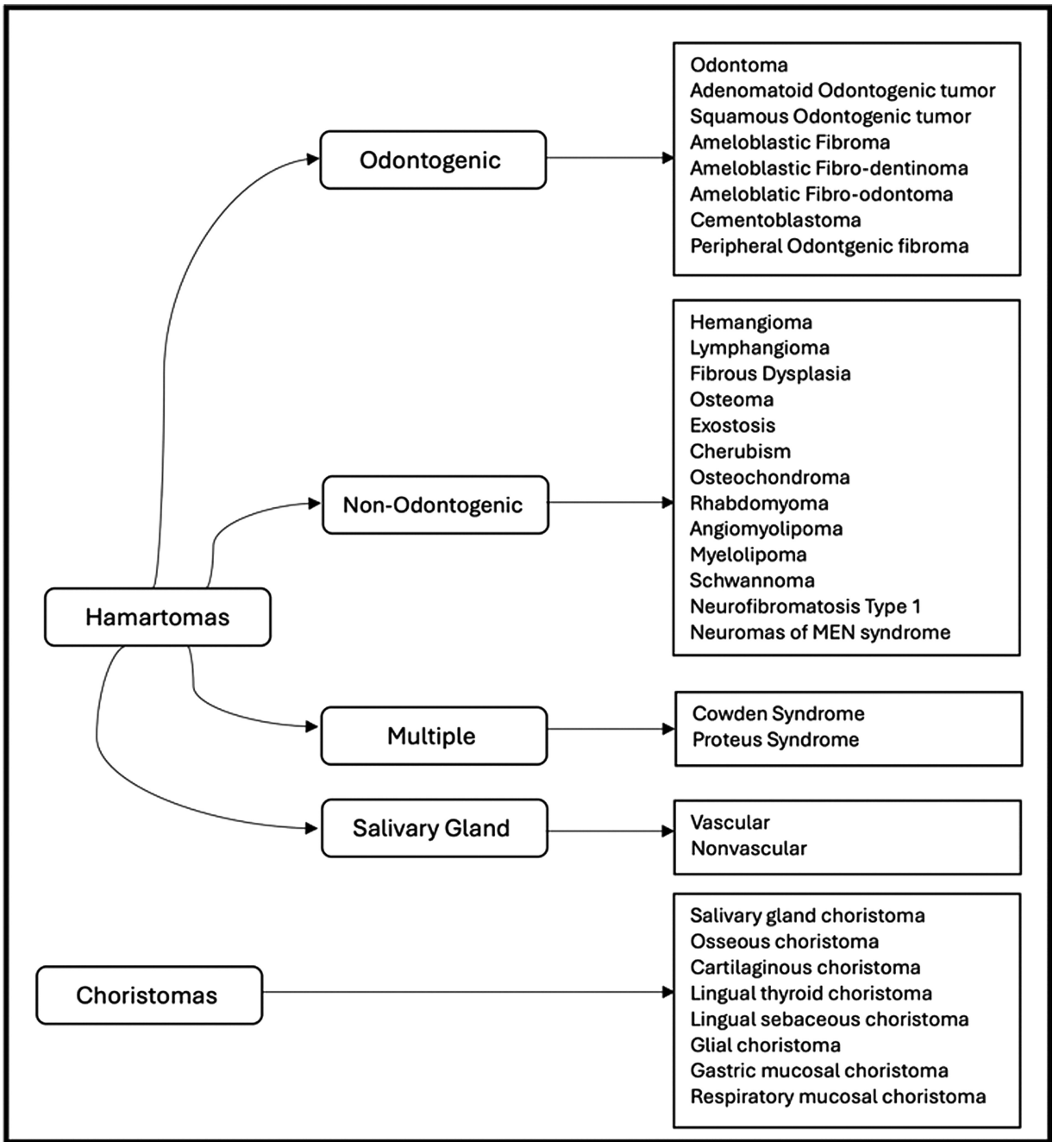

Fig. 2 Mind map of hamartomas and choristomas of the oral cavity. MEN, multiple endocrine neoplasia.

proportions of blood vessels, and may or not be encapsulated.[38] Sometimes eosinophilic adenomatous nests, oncocytic hyperplasia, sebaceous metaplasia, and lymphoid tissue component can also be observed.[39] It is not easy to distinguish them from benign tumors or salivary gland hyperplasia. Cases with similar histological features have been reported as sialolipoma[40] or as sialoangiolipoma,[41] depending on the vascularity.

Miscellaneous Hamartomas: Cowden's Syndrome

Multiple hamartoma syndrome or CS is an autosomal dominant disorder linked to mutation in the PTEN and characterized by trichilemmomas, oral cobblestone fibromas, palmoplantar hyperkeratosis, fibrocystic changes of the breast, sclerotic fibromas, thyroid adenomas, and gastrointestinal polyps, to name a few.[42]

It was first described in 1963 and named after a patient, Rachel Cowden.[43] CS is of particular importance interest to the dental surgeon because mucocutaneous lesions are common, especially oral papillomas (cobblestone-like appearance), which are primarily seen on the tongue.[44] Although the term multiple hamartoma syndrome is reserved for Cowden's disease, multiple hamartomas are seen in other conditions like tuberous sclerosis, Brooke–Spiegler syndrome, Peutz–Jeghers syndrome, and Birt–Hogg–Dubé syndrome.[45]

Choristomas

When compared to hamartomas, choristomas are quite uncommon in the oral cavity. They can be listed as follows: salivary gland choristoma, cartilaginous choristoma, osseous choristoma, lingual thyroid choristoma, lingual sebaceous choristoma, glial choristoma, and respiratory/gastric mucosal choristomas (►**Fig. 2**).[46] Of all the various choristomas, the most popular one is Stafne's bone cavity or intraosseous heterotopic location of salivary gland tissue. Bouquot et al had microscopically examined 5,034 maxillofacial bone samples and observed that the frequency of salivary gland tissue was no less than 2.6 of 1,000 biopsied marrow samples.[47] Ectopic salivary glands is known to occur in other areas like the buccinator muscle[48] or gingiva.[49] Another good example of intraoral choristoma is ectopic bone formation, especially common in the tongue. These are well-circumscribed, sessile, or pedunculated growths histologically characterized by the presence of the lamellar bone. Tongue choristomas, first defined in 1913, are frequently seen during the third and fourth decades of life, usually in front of the foramen cecum.[50] Very rarely one may come across choristomas in the oral cavity lined with respiratory mucosa or gastric mucosa[51] or comprising mature glial cells.[52]

Conclusion

Hamartomas and choristomas, although not very common, are known to occur in the oral cavity. Although hamartomas/choristomas are malformations, all malformations are not hamartomas/choristomas. To add to the confusion, the clinical features of hamartomas are similar to those of benign tumors or reactive lesions of the oral cavity and are frequently misdiagnosed. We have, therefore, attempted to make a distinction between these terms and have listed the inclusion and exclusion criteria for labeling a malformation as a hamartoma or a choristoma.

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Conflict of Interests
None declared.

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