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Named cells in cysts associated with orofacial region: Do they aid in diagnosis

Manjeeta Mahesh Sinai Dhume
Lecturer, Department of Oral and Maxillofacial pathology, Goa Dental College and Hospital, Goa University Bambolim, Goa, India

Abstract
The extensive examination of cellular and nuclear structures, as well as their changed presentation in a particular pathology is part of the histological analysis of the tissue. This is done by utilizing a wide range of microscopic methods and staining treatments. The observation of different histopathological entities found in various illnesses is one of the distinctive and fascinating properties. Histopathological cells are frequently a crucial diagnostic tool for determining the underlying disease. Hence the present review speaks about the named cells in cysts of orofacial region and their role in pathogenesis of the same.

Keywords: Named cells, bodies, cysts of orofacial region

Introduction

The 5th edition of classification for odontogenic cysts is not conceptually very different from the previous 2017 classification of odontogenic lesions. This was made available for public in March 2022. The classification of odontogenic cyst was removed in the 3rd edition. But later in 2017 4th edition it was added and continues to be the same in the latest classification. Only one new entity that is non odontogenic; Post-surgical ciliated cyst has been added to 2022 classification. 5th edition also gives the essential and desirable diagnostic criteria' for each entity which aids in diagnosis [1]. Few of the odontogenic cysts contain named cells which may or may not act as diagnostic aids. The present review gives a brief compilation of the same.

Material and methods

A review of old 2017 classification of cysts of the jaws was done along with the new 2022 classification.

All the named cells and bodies were noted and literature review was done of them.

Results

The old 2017 classification and the new 2022 classification have been tabulated. (Table 1) The results are as follows [2]

After reviewing all the cysts only two named cells have been noted –Rushton Bodies and Ghost Cells. These are described as follows:

Table1: 2017 and 2022 classifications of odontogenic cysts

2017 classification		2022 classification
Odontogenic cysts of inflammatory origin	Odontogenic & non-odontogenic developmental cysts	Radicular cyst Inflammatory collateral cysts Post-surgical ciliated cyst Nasopalatine duct cyst
Radicular cyst Inflammatory collateral cysts	Dentigerous cyst	Gingival cyst
	Odontogenic keratocyst	Dentigerous cyst
	Lateral periodontal cyst and botryoid cyst	Orthokeratinized odontogenic cyst
	Gingival cyst	Lateral periodontal cyst and botryoid cyst
	Glandular odontogenic cyst	Calcifying odontogenic cyst
	Calcifying odontogenic cyst	Glandular odontogenic cyst
	Orthokeratinized odontogenic cyst Nasopalatine duct cyst	Odontogenic keratocyst

1. Rushton bodies

Author: First noted by Dewey in 1918. Following this they were mentioned in early literature by Lund in 1924. But were described in detail by Martin A Rushton in1955 [3]. Hence named as Rushton hyaline bodies or Rushton bodies.

Morphology: Eosinophilic bodies within the epithelium of odontogenic cysts. Described by Rushton they as bodies measuring 0.1mm in length. He described 3 morphological patterns as follows [3] and the fourth one proposed by Morgan and Johnson

a. **Hairpin shaped:** Linear, straight or curved into various types
b. Broken up pieces of plate appearance
c. Circular or polycyclic agglomerations, sometimes laminated [4]
d. Morgan and Johnson described the fourth type as an elongated type, lining cleft like spaces, which are probably cholesterol clefts [5]

Pathogenesis

Rushton bodies have drawn the most attention because they don't often go together with other local lesions and seem to be unique to odontogenic cysts [6].

Regarding the source of Rushton bodies, there is some uncertainty. Sir Dewey believed they were caused by the hyaline degeneration of recently established capillaries as early as 1918. Sir Rushton (1955) hypothesized that they might be made of keratin or a material similar to keratin [7,

[8]. Hyaline bodies are comparable to cotton fibers, thus Medak and Weinmann (1960) speculated that they might be leftovers from a previous operation. However, many cysts with hyaline bodies were recorded in individuals who had no prior surgical operations.

Hyaline bodies were reported to share histochemical properties with keratin by Wertheimer *et al*. in 1962, supporting the idea that they are a secretory byproduct of the odontogenic epithelium and are generated similarly to secondary enamel cuticle [7, 9, 10]. Hyaline bodies were thought to be of haematogenous origin by Bouyssou *et al*. in 1965 and Sedano *et al*. in 1968 because they showed histochemical reactivity for hemoglobin. They hypothesized that Rushton bodies originate from the thrombi in the connective tissues varicose venules that had been strangled by growing epithelium. Dent *et al*., (1967) concluded that the histochemical reactions were not specific for haemoglobin [7, 9, 10].

Study conducted by Browne and Matthews (1985) on 14 dental cysts containing intraepithelial hyaline bodies showed that the cores of some circular and polycyclic for the fibrinogen supports that these bodies are produced by cellular reaction to extravasated serum [11].

According to Sakamoto *et al*. (2012), hyaline bodies are amyloids that develop as a result of two distinct biological occurrences: the first is a unique change of epithelial differentiation to produce hair keratin, and the second is hemorrhage to produce hemoglobin. Their findings therefore harmonized the two ideas of keratinous nature vs haematogenous origin, concluding that both substances are necessary for the formation of hyaline bodies [12].

Jensen and Erickson (1974) conducted ultrastructural studies that disproved the hyaline bodies' composition of keratin and any structural resemblance to the secondary enamel cuticle as well as their production by epithelial cells or their haematogenous origin [13].

Hyaline bodies, according to Morgan and Johnson (1974), are a secretory byproduct of odontogenic epithelium that is deposited on the surface of particles like cell fragments or cholesterol crystals in a process similar to the development of dental cuticle on the unerupted regions of enamel surfaces. Neither a keratinous nor a haematogenous origin could be supported by the available evidence [14].

According to some ultrastructural studies, two types of hyaline bodies can be identified that is the lamellated and homogenous types. El-Labban (1979) proposed that the lamellar pattern may result from segregation of components within the mass rather than from incremental expansion and that the granular type comes from degenerating RBCs [15].

Hyaline bodies are made by the epithelium of odontogenic cysts and have direct contact with the outer layer of the neighboring cyst epithelium via its intercellular bridges, as demonstrated by Philippou *et al*. in 1990 [16]. In the year 1980 investigation by Kulkarni *et al*. showed that hyaline bodies responded differently to keratin but were identical to the dental cuticle [17].

Series of various theories have been contemplated regarding the origin of Rushton bodies. However, the origin of these hyaline bodies remains unsolved till date.

Stain: Masson's Trichrome stain, Orcein stain, Mallory aldehyde fuchsin stain, Papanicolaou stain, Gomori stains, Prussian blue stain, Weigert's elastin solution, Rhodamine B stain, Thioflavin T stain, Congo red stain [18].

Immunohistochemical analysis shows positivity for hair keratin, keratin 17 and hemoglobin α-chain [18].

Associated conditions: Radicular cyst, Residual cyst, and Plexiform ameloblastoma (Not Pathognomic)

2. Ghost cells

Definition: It is a dead cell in which the cell outline remains is visible, but whose nucleus and cytoplasmic structures are not stainable [19].

History: Many investigators have made efforts to clarify the nature of ghost cells by employing methods ranging from conventional histochemistry to immunocytochemistry. Many hypothesis have been proposed and debated upon. The description of Ghost cell as dyskeratotic cells which are similar to viable cells but have distinct outline was made by Highman and Ogden as early as 1936 [19].

According to WHO 2004 classification for odontogenic tumors contemplated ghost cells as transient squamous cells at varied stages of differentiation [20].

Pathogenesis: Ghost cells are always epithelial in origin and can originate from any layers of epithelium i.e., basal, intermediate or superficial.

Various theories proposed regarding the origin of these cells enumerated as follows:

1. Local hypoxia and degeneration

Ischemia was taken into account as a possible cause, particularly in odontomas where the disorganized growth of hard tissue was thought to cut off the blood supply of ameloblast-like cells nearby. This notion appears to be supported by the dilated, degraded membranous organelles and the larger appearance of ghost cells, both of which were attributed to intracellular edema [21]. However, the sporadic appearance of ghost cells close to blood vessels disproved this theory.

2. Form of coagulative necrosis

Features of coagulative necrosis are congruent with characteristics of ghost cells, such as the loss of nuclei and the obvious preservation of basic cellular outlines. It was thought that this coagulative necrosis was probably what caused the changed or absence of cytokeratin expression by ghost cells [22].

3. Metaplastic transformation

The odontogenic epithelium inherently does not have the potential to produce keratin. Hence it was thought to originate through metaplasia of odontogenic epithelium with abnormal keratinization [23].

4. Abnormal terminal differentiation or apoptosis

During terminal differentiation, an apoptotic mechanism results in the formation of ghost cells. But these ghost cells were not exactly identical to the normal terminal differentiation of keratinocytes [24].

5. Abortive formation of enamel matrix

A few of researchers have discovered that the ghost cells of odontogenic lesions contain enamel matrix proteins [25].

6. Aberrant keratinization and/or accumulation of hard keratin

According to Regezi *et al*., ghost cells were an abnormal or uncommon form of keratin rather than real keratin. This was stated since ghost cells showed positivity for various special stains used for keratin [26].

7. Mucin induced ghost cell transformation

Based on their observations of nonodontogenic oral diseases including irritant fibroma and oral submucous fibrosis, Sarode *et al*. put up this novel explanation for ghost cell metamorphosis. They have distinguished Totos bodies from individual cell keratinization, spongiotic artifact, and ghost cell-like formations.

More research is required to determine the nature of the changes seen in these non-odontogenic mucosal lesions and to compare them to the histogenesis of ghost cells in odontogenic lesions [27].

Various molecular pathways have been associated with Ghost cell formation. These include Wnt-ß-catenin-T-cell Factor [TCF] and Notch Signaling pathway.

Morphology: Pale eosinophilic squamous cells with shadowing cytoplasmic outline, showing pyknotic nuclei or central clear areas indicative of the site previously occupied by the nucleus.

Diagnostic stains

a. Haematoxylin and eosin: Pale Pink
b. Goldner stain: Pale red
c. Masson trichrome stain: Light red
d. Rhodamine stain: Yellow fluorescence
e. Mallory's aniline blue stain: Dull orange brown to red

Associated conditions: Eruption cyst, Glandular odontogenic cyst, Calcifying epithelial odontogenic cyst

It is also seen in some odontogenic tumors, Non-odontogenic conditions and others.

The behavior of neoplasms is caused by the transition of epithelial cells into more resilient terminally differentiated apoptotic cells, or "Ghost cells," which also aid in reducing the stress of the developing neoplasm. This data suggests that they reflect the behavior of that lesion rather than having an impact on the prognosis.

Discussion

The histogenesis and pathogenesis of the Rushton bodies and Ghost cells still remain questionable. Even though modern world has introduction of various molecular and genetic analysis techniques easily accessible, the exact role of these cells in pathogenesis of various pathological conditions is questionable.

Conclusion

Histopathological bodies are crucial for the diagnosis of diseases in oral pathology. These traits, some of which are pathognomonic, frequently serve as symptoms of the disease's etiology. Having knowledge about them helps in understanding the nature of the disease and its progression over time.

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