International Journal of Research in Medical Sciences
Dhume MMS et al. Int J Res Med Sci. 2023 Dec;11(12):4601-4606
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012

DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20233742

Review Article

Named cells and inclusion bodies in bacterial and viral infections associated with oral cavity

Manjeeta M. S. Dhume[1]*, Clarence P. Dias[2]

[1]Department of Oral and Maxillofacial Pathology, Goa Dental College and Hospital, Goa University, Bambolim, Goa, India
[2]Department of Periodontics, Goa Dental College and Hospital, Goa University, Bambolim, Goa, India

Received: 05 October 2023
Accepted: 06 November 2023

***Correspondence:**
Dr. Manjeeta M. S. Dhume,
E-mail: dhumemanjeeta.md@yahoo.com

ABSTRACT

An oral pathologist can explore the world of intricate features in complicated tissues thanks to this visual speciality. Because our eyes are more accustomed to seeing normal morphology in cells and structures, we can become stuck in a state of flux when we encounter specific ill cells or bodies. Although they may appear to be deceiving, they aid the pathologist in making a diagnosis because they are pathognomonic for numerous diseases and conditions. With a focus on pathogenesis, microscopic features, and stains used to highlight features of the same, the current article is an attempt to compile various histopathological bodies seen in various diseases associated with bacterial and viral diseases associated with the oral cavity.

Keywords: Named cells, Inclusion bodies, Bacterial, Viral

INTRODUCTION

The complementary DNA that develops from the translation of a messenger RNA when a foreign gene (the infectious agent) is introduced into a cell may code for a protein that does not undergo further modification and transport, causing precipitation in the cell and the formation of an inclusion body. In certain diseases or pathological conditions, cells can change, and these altered cells may end up being pathognomonic for that specific disease. Named cells are tittled based upon the scientists who first identified and characterized them. These cells exhibit specific staining reactions and have a characteristic morphology. Present articles tires to give a complied data regarding different types of cells and bodies associated with bacterial and viral infections affecting the oral cavity.

BACTERIAL INFECTIONS

Bacteria are found everywhere. They are crucial to preserving the ecosystem in which we live. The majority of microorganisms in the world do not cause illness or infection. The public's health is significantly impacted by these bacterial diseases. Since there is a larger selection of antimicrobial medicines with effectiveness against bacteria, bacterial illnesses are typically easier to treat than viral infections. But unlike infectious diseases brought on by viruses and parasites, bacterial resistance to antibiotics is a rapidly expanding issue that could have catastrophic effects.[1]

The various types of named cells and bodies seen in bacterial infections are as follows.

Giant cells

Definition

A giant cell can be described as an unusually large, huge or gigantic cell; as a large multinucleated often phagocytic cell, cell with more than one nucleus, a multinucleated mass of cytoplasm that is not separated into cells. Bacterial

infections associated with giant cell formation include- tuberculosis, leprosy, syphilis and actinomycosis.

In relation to bacterial infections the aggregates of inflammatory cells along with other epithelial cells forms a granuloma. Granulomatous inflammation is a distinctive pattern of chronic inflammatory reaction characterized by focal accumulation of activated macrophages, which often develop an epitheloid appearance. A granuloma is a focus of chronic inflammation consisting of a microscopic aggregation of macrophages that are transformed into epithelium like cells surrounded by a collar of mononuclear leukocytes, principally lymphocytes, and plasma cells.[2] A complex interplay is seen between invading organisms or prolonged antigenemia, macrophage activity, a Th1 cell response, B cell overactivity, and a vast array of biological mediators.[3]

Epitheloid cells have a pale pink cytoplasm and blurry cell borders in hematoxylin and eosin sections, and they appear to be fusing together. These cells can combine to create enormous cells that have a diameter of 40–50 m. The Langhans type of these enormous cells has a vast mass of cytoplasm with 20 or more nuclei dispersed randomly or in a horseshoe pattern near the periphery.[4] These enormous cells can be created through nuclear division and cell fusion without cytoplasmic segregation.[5] The nuclei of Langhans giant cells are either concentrated at the giant cell's two poles or organized in a horseshoe configuration at its perimeter.[6]

The exact etiologic agent for granulomatous disease must be determined through additional research, including special stains (for tuberculosis, for example), culture procedures (for fungi, for example), molecular techniques (for syphilis, for example), and serologic tests.[2]

Mycobacterium tuberculosis (*M. tuberculosis*) and *M. bovis*, an acid- and alcohol-fast bacteria, are the causative agents behind tuberculosis.[7] Epitheloid granuloma with Langhans large cells are a hallmark of histopathology.[8] A type IV reaction is brought on by the infection, activated macrophages, interferon (IFN) cytokine, and T cell activity. The tuberculous granuloma experiences central caseation necrosis as a result of this response and ischemia.[9] Using Ziehl-Neelsen staining or immunofluorescence with auramine-rhodamine, the mycobacteria can be demonstrated. Other diagnostic procedures include mycobacterial culture and PCR-based mycobacterial DNA detection.

Mycobacterium leprae, which results in granulomatous illness, is the cause of leprosy. A granulomatous inflammatory response can be observed under a microscope along with the formation of granulomas and the presence of Langhans large cells.[10] A positive lepromin test and Fite stain can be used to show the presence of microorganisms.[11]

Treponema pallidum, a spirochete, is the source of the sexually transmitted infection known as syphilis, which can be identified by silver staining or immunofluorescence on dark backgrounds. The treponema pallidum hemagglutination assay (TPHA), the venereal disease research laboratory (VDRL), and fluorescent antibody tests are other diagnostic procedures. In syphilis, lymphocytes and plasma cells completely round the granulomas.[12]

Actinomycosis is a long-lasting bacterial illness that resembles fungal infections both clinically and microscopically. Actinomyces israelii, an anaerobic, gram positive bacterium, is the cause. This disease manifests as a granulomatous reaction with central abscess formation. Yellow granules known as "sulfur granules" that are aggregates of the causing bacteria can be found in the pus draining from the lesion. Using Gomori's methanamine silver stain to identify the organism, microscopic examination of tissue sections, and culture testing can provide a definitive diagnosis.[10]

Virchow/Lepra cell

The intracellular pathogen *Mycobacterium leprae* is the cause of leprosy, a chronic infectious disease that affects the skin and peripheral nerve system. The most serious symptom experienced by leprosy patients is peripheral nerve damage. When the diagnosis is made too late, nerve dysfunction may become irreversible, resulting in physical abnormalities and persistent disability, which are hallmarks of the condition.[13] A peculiar type of giant cells seen in lepromatous leprosy is known as Virchow/Lepra cell.

Pathogenesis

Histiocytes gradually change into lepra cells, a form of activated macrophages and become foamy because of accumulation of abundant poorly processed mycobacterial lipid material due to failure of phospholipase activity.

Morphology

Vary in size from that of a lymphocyte to several times this diameter. Protoplasm, is filled with vacuoles, and contains enormous numbers of lepra bacilli. Nucleus is usually single and is pressed to one side by the vacuoles and bacilli that crowd the cell body.[14]

Stain

Modified Fite Faraco stain- Lepra cell. Modified Ziehl-Neelsen and fluorescent stain – *Mycobacterium Leprae.*

Asteroid bodies

An asteroid body is a microscopic finding seen within the giant cells of granulomas in diseases such as sarcoidosis

and foreign-body giant cell reactions. It was first discovered by Friedman in 1944.

Pathogenesis

The earliest theory about the pathogenesis has been linked to the structure of the cytosphere which is an ordered central subcellular structure consisting of a pool of centrioles, radiating microtubules, and radially arranged Golgi networks.[15]

Other researchers noticed in the 1990s that there are loosely distributed myelin membranes in the halo of transparent vacuoles surrounding the asteroid body, which may be excessive leftovers of cellular membranes created following the fusing of active histiocytes.[16] But the electron microscopic studies of asteroid bodies may point towards a possible role of autophagy in the formation of asteroid bodies.

Morphology

Measure up to 30 µm in diameter and appear to float in cytoplasmic vacuoles. Eosinophilic structures with a centre that is brown red with blue bent spokes radiating into the cytoplasm evoking images of spiders or open umbrella frames.

Stain used

Haematoxylin and Eosin stain was used.

Associated conditions

Associated conditions are sarcoidosis, foreign-body giant cell reactions, and lymphadenopathies.

Shaumann bodies

The exact nature and histogenesis of the birefringent crystals occuring both within and outside these bodies remain unsettled.

Morphology

Complex concentrically stratified concretions, up to 150 µm in diameter, that often enclose hematoxyphilic mineralized components or birefringent crystalline material. Concretion grows as successive layers of mineral precipitate around a central core. The small ones are usually spherical, compact and basophilic and large ones are seldom preserved intact, appearing empty or fragmented, with laminated polycyclic contours. Crystals are thought to be composed mainly of calcite and is maintained that they are endogenous, invariably constituting the nidus around which the bodies evolved

Stain used

Haematoxylin and Eosin stain was used.

Associated conditions

Associated conditions are sarcoidosis, beryllium disease, tuberculosis, granulomas of Crohn's disease, hypersensitivity pneumonia, histoplasmosis, lymph nodes draining cancer.[17]

VIRAL INFECTIONS

Nucleic acids, proteins, and occasionally lipids and glycans make up the particles that make up viruses. Viral particles vary greatly in terms of their structural, molecular, and genetic structure between isolates. Viral genomes can encode anything from a few genes to several hundred, and their sizes range from a few tens of nanometers to micrometers. Viruses use host cells to replicate, proliferate, and eventually spread from cell to cell and from host to host as obligate intracellular parasites.The named cells and bodies in viral infections helps in identification of various diseases.[18]

Koilocytes

Ernest Ayre first described and demonstrated koilocytes in 1951. Koilo= hollow, cyte=cell

Morphology

Squamous cells, predominantly superficial and intermediate cells,containing a hyperchromatic nucleus, with a large, well-demarcated, clear perinuclear zone surrounded by a dense peripheral cytoplasmic rim, appearing crenated, “raisin-like” or “spoon-like” in shape.[19]

Pathogenesis

The role of cytoplasmic vacuolization in viral replication is unclear. However, it could contribute to the fragility of keratinocytes and release of viral-laden nuclei from human papilloma virus lesions.[20]

By interfering with keratin integrity and cornification layer assembly, the papillomavirus is able to migrate to the epithelium's superficial layers. These alterations are collectively known as the "cytopathic effect."

Stain

Haematoxylin and Eosin stain, immunohistochemical (p16) was used.

Associated conditions

Associated conditions were human papilloma virus infections, squamous papilloma- HPV 6 and HPV 7, verruca vulgaris- HPV 2 and 4 types, focal epithelial hyperplasia/Heck’s disease- 13 or 32 with a site specificity of keratinized and nonkeratinized surfaces respectively, in the superficial layers, condyloma acuminatum- HPV types

6 and 11, verrucous carcinoma- HPV 6, 11, 16, and 18, and oral squamous cell carcinoma- HPV 16, 11, and 27.[20]

Wartin Finkeldey cells

Morphology

Round or lobulated, 25-150 μm in diameter, with abundant acidophilic cytoplasm and 50-100 darkly stained nuclei distributed in grape like clusters in the center of the cell. Relatively small amount of cytoplasm, few organelles and no virus particles.[21]

Pathogenesis

Derived from the follicular dendritic cells of the lymph nodes.

Stain used

Haematoxylin and Eosin stain was used.

Associated condition

Associated conditions include: measles, Kimura disease, systemic lupus erythematosus, and human immunodeficiency virus.[21]

Hendreson-Patterson bodies- intracytoplasmic bodies

Morphology

Eosinophilic intracytoplasmic inclusions, that accumulate and progressively enlarge until they replace the entire cell they occupy.

They are largest at the epidermal surface and compress the keratinocyte nuclei.[22]

Pathogenesis

Accumulation of the virus particles in the cytoplasm of the cell.

Stain

Haematoxylin and Eosin stain was used.

Associated condition

Associated condition was molluscum contagiosum.

Cowdry type A (Lipshutz bodies) and type B inclusion bodies

These intranuclear eosinophilic inclusion bodies are composed of nucleic acid and protein and are seen in cells infected with herpes simplex virus infection (HPV), yellow fever, polio, and adenovirus.

Cowdry type A

Morphology

Round eosinophilic material surrounded by a clear halo. They are acidophilic material of droplet-like. They contain intact and disrupted virions and push darkly stained host cell chromatin to the edges of the nucleus.[23] HSV infections are associated with acantholysis with solitary keratinocytes in the blister cavity. Keratinocytes with nuclear with nuclear changes such as margination of the nuclear chromatin, multinucleation, and nuclear inclusions are commonly seen.[24]

The viral inclusions are small pink deposits with a clear halo seen within the nucleus called Lipschutz bodies. These nuclear inclusions are of an hourglass appearance and eosinophilic.They occur in the stratum spinosum and stratum granulosum appearing as dense ovoid bodies and are commonly surrounded by a clear zone analogous to the "ballooning degeneration" seen in viral diseases affecting skin.[25]

Pathogenesis

Infected cells shows ballooning or enlargement of nucleoli as the earliest manifestation, and it eventually undergoes disaggregation and fragmentation.[26] The other structural changes that are induced by the virus in the host cell include chromosomal margination, duplication and folding of the intracellular membrane, fragmentation of Golgi stacks, insertion of viral proteins into cellular membranes, polykaryocytosis and rearrangement of the microtubular network.[27]

Associated conditions

Associated conditions were herpes simplex virus infection, and yellow fever.

Lipshutz bodies

Morphology

Eosinophilic nuclear inclusions having enlarged nuclei and surrounded by a clear halo with an enlarged nuclei.

Associated conditions

Associated conditions were varicella and herpes simplex.[24]

Cowdry type- B

Morphology

Intranuclear eosinophilic amorphous bodies. They are surrounded by a clear halo without other nuclear changes during early stages of development of the inclusion.

Stains

Giemsa, hematoxylin and eosin, Papanicolaou, Seller, Mallory aldehyde fuchsin, periodic acid Schiff, Gomori, and Orcein stains.[27]

Associated condition

Associated condition was neural cells.

Owl eye inclusions

Hodgkin's lymphomas and cytomegalo virus disease show sizeable intranuclear inclusion bodies known as 'Owl's eye' inclusion bodies

Morphology

The inclusion bodies stain dark pink and are called "owl's eye" inclusion bodies haematoxylin and eosin stained sections. Affected cells are strikingly enlarged, often to a diameter of 40 um in size, and show cellular and nuclear polymorphism.

The intranuclear inclusions in CMV-infected cells are large and deep purple. These nuclear inclusions involve half of the nuclear diameter and are usually set off the nuclear membrane by a clear halo. There is usually some clearing around the inclusion, and the chromatin tends to condense around the periphery of the nucleus.[28]

Pathogenesis

CMV is a lytic virus that both in vitro and in vivo has a cytopathogenic effect. The viral double-stranded DNA genome is created in the nucleus of the host cell, specifically in compartments designed for viral replication. This DNA alters the nuclear and cytological structure, generates aberrant proteins, and replicates more quickly.[29]

Table 1: Conditions with associated cells and bodies.

Named cells/bodies	Disease	Diagnostic significance
Lepra or Virchow cell	Leprosy	Pathognomic
Asteroid bodies	Sarcoidosis	Not pathognomic
Shaumann bodies	Numerous lesions	Not pathognomic
Koilocytes	HPV infection	Pathognomic
Warthin Finkeldey cells	Measles	Not pathognomic
Henderson Patterson bodies	Molluscum contagiosum	Pathognomic
Owl eye inclusions	CMV infections	Pathognomic

Stains used

Periodic acid Schiff stain, H & E, Gomori's stain, Giemsa, Papanicolaou, Sellers stains were used.

Associated conditions

Associated conditions were cytomegalovirus (CMV) or human herpesvirus 5 (HHV-5) infections are frequently associated with salivary glands though they may be found throughout the body. Conditions in which cells and bodies are pathognomic and not pathogonomic are enlisted as follows (Table 1).

CONCLUSION

The body undergoes a number of metabolic changes that lead to cellular abnormalities as the illness progresses. These cellular changes might be seen at different stages in the progression of a disease. Just as the existence of these bodies shows the presence of a disease, the lack of them suggests that the disease is waning. Based on the presence of these histological entities at various stages of the disease's evolution, it may be useful to stage the disorders.

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.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Doron S, Gorbach SL. Bacterial Infections: Overview. International Encyclopedia of Public Health. 2008;273-82.
2. Kumar V, Abbas AK, Fausto N. 7th edition. Saunders: Pennsylvania. Pathologic Basis of Disease. 2006.
3. James DG. A clinicopathological classification of granulomatous disorders. Postgrad Med J. 2000;76:457-65.
4. Soler P, Bernaudin JF. Physiology of granulomas. Rev Pneumol Clin. 1993;49:257-61.
5. Macfarlane RS, Reid R, Collander R. Pathology illustrated. 5th edition. London: Churchill Livingstone. 2000.
6. Mohan H. Textbook of pathology. 5th edition. Anshan: Jaypee India. 2005.
7. Macfarlane RS, Reid R, Collander R. Pathology illustrated. 5th edition. London: Churchill Livingstone. 2000.
8. Cawson RA, Odell EW. 7th edition. Cawson's essentials of oral pathology and oral medicine. London: Churchill Livingstone. 2002.

9. Macfarlane RS, Reid R, Collander R. Pathology illustrated. 5th edition. London: Churchill Livingstone. 2000.
10. Regezi JA, Sciubba JJ, Jordon RC. Oral pathology, clinical pathologic correlations. 4th edition. Missouri: Saunders. 2003.
11. Greenberg MS, Glick M. Burket's oral medicine, diagnosis and treatment. 10th edition. Canada: BC Decker. 2003.
12. Maize JC, Walter HC, Burgdorf MD, Hurt MA, LeBoit PE, Metcalf JS, et al. Cutaneous pathology. 1st edition. Pennsylvania: Churchill Livingstone. 1998.
13. de Macedo CS, Lara FA, Pinheiro RO, Schmitz V, de Berrêdo-Pinho M, Pereira GM, et al. New insights into the pathogenesis of leprosy: contribution of subversion of host cell metabolism to bacterial persistence, disease progression, and transmission. F1000Res. 2020;9:F1000.
14. Sugawara-Mikami M, Tanigawa K, Kawashima A, Kiriya M, Nakamura Y, Fujiwara Y, et al. Pathogenicity and virulence of Mycobacterium leprae. Virulence. 2022;13(1):1985-2011.
15. Cain H, Kraus B. Immunofluorescence microscopic demonstration of vimentin filaments in asteroid bodies of sarcoidosis. A comparison with electron microscopic findings. Virchows Arch B Cell Pathol Incl Mol Pathol. 1983;42(2):213-26.
16. Papadimitriou JC, Drachenberg CB. Ultrastructural analysis of asteroid bodies: evidence for membrane lipid bilayer nature of components. Ultrastruct Pathol. 1992;16(4):413-21.
17. Gupta N, Rajwanshi A, Gupta D. Schaumann body in a case of sarcoidosis diagnosed on transbronchial Erratum in: J Cytol. 2011;28(4):184.
18. Lozach PY. Cell Biology of Viral Infections. Cells. 2020;9(11):2431.
19. Singh P, Sowmya SV, Rao RS, Augustine D, Haragannavar VC, Nambiar S. Koilocytes in Oral Pathologies. World J Dent. 2018;9(2):149-53.
20. Bharti AH, Chotaliya K, Marfatia YS. An update on oral human papillomavirus infection. Indian J Sex Transm Dis. 2013;34(2):77-82.
21. Madakshira MG, Bajaj R, Kaur K. Warthin-Finkeldy cells - A soft indicator in cytodiagnosis of Kimura. J Cytol. 2017;34(3):154-5.
22. Rao K, Priya N, Umadevi H, Smitha T. Molluscum contagiosum. J Oral Maxillofac Pathol. 2013;17(1):146-7.
23. Eyons AS, Kaslow RA. Viral Infections of Humans: Epidemiology and Control. Br J Biomed Sci. 1998;1029;813.
24. Antonio C, Slootweg PJ. Pathology of the head and neck. Vesiculobullous Diseases. 1999;72.
25. Kulkarni M, Agrawal T, Dhas V. Histopathologic bodies: An insight. J Int Clin Dent Res Org. 2011;3(1):43.
26. Patil S, Rao RS, Sharath S. Named Cells and Bodies in Oral Pathology-Part I: A Ready Reckoner. Int J Clin Dent Sci. 2013;4.
27. Patankar V. Inclusion bodies in infectious diseases - a narrative review. IJCSPUB. 2022;12:3.
28. Naziya NJ, Jayanthi P, Harish RK, Rathy R, Sunil S. Histopathological Bodies in Oral Pathology. Oral Maxillofac Pathol J. 2017;8(2):114-7.
29. Gupta M, Shorman M. Cytomegalovirus. In: StatPearls. Treasure Island (FL): StatPearls Publishing. 2021.

Cite this article as: Dhume MMS, Dias CP. Named cells and inclusion bodies in bacterial and viral infections associated with oral cavity. Int J Res Med Sci 2023;11:4601-6.