REVIEW

Named Cells and Bodies in Oral Pathology - Part II: A Ready Reckoner

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Abstract

It is important for the Oral pathologist to be familiar with the different pathological cells and bodies as they very often tend to appear in various diseases and conditions. In spite of most of these inclusions not being pathognomic, they inevitably help the pathologist in making a diagnosis. Besides assisting in recognition of a lesion, this article will also be of immense help to the student population.
This article is split into two parts for the ease of the reader. Part I provided an overview of various pathological cells and bodies pertaining to diseases, metabolic disorders and neoplasms. Part II will brief about those associated with cysts, tumors and infections.

Keywords: Inclusion bodies, cells, disease, disorder

Introduction

Inclusion bodies are dense particles of aggregated protein. They may be formed due to viral and bacterial infections, genetic disorders and a number of other reasons. Studies on the structure and morphology of these bodies formed from various proteins have revealed that these aggregates can exhibit amorphous as well as partly ordered structure. The nature of the aggregating protein, predisposing conditions for aggregate formation and the site of inclusion body formation contribute to their structural heterogeneity. The normal cells, in certain conditions, are modified, to become pathological, which may be pathognomic thus giving a clue to diagnosis[1].

Cysts and Tumors:

Rushton bodies:

Morphology: Rushton bodies appear as eosinophilic, linear, straight or curved or hairpin shaped, circular or polycyclic forms, often with a granular core and sometimes concentrically laminated. They are found almost always within the epithelial lining and only rarely in the fibrous capsule and are often calcified[2].
Pathogenesis: It is known that hyaline bodies result from the entrapment of blood vessels by the epithelium and consequent vessel thrombosis.
Another hypothesis is that cell debris or cholesterol crystals present in the cyst might stimulate epithelial cells to secrete amorphous substances that later undergo to calcification, originating Rushton bodies.
It is also suggested that these structures are formed by apoptosis of epithelial cells accompanied by intracellular dystrophic calcification[3].

Stain: Hematoxylin & Eosin(H&E), Orcein, Mallory aldehyde fuschin, Papanicolaou(PAP) and Gomori.

Disease or disorder: Dentigerous cyst, radicular cyst

Touton type Giant cell:

Morphology: These cells have a peripheral wreath like arrangement of nuclei with amphophilic cytoplasm, centrally and pale foamy cytoplasm at the periphery of the cell outside the nucleus.(Fig.1)

Pathogenesis: Fusion of many macrophages to form multinucleate giant cells.

Stain: H&E

Disease or disorder: Fibrous histiocytoma

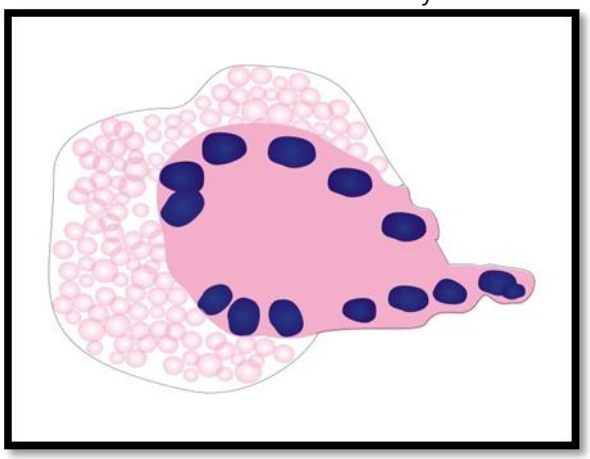

Fig.1 Touton giant cell

Ghost cells:

Morphology: Ghost cells are large pale anucleate cells with homogeneous pale eosinophilic cytoplasm and very pale to clear central areas instead of a basophilic nucleus[4].
They are characterized preservation of basic cellular outlines, which may be blurred at times resulting in groups of ghost cells appearing fused. Dystrophic calcification may occur in some of the ghost cells, initially seen as fine basophilic granules and later as small spherical bodies[5].

Pathogenesis: Ghost cells could represent normal or abnormal keratinization, squamous metaplasia with subsequent calcification caused by ischemia, could be derived from the metaplastic transformation of odontogenic epithelium or may be the product of abortive enamel matrix in odontogenic epithelium[6].

Stain: H & E
Disease or disorder*:* Odontoma, Craniopharyngeoma, Ameloblastic fibroodontoma, Calcifying cystic odontogenic tumor.

Pale and Dark cells:

Morphology*:* Odontogenic myxoma is made up of loosely arranged , spindle shaped and stellate cells , many of which have long fibrillar processes that tend to intermesh.
Ultrastructurally,White and his associates described '**pale**' cells which probably represented active secretory cells, and, the '**dark'** cell, contained collagen fibrils, and this suggested a disturbance in the secretory process of collagen molecules so that they become crystallised into fibrils intracellularly[7].
Disease or Disorder: Odontogenic myxoma

Birbeck Granules

Morphology*:* Birbeck granules are highly characteristic rod shaped structures, with local distensions, of variable length with an internal periodically striated lamella corresponding to langerin[8].
Pathogenesis: They are formed as a result of Langerin('C' type lectin) endocytosis and are accumulations of langerin in tubular endosomes, thus, representing endocytic structures that are formed during receptor mediated endocytosis [8,9].
Stain: Birbeck granules are ultramicroscopic structures and are recognised by monoclonal antibody anti-Lag[10].
Disease or disorder: Langerhan's Histiocytosis

<u>Bacterial infections:</u>

Lepra or Virchow cell:

Morphology: They vary in size from that of a lymphocyte to several times this diameter. Their protoplasm, is filled with vacuoles, and contains enormous numbers of lepra bacilli. The nucleus is usually single and is pressed to one side by the vacuoles and bacilli that crowd the cell body[11]. In developing lesions , the intracellular bacilli are arranged in small bundles; in advanced lesions , dense masses of bacilli called *globi* may replace nearly the entire cytoplasm of the macrophage[12].
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[13].
Stain: Modified Fite Faraco stain[14]
Disease or disorder: Leprosy

Asteroid bodies:

History*:* Discovered by Friedman in 1944.
Morphology: Measuring up to 30 µm in diameter and apparently floating in cytoplasmic vacuoles, they are eosinophilic structures with a centre that is brown red and blue bent spokes radiate into the cytoplasm,

evoking images of spiders or open umbrella frames[15,16] (Fig.2)

Pathogenesis: The prevailing specific environment of the underlying granulomatous disease, together with the internal characteristics of the structure and function of the giant cells, in particular in states of exhaustion may play a part in their development[17].

Stain: H & E

Disease or disorder: Sarcoidosis, Sporotrichosis

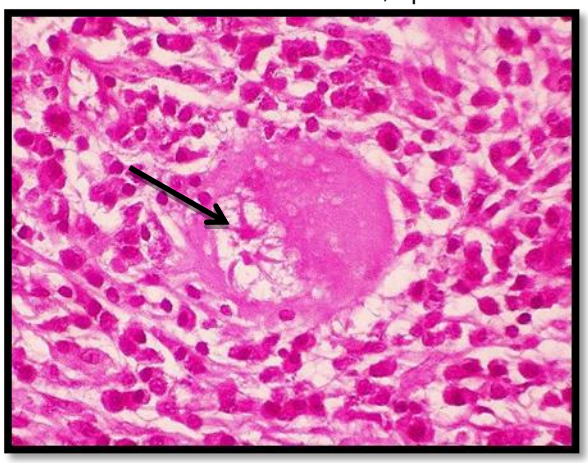

Fig.2 Asteroid Bodies

Aschoff bodies:

Morphology: The Aschoff Nodule is round or oval shaped, consists of a variety of cells arranged more or less in several parallel rows:
Cardiac histocyte (also called Anitschkow cell or myocyte, Aschoff cell, and myocardial reticulocyte) few lymphocytes, occasional polymorphonuclear leukocytes and rarely mast cells.Foci of fibrinoid degeneration or necrosis or both are present between and adjacent to the cells[18,19]. (Fig.3)

Pathogenesis: Aschoff body generally considered to be a granuloma, results from injury to the collagen fibres.

Stain: H&E

Disease or disorder: Rheumatic fever

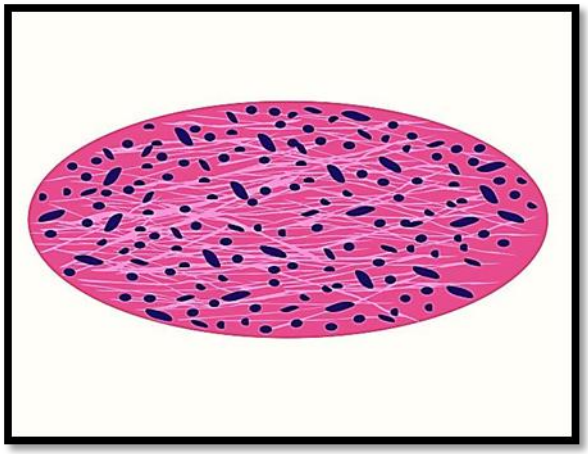

Fig.3 Aschoff body

Donovan bodies:

Morphology: Donovan bodies appear as clusters of blue or black,oval, staining organism with a basophilic nucleus and rod shape or safety pin appearance in the cytoplasm of large mononuclear cells[16,20].

Pathogenesis: Intracellular gram negative organisms found within histiocytes[21].

Stain: Wright, Giemsa

Disease or disorder: Lieshmaniasis

Mallory bodies:

History: First described by Mallory in 1911.

Morphology: Mallory bodies are filaments of intermediate diameter that contain intermediate filament components (e.g., cytokeratins) observable by conventional light microscopy or immunohistochemical methods[22]. (Fig.4)

Pathogenesis: Hyperphosphorylation of cytokeratin 8 and 18 (Stumptner et al., 2000) or ubiquitination of cytokeratin proteins .(Yuan et al.,1996)[23].

Stain: H&E

Disease or Disorder: Liver diseases

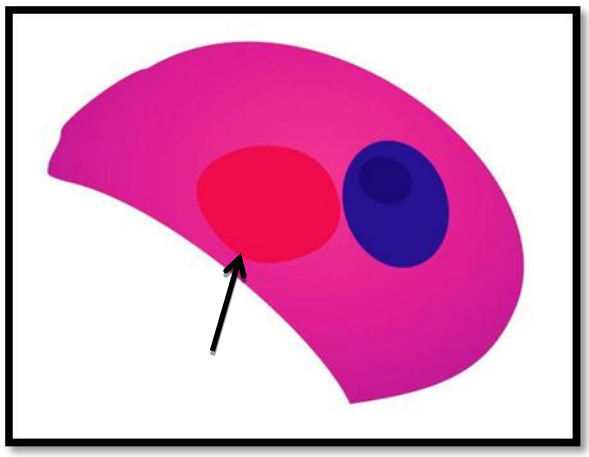

Fig.4 Mallory body

Giant cells:

Giant cells are large multinucleated cells of monocyte/macrophage lineage. Foreign body giant cells, Langhan's giant cells,Touton giant cells, tumour giant cells, are various types of giant cells apart from the miscellaneous types of giant cells such as Aschoff cells of rheumatic nodule and Reed Sternberg cells of Hodgkin's lymphoma[24].

Morphology: There are many types of giant cells. Giant cells are basically large cells with multiple nuclei ranging form 20-100.(Fig.5)

Pathogenesis: Macrophages are present in all tissues and can fuse with other macrophages to differentiate

into multinucleate osteoclasts (in bone) or giant cells (in multiple tissues). Multinucleation is an essential step in the differentiation of osteoclasts, as mononucleated macrophages cannot resorb bone efficiently, and may also be essential in the differentiation of giant cells, which form in tissues in response to foreign particles.

Stain: H & E

Diseases or disorders: Chronic inflammatory lesions

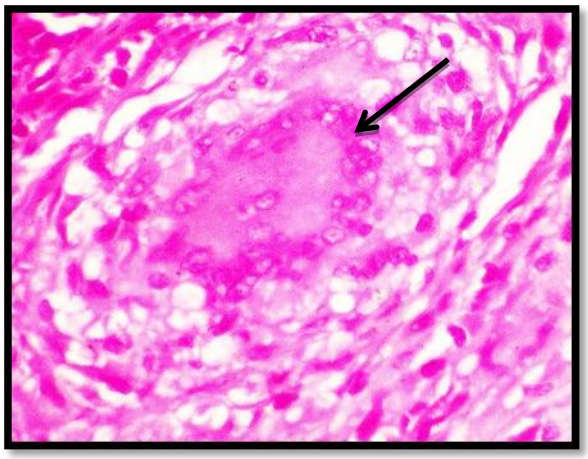

Fig. 5 Giant cells

Shaumann Bodies:

Morphology: Schaumann bodies (SBs) are complex concentrically stratified concretions, up to 150 µm in diameter, that often enclose hematoxyphilic mineralized components or birefringent crystalline material. They occur in multinucleated giant cells (MGCs) but not in epithelioid cells. The small ones are usually spherical, compact and basophilic and large ones are seldom preserved intact, appearing empty or fragmented, with laminated polycyclic contours[25].(Fig.6)

Pathogenesis: The exact nature and histogenesis of the birefringent crystals occuring both within and outside these bodies remain unsettled (Engle, 195 1; Vortel, 1962; Zak, 1964). The 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[25].

Stain: H & E

Disease or disorder: Sarcoidosis, beryllium disease, tuberculosis (seldom seen), granulomas of Crohn's disease, hypersensitivity pneumonia, histoplasmosis, and lymph nodes draining cancer.

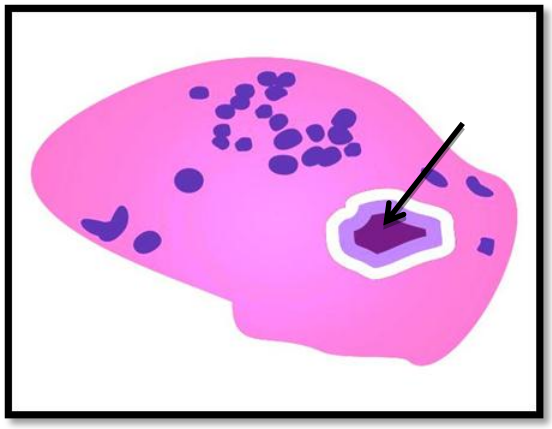

Fig.6 Shaumann body

Viral Infections:

Koilocytes:

History: Ernest Ayre, described and demonstrated koilocytes in 1951[26].

Morphology: Koilocytes are 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. One of the most distinctive features is binucleation and multinucleation(multilobation of a single nucleus[27,28].(Fig.7)

Pathogenesis: In histologic sections they are thought to represent a cytopathic effect of HPV.

Stain: H&E

Disease or disorder: Squamous papilloma, Verruca vulgaris

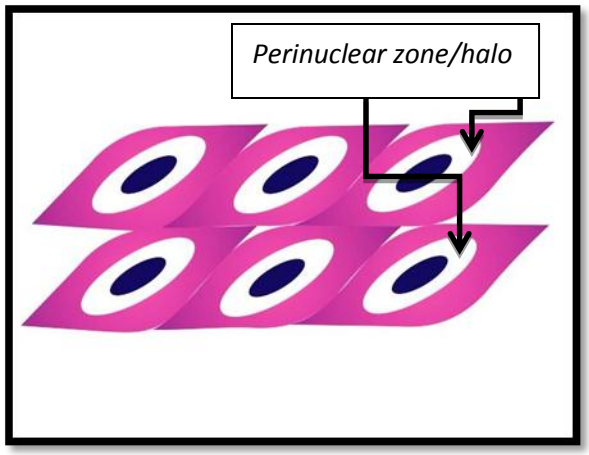

Fig.7 Koilocytes

Warthin Finkeldey cells:

Morphology: These polykaryocytes which are large syncytia are 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. They account for a relatively small amount of cytoplasm, few organelles and no virus particles[29]. (Fig.8)

Pathogenesis: A study of these giant cells in HIV infection in which electron microscopy, immunohistochemistry and in situ hybridisation were used concluded that the multinucleated cells are derived from the follicular dendritic cells of the lymph nodes.

Stain: H & E

Disease or disorder: Measles

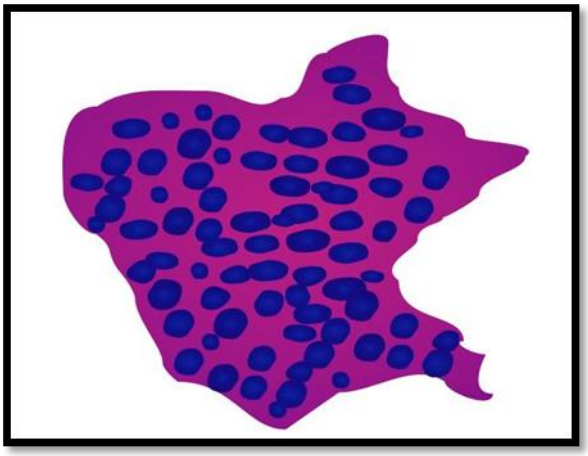

Fig.8 Warthin Finkeldey cells

Intracytoplasmic Bodies:

Henderson-Patterson bodies:

Morphology: Henderson-Patterson bodies are eosinophilic intracytoplasmic inclusions, that accumulate and progressively enlarge until they replace the entire cell they occupy[30].

They are largest at the epidermal surface and compress the keratinocyte nuclei[31,32](Fig.9)

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

Stain: H & E

Disease or disorder: Molluscum Contagiosum

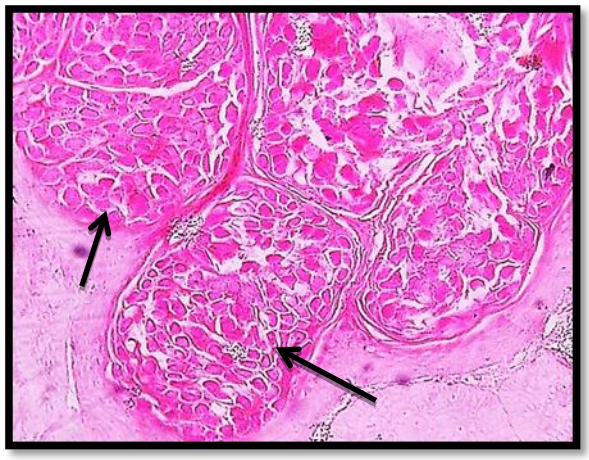

Fig.9 Henderson-Patterson bodies

Councilman bodies:

History: Councilman, an American pathologist, discovered Councilman Bodies[33].

Morphology: Councilman bodies are small, hyaline, round or oval eosinophilic inclusions,composed of densely packed organelles,fat vacuoles and ceroid pigments, in the cytoplasm of hepatic cells; in yellow fever they are believed to represent necrosis around viral particles[33,34,35,36].

Pathogenesis: It is thought that they are derived from condensed and irreversibly damaged liver cells in spite of seemingly preserved organelles[37].

Stain: H & E

Disease: Yellow fever, Councilman bodies can also be seen in other forms of toxic or viral hepatitis and more rarely in bacterial or parasitic infections.

Gaurnieri bodies:

Morphology: Gaurneiri bodies can be seen in the cytoplasm of cells infected with small pox virus. These are aggregates of virions that have developed in the cytoplasm[38]. In cells stained with eosin, they appear as pink blobs in the cytoplasm of affected epithelial cells[39].

Pathogenesis: Aggregation of virions in the cytoplasm.

Stain: H & E, Giemsa, Feulgen, Gispen's modified silver stain[39]

Disease or disorder: Variola, Vaccinia, human cowpox, parapox[16].

Negri bodies (Refer part 1- Neural Diseases)

Intranuclear Bodies

Owl Eye inclusions:
Morphology: These intranuclear inclusion bodies are eosinophilic, surrounded by a clear halo, within a considerably enlarged(megalic) cell, but there is usually only one nucleus[40].(Fig.10)
Pathogenesis: Cytopathic effect of the virus.
Stain: H & E
Disease or disorder: Cytomegalo virus (CMV) infection.

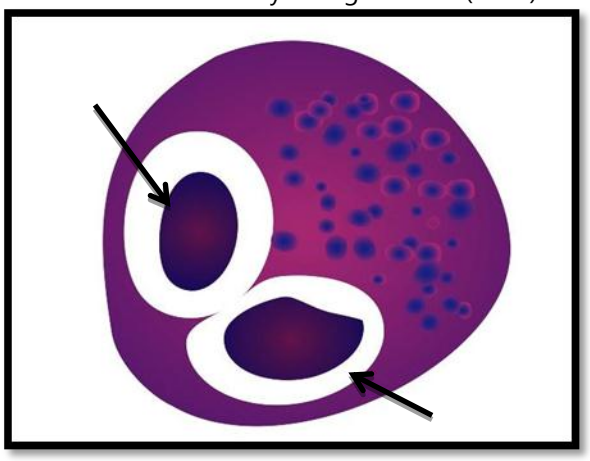

Fig 10.Owl Eye Inclusion

Cowdry Type A and Type B bodies:

Morphology: There are two types of Cowdry inclusion bodies:

1) Type A inclusions(Lipshutz Bodies) are represented by single homogenous eosinophilic bodies occupying most of the central areas of the nucleus and clearly separated from the marginated chromatin
2) Type B inclusions are formed by condensation of basophilic material, including nuclear chromatin, into a single central mass or into multiple discrete bodies[41].

Pathogenesis: Aggregation of virus particles
Stain: H & E, PAP
Disease or disorder: Type A inclusions – Herpes virus Type B inclusions - Poliomyelitis, Borna disease and Rift Valley fever.

Mycotic infections:
Asteroid Bodies (Also seen in Sarcoidosis. Refer bacterial infections)
Disease or disorder: Sporotrichosis

SUMMARY

Disease/Infection/Disorder	*Cell/Inclusion body*	*Stain/Marker*	*Diagnostic Significance*
DISEASES OF BLOOD			
Hemolytic anemias, Pernicious anemia, Hereditary Spherocytosis, hyposplenism	Howell-Jolly bodies	Wright, Feulgan	Not pathognomic
Sideroblastic anemia, haemolytic anemia, hyposplenism	Pappenheimer bodies	Wright, Prussian blue	Not pathognomic
G6PD deficiency	Heinz bodies	Brilliant cresyl blue, Crystal violet	Pathognomic
Disturbed erythropoiesis, Lead poisoning, β Thalessemia, Megaloblastic anemia	Basophilic stippling	Wright	Not pathognomic
Lead poisoning, Pernicious anemia,	Cabot's rings	Romanovsky	Not pathognomic

β Thalessemia	Fessas bodies	Methyl Violet	Pathognomic
Severe burns, infections, Chediak Higashi syndrome	Dohle bodies	Romanovsky	Not pathognomic
Infectious mononucleosis	Downey cells	Romanovsky	Pathognomic
Sickle cell anemia, Iron deficiency anemia, Megaloblastic anemia	Anitschow cells	H&E	Not pathognomic
Mott cell	Reactive plasmacytosis, Burkitt's lymphoma, Large B-cell lymphoma, lymphoplasmablastic lymphoma, multiple myeloma, and syndromic conditions like Wiskott - Aldrich syndrome and von Recklinghausen's neurofibromatosis.	H&E, PAS, MGG, B-220, CD-5, CD-43, CD11b	Not pathognomic
Multiple myeloma	Russell bodies	H&E, PAS, Gram, Millon reaction, Phloxine-tartrazine	Pathognomic
DISEASES OF SKIN			
Lichen planus	Civatte or colloid bodies	H&E, PAS	Not pathognomic
Darier's disease	Corps ronds and grains	H&E	Not pathognomic
Pemphigus group of diseases	Tzanck cells	H&E, Giemsa, PAP, Wright, Methylene blue, Toluidene blue	Pathognomic
Granular cell tumor	Pustulo-ovoid bodies	PAS, CD 68	Pathognomic
Buffy coat of peripheral blood (test for SLE)	LE cell	Romanosky	Pathognomic
Systemic Lupus Erythematosis	LE body	Wright, Giemsa	Pathognomic
NEURAL DISEASES			
Neurilemmoma/Schwannoma	Verocay bodies	H&E	Pathognomic

Rabies	Negri bodies	H&E	Pathognomic
BACTERIAL INFECTIONS			
Leprosy	Lepra or Virchow cell	Modified Fite Faraco stain	Pathognomic
Sarcoidosis	Asteroid bodies	H&E	Not Pathognomic
Rheumatic fever	Aschoff bodies	H&E	Pathognomic
Leishmaniasis	Donovan bodies	Wright, Giemsa	Pathognomic
Liver diseases	Mallory bodies	H&E	Not Pathognomic
Inflammatory diseases(TB, Sarcoidosis, Actinomycosis)	Giant cells	H&E	Not Pathognomic
Sarcoidosis, tuberculosis, beryllium disease,Crohn's disease, hypersensitivity pneumonia, histoplasmosis, and lymph nodes draining cancer.	Shaumann bodies	H&E	Not Pathognomic
VIRAL INFECTIONS			
Squamous papilloma	Koilocytes	H&E	Pathognomic
Measles	Warthin Finkeldey cells	H&E	Pathognomic
	Intracytoplasmic Bodies:		
Molluscum contagiosum	Henderson Patterson bodies	H&E	Pathognomic
Yellow fever,Hepatitis	Councilman bodies	H&E	Pathognomic
Variola, Vaccinia, Human Cowpox, Parapox	Gaurnieri bodies	H&E,Giemsa,Fuelgan, Gispens modified silver stain	Pathognomic
Rabies	Negri bodies	H&E	Pathognomic

Intranuclear bodies:			
CMV infections	Owl eye inclusions	H&E	Pathognomic
Herpes simplex	Cowdry type A(Lipshutz)	H&E	Pathognomic
Poliomyelitis, Rift valley fever, Borna fever	Cowdry type B	H&E	Not Pathognomic
MYCOTIC INFECTIONS			
Sporotrichosis	Asteroid Bodies	H&E	Not Pathognomic
METABOLIC AND STORAGE DISORDERS			
Niemann Pick disease	Niemann pick cells	Giemsa, Wright, PAS, Sudan black B, Oil red O, CD 68	Pathognomic
Gaucher's disease	Gaucher cells	Wright, PAS, iron, CD68, CD14	Pathognomic
Hurler syndrome	Alder-Reilly bodies	Toluidene blue	Pathognomic
Hurler syndrome	Gargoyle cells	H&E, Toluidene blue, Alcian blue	Pathognomic
NEOPLASTIC DISEASES			
Multiple Myeloma	Russell bodies	H&E, PAS, Gram, Millon reaction, Phloxine-tartrazine.	Pathognomic
Hodgkins Lymphoma	Reed-Sternberg cells(Pop corn cells, Lacunar cells)	H&E, CD15, CD20, CD 30	Pathognomic
Verrucous Xanthoma	Foam cells	H&E, PAS, Scharlach R, Sudan III, CD 68, vimentin, cathepsin B	Pathognomic
Rhadomyosarcoma	Racquet cells	H&E	Not Pathognomic

Granular cell tumor, Granular cell ameloblastoma, Central granular cell odontogenic fibroma, Granular cells in odontogenic cysts and lichen planus	Granular cells	H&E, PAS, CD-68 and alpha 1 antitrypsin positive	Not Pathognomic
Squamous Papilloma	Koilocytes	H&E	Pathognomic
Neurilemmoma/Schwannoma	Antoni type A and Type B	H&E, laminin	Pathognomic
Papillary thyroid carcinoma (PTC), meningioma, and papillary serous cystadenocarcinoma of ovary	Psammoma bodies	H&E, PAP	Pathognomic
Basal cell adenoma, pleomorphic adenoma, myoepithelioma, cystadenoma, canalicular adenoma, polymorphous low grade adenocarcinoma, Warthin's tumor, acinic cell carcinoma and mucoepidermoid carcinoma.(	Oncocytes	H&E, PTAH, Anti-mitochondrial	Not Pathognomic
Central and Peripheral Giant cell granuloma	Giant cell	H&E	Not Pathognomic
Neurilemmoma/Schwannoma	Verocay bodies	H&E	Pathognomic
CYSTS AND TUMORS			
Dentigerous cyst, Radicular cyst	Rushton bodies	H&E, Orcein, Mallory aldehyde fuschin, Pap and Gomori.	Not Pathognomic
Fibrous Histiocytoma	Touton giant cells	H&E	Not Pathognomic
Calcifying cystic Odontogenic tumor, Craniopharyngioma, Ameloblastic fibroodontoma, Odontoma	Ghost cells	H&E	Pathognomic
Odontogenic Myxoma	Pale and dark cells		Pathognomic
Langerhan's Histiocytosis	Birbeck granules	Anti-Lag antibody	Pathognomic

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