1.There are two types of immune response: acute and chronic. Using your knowledge of "inflammation" generally speaking, and which type of immune response it comes under, which cell is involved in inflammation acutely?

a. Neutrophils
b. Plasma cells
c. Lymphocytes
d. Memory cells

2.Which of the following is not a function of inflammation?
a. Delivery of leucocytes
b. Concentration of toxins
c. Initiates repair process
d. Vasodilation

Explanation :- Rather than concentrate toxins, the increased interstitial permeability leads to their relative dilution.

3.What typifies chronic inflammation?

a. immediate defensive reaction
b. vascular and exudative processes predominate
c. transient inflammation
d. ***scar tissue formation***

4.Which of the following are not classic signs of acute inflammation

a. Rubor
b. Dolor
c. collagen deposition
d. loss of function

Explanation :- rubor, dolor, calor, tumour and loss of function are all the classic hallmarks of inflammation (that is redness, pain, heat, swelling in the latin forms respectively)

5.If you were to look at an acute inflammation episode with a light microscope, what would you find?

a. keloid formation

b. satellite cells

c. collagen deposition

d. leucocyte infiltration diffusely

Explanation:
vascular and granulation tissue (leucocytes termed neutrophils, eosinophils and basophils) typify the acute inflammatory response.

6.Which of the following is a low-risk for chronic inflammation?

a. AIDS

b. bed sores for the bed-bound

c. suture material left in situ

d. ***first-degree burn***

7.Cytotoxic T lymphocyte infiltrate of tissue is a sign of?

a. acute inflammation
b. chronic inflammation
c. ischemic foci
d. caseous necrosis

8.Which of the following is a classical pattern of inflammation?
a. Keloid
b. Caseous
c. Serous
d. Monopathic

Explanation:- GUS is the mnemonic to remember common patterns of inflammation: granuloma, ulcer, suppuration, serous and fibrinous.

9.A patient with insidious fever, hemoptysis and recurrent URTI has a lung parenchyma granuloma pathologically assessed. What will the outer ring of the granuloma show?

a. Eosinophil

b. Lymphocytes

c. Necrosis

d. Basophils

10.A granuloma has formed on the skin surface post laceration. What is the inner collar about the necrotic tissue?

a.T cell

b. B cell

c. NK cell

d. Macrophage

11.An aphthous break in the mucosal epithelium is otherwise known as what?

a. An ulcer

b. ***A granuloma***

c. A serous inflammatory event

d. A suppuration

Explanation :-An epithelial break is an ulcer by definition.

GRANULOMATOUS INFLAMMATION

DR. RAKSHA VAIDYA

GRANULOMATOUS INFLAMMATION

- Granuloma formation is a protective response to chronic infection or presence of foreign material.

- It isolates a persistent offending agent, prevents it from dissemination and restricting the inflammation

- This protects the host

- Granuloma is defined as a circumscribed lesion of about 1mm in diameter composed predominantly
- Modified macrophages
- Rimmed at the periphery by lymphoid cells
- With a collar of fibroblast proliferation

Granulomatous Diseases

- BACTERIAL
- Tuberculosis
- Leprosy
- Brucellosis
- Salmonellosis
- Listeriosis
- Syphilis
- Q fever

- FUNGAL
- Histoplasmosis
- Blastomycosis
- Coccidiomycosis

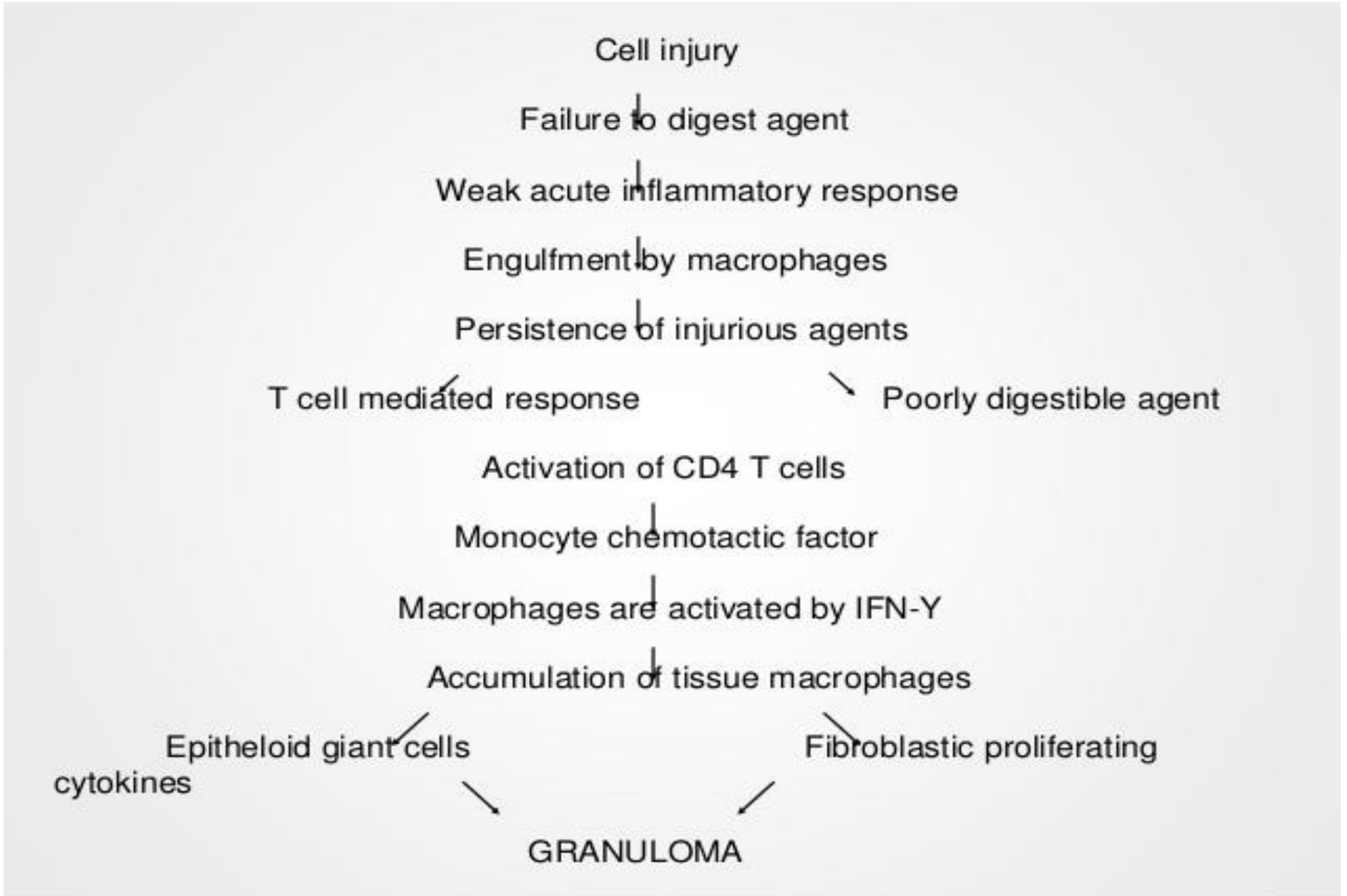
- Hypersensitivity Pneumonitis

- HELMINTHIC
- Schistosomiasis
- Trichinosis
- FOREIGN BODY TYPES
- Silica granulomatosis
- Foreign body pneumonitis

- VIRAL, CHLAMYDIAL
- Cat-scratch disease
- Lymphogranuloma venerum
- METAL INDUCED
- Berylliosis
- Zirconium Granulomatosis

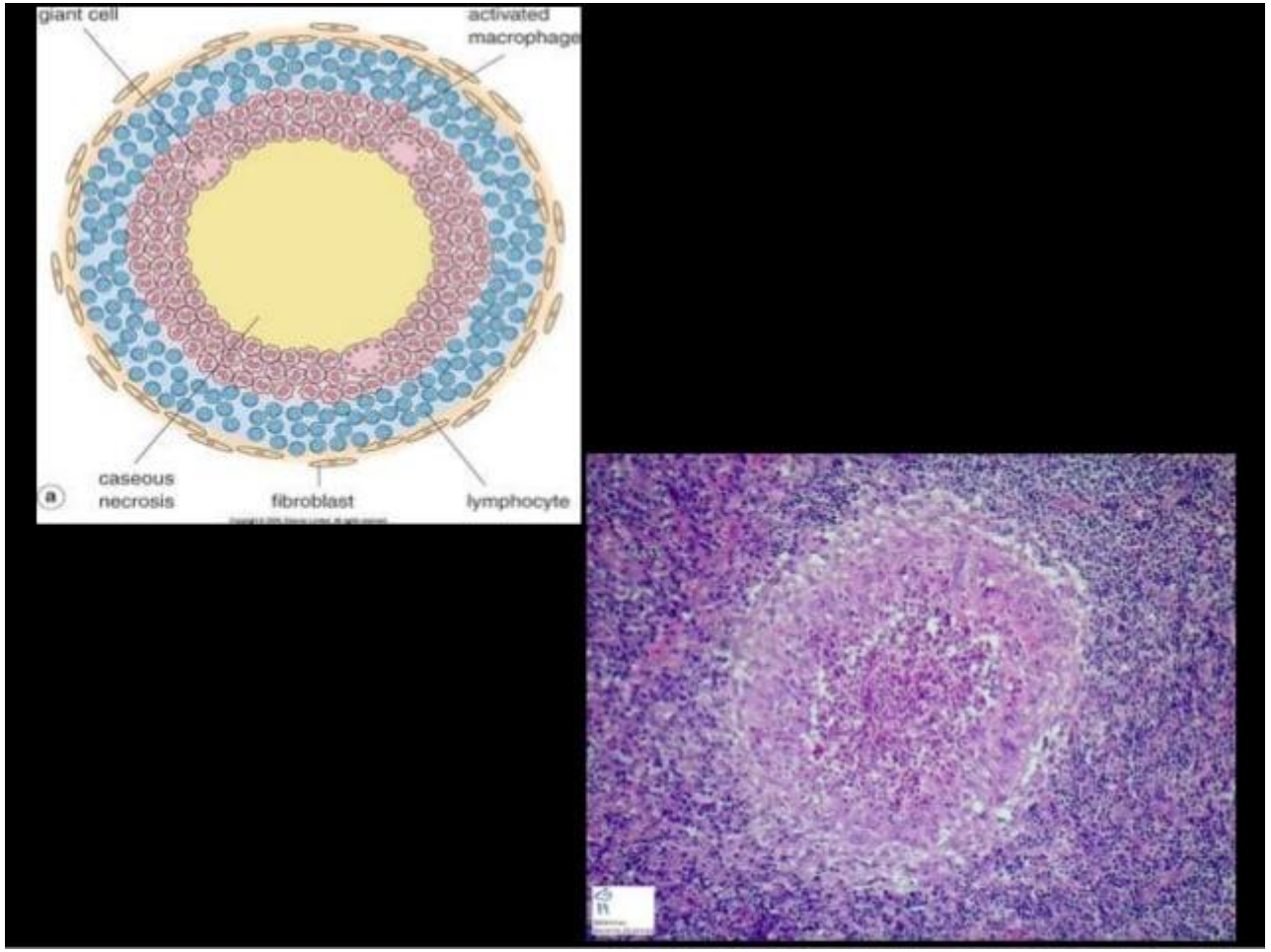
- UNKNOWN CAUSES
- Sarcoidosis
- Crohns disease
- Wegeners granulomatosis
- Giant cell arteritis
- Rheumatoid arthritis

Formation of granuloma


Cell injury
Failure to digest agent
Weak acute inflammatory response
Engulfment by macrophages
Persistence of injurious agents
T cell mediated response
Poorly digestible agent
Activation of CD4 T cells
Monocyte chemotactic factor
Macrophages are activated by IFN-Y
Accumulation of tissue macrophages
Epitheloid giant cells
cytokines
Fibroblastic proliferating
GRANULOMA

- CYTOKINES:
- Cytokines are formed by activated CD4 T cells and also by activated macrophages
- IL-1 and IL-2: proliferation of T cells
- Interferon Y: activation of macrophages
- TNF-alfa: fibroblast proliferation, activates endothelium
- Growth factors: TGF

COMPOSITION OF GRANULOMA


giant cell
activated
macrophage
caseous
necrosis
fibroblast
lymphocyte
a

EPITHELOID CELLS

- Macrophages become large and polygonal with pale, oval nuclei and abundantly cloudy eosinophilic cytoplasm
- Called epitheloid cells due to resemblance to epithelial cells
- Apposing cell membranes of epitheloid cells exhibit a high degree of inter digitation
- Macrophages become epitheloid cells:

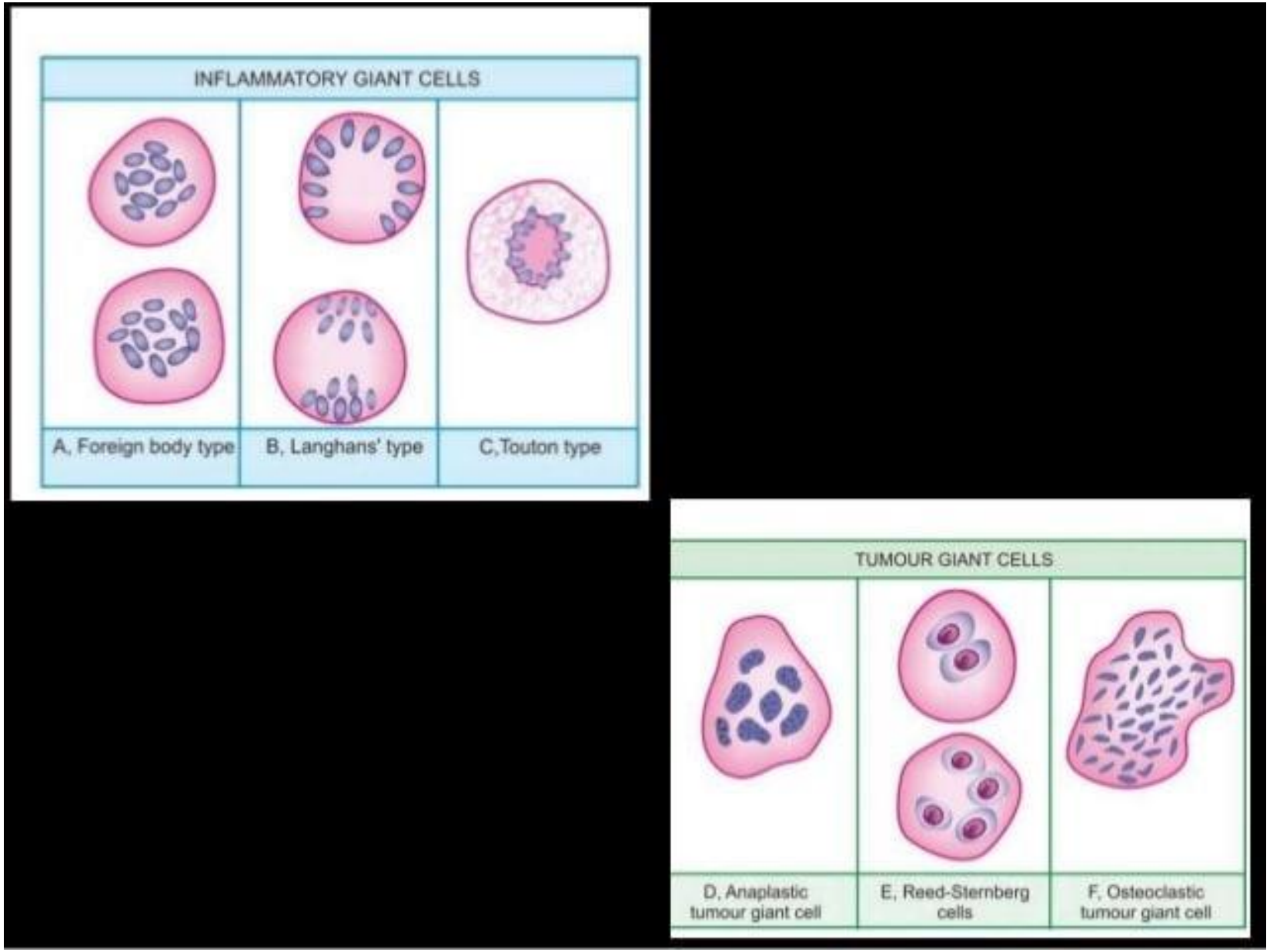
1. No phagocytosis
2. Completely phagocytosed the material
3. Extruded phagocytosed material by exocytosis

GIANT CELLS

- When macrophages encounter insoluble material they coalesce to form giant cells
- Mostly non proliferating macrophages fuse in this manner
- FOREIGN BODY:
- Large number of nuclei: 50-100
- Regular in size
- Scattered in the cytoplasm
- Site of : Suture
- Haemorrhage
- Atheroma

- LANGHANS TYPE:
- The nucleus are arranged around the periphery like a horse shoe
- EXAMPLES: Tuberculosis
- Leprosy
- Syphilis
- TOUTON GIANT CELLS:
- The cytoplasm has a foamy or vacoulated appearance
- Typically seen in Xanthomas

- Osteoclastic giant cells
- Aschoff cells
- Tumour giant cells
- Virugenic giant cells


INFLAMMATORY GIANT CELLS
A, Foreign body type
B, Langhans' type
C, Touton type
TUMOUR GIANT CELLS
D, Anaplastic tumour giant cell
E, Reed-Sternberg cells
F, Osteoclastic tumour giant cell

- LYMPHOID CELLS
- Cell mediated immunity reaction to antigen
- Lymphocytes are an integral composition of granuloma
- Plasma cells are indicative of accelerated immune response
- NECROSIS:
- Feature of some granulomatous conditions
- FIBROSIS:
- Feature of healing by proliferating fibroblasts at periphery of granuloma

TYPES OF GRANULOMAS

FOREIGN BODY	Talc, Suture material
NECROTISING GRANULOMAS	Mycobacterium Tuberculosis, Histoplasma Capsulatum, Granuloma Annulare
NON NECROTISING GRANULOMAS	M.Leprae, Sarcoidosis, SLE,
SUPPURATIVE GRANULOMAS	Actinomyces, Chlamydia Trachomatis
HISTIOCYTIC RESPONSE, NO GRANULOMAS	Listeria Monocytogenes,Mycosis Fungoides

- FOREIGN BODY GRANULOMA:
- Relatively inert foreign bodies
- Absence of T cell mediated immune response
- Found around materials such as Talc, Sutures etc
- They do not incite any specific inflammation or immune response
- Foreign material can be identified in the centre of granuloma
- Viewed with polarised light- REFRACTILE

- IMMUNE GRANULOMAS
- Variety of agents inducing persistent T cell mediated immune response
- It is usually seen when the inciting agent is difficult to eradicate

According to diseases

Granulomatous inflammation

- distinctive chronic inflammation type
- cell mediated immune reaction (delayed)
- aggregates of activated macrophages → epithelioid cell → multinucleated giant cells (of Langhans type x of foreign body type)
- NO agent elimination but walling off
- intracellulary agents (TBC)

Granulomatous inflammation

- 1. Bacteria
  - **TBC**
  - leprosy
  - syphilis (3rd stage)
- 2. Parasites + Fungi
- 3. Inorganic metals or dust
  - silicosis
  - berylliosis
- 4. Foreign body
  - suture (Schloffer „tumor“), breast prosthesis
- 5. Unknown - **sarcoidosis**

Tuberculosis – general pathology

- 1. TBC nodule – proliferative
- Gross: grayish, firm, 1-2 mm (milium) → central soft yellow necrosis (cheese-like – caseous) → calcification
- Mi: central caseous necrosis (amorphous homogenous + karyorrhectic powder) + macrophages → epithelioid cells → multinucleated giant cells of Langhans type + lymphocytic rim
- 2. TBC exsudate – sero-fibrinous exsudate (macrophages)

Leprosy

- ***M. leprae,*** Asia, Africa
- in dermal macrophages and Schwann cells
- air droplets + long contact
- rhinitis, eyelid destruction, facies leontina
- 1. lepromatous – infectious
  - skin lesion – foamy macrophages (Virchow cells) + viscera
- 2. tuberculoid – steril
  - in peripheral nerves – tuberculoid granulomas - anesthesia
- death – secondary infections + amyloidosis

Syphilis

- ***Treponema pallidum*** (spichochete)
- STD + transplacental fetus infection
- acquired (3 stages) x congenital
- basic microspical appearance:
  - 1. proliferative endarteritis (endothelial hypertrophy → intimal fibrosis → local ischemia) + inflammation (plasma cells)
  - 2. gumma – central coagulative necrosis + specific granulation tissue + fibrous tissue

Syphilis

- 1. primary syphilis - contagious
- chancre (ulcus durum, hard chancre)
- M: penis x F: vagina, cervix
- painless, firm ulceration + regional painless lymphadenopathy
- spontaneous resolve (weeks) → scar

Syphilis

- 2. secondary syphilis - contagious
- after 2 months
- generalized lymphadenopathy + various mucocutaneous lesions
- condylomata lata - anogenital region, inner thighs, oral cavity

Syphilis

- 3. tertiary syphilis
- after long time (5 years)
- 1) cardiovascular - syphilitic aortitis (proximal a.)
  - endarteritis of vasa vasorum → scaring of media → dilation → aneurysm
- 2) neurosyphilis – tabes dorsalis + general paresis
  - degeneration of posterior columns of spinal cord → sensory + gait abnormality
  - cortical atrophy → psychic deterioration
- 3) gumma – ulcerative lesions of bone, skin, mucosa – oral cavity

Congenital syphilis

- 1) abortus
  - hepatomegaly + pancreatitis + pneumonia alba
- 2) infantile syphilis
  - chronic rhinitis (snuffles) + mucocutaneous lesions
- 3) late (tardive, congenital) syphilis
  - > 2 years duration
  - Hutchinson triad – notched central incisors + keratitis (blindness) + deafness (injury of n. VIII)
  - mulberry molars + saddle nose