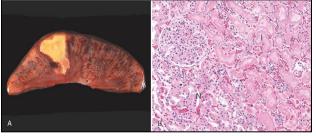
Unit II – Problem 6 – Pathology: Necrosis and Tuberculosis

C

NECROSIS

- **Definition**: cell death that is associated with loss of membrane integrity and leakage of cellular contents culminating in dissolution of cells, largely resulting from the degradative action of enzymes on lethally injured cells. It often causes local host reaction (inflammation) to eliminate dead cells and start the subsequent repair process.
- Patterns of necrosis:
 - Coagulative necrosis:
 - \checkmark Underlying tissue architecture is preserved for at least several days.
 - ✓ Affected tissues take on a firm texture.
 - ✓ Injury denatures enzymes blocking the proteolysis of the dead cells.
 - ✓ Characteristic of infarcts in all solid organs except the brain.



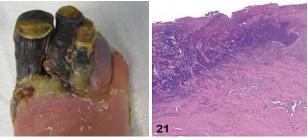
• Liquefactive necrosis:

- ✓ It occurs in focal bacterial or fungal infections.
- ✓ Infection stimulates accumulation of inflammatory cells and enzymes of leukocytes digest the tissue.
- \checkmark It also occurs in hypoxic death of cells within the central nervous system.
- \checkmark Tissue is completely digested and transformed into a liquid viscous mass.

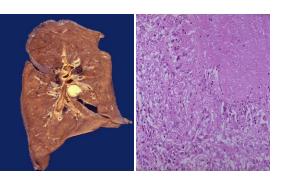


• Gangrenous necrosis:

- \checkmark It is not a distinctive pattern but a clinical term.
- ✓ Coagulative necrosis affecting multiple tissue layers of a limb, generally the lower leg that has lost its blood supply.
- ✓ Might be modified by the liquefactive action of bacteria and leukocytes (wet gangrene).

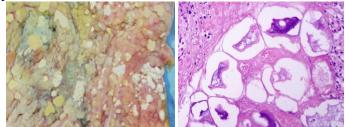


- Caseous necrosis:
 - ✓ It occurs in tuberculosis.
 - ✓ Gross: cheesy-white appearance.
 - ✓ Histology: collection of fragmented or lysed cells with an amorphous granular pink appearance within a granuloma.

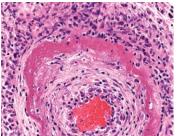




- Fat necrosis:
 - ✓ Descriptive term of focal areas of fat destruction.
 - ✓ Mostly due to release of activated lipases in acute pancreatitis.
 - ✓ Enzymes liquefy membranes of fat cells and lipases split the triglyceride esters contained within fat cells.
 - ✓ Released fatty acids combine with calcium → visible chalky white areas (fat saponification).
 - ✓ Histology: necrotic fat cells with basophilic calcium deposits surrounded by inflammatory reaction.



- Fibrinoid necrosis:
 - ✓ Seen by light microscopy.
 - ✓ Usually in immune reactions in which complexes of antigens and antibodies are deposited in the walls of arteries.
 - ✓ Deposited immune complexes + leaked fibrin → bright pink and amorphous appearance.
 - ✓ Seen in immunologically mediated diseases (e.g. SLE and polyarteritis nodosa)



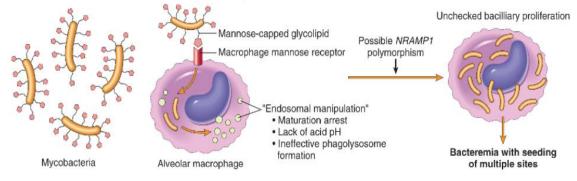
TUBERCULOSIS

- <u>Cause of tuberculosis</u>: Mycobacterium tuberculosis.
- Primary tuberculosis is characterized by:
 - Focal casseating necrosis in:
 - \checkmark Lower lobes of the lung.
 - ✓ Hilar lymph nodes.
 - These foci will undergo fibrosis and calcification forming what is known as Ghon complex (thus Ghon complex is defined as area of fibrosis and calcification indicating the patient has been exposed to primary tuberculosis. These calcified nodules are sub-pleural).
 - Primary tuberculosis is ASYMPTOMATIC (has no signs or symptoms) but results in positive PPD test.

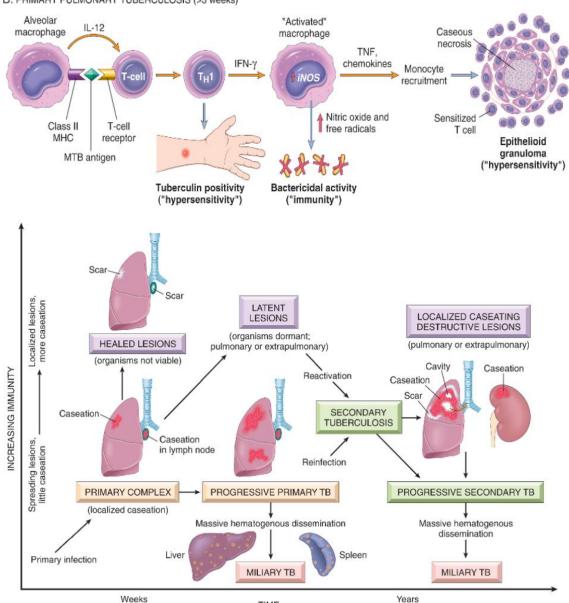
- Secondary tuberculosis:

- It arises with reactivation of tuberculosis due to immunocompromised conditions, AIDS or aging.
- It occurs at apex of the lung (where oxygen tension in highest!).
- Secondary tuberculosis is characterized by:
 - ✓ Foci of casseous necrosis.
 - ✓ Miliary pulmonary tuberculosis: ting little regions of tuberculosis scattered across the entire lung.

A. PRIMARY PULMONARY TUBERCULOSIS (0-3 weeks)



B. PRIMARY PULMONARY TUBERCULOSIS (>3 weeks)

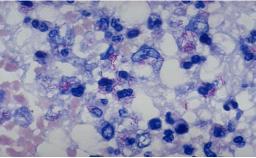


TIME

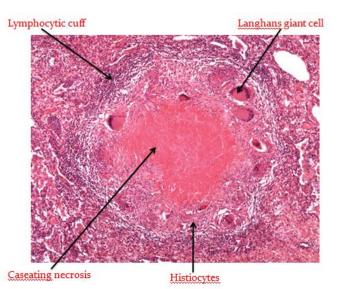
- <u>Clinical features of tuberculosis:</u>
 - Fever and night sweats.
 - Cough with hemoptysis.
 - Weight loss.

- Morphology:

• **Biopsy**: it reveals caseating granulomas (which are seen in tuberculosis and fungal infection too). Therefore, acid-fast stain will be done and it will reveal the presence of red acid-fast bacilli.



- Tuberculosis granuloma is described by the presence of:
 - ✓ Casseous necrosis.
 - ✓ Epithelioid histiocytes.
 - ✓ Lymphocyte infiltration.
 - ✓ Giant cells.



- Systemic spread of tuberculosis can occur to:
 - Meninges (at base of the brain) resulting in meningitis.
 - Cervical lymph nodes.
 - Kidney: producing sterile pyuria.
 - Lumbar vertebrae (Pott's disease).

