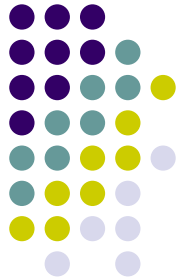
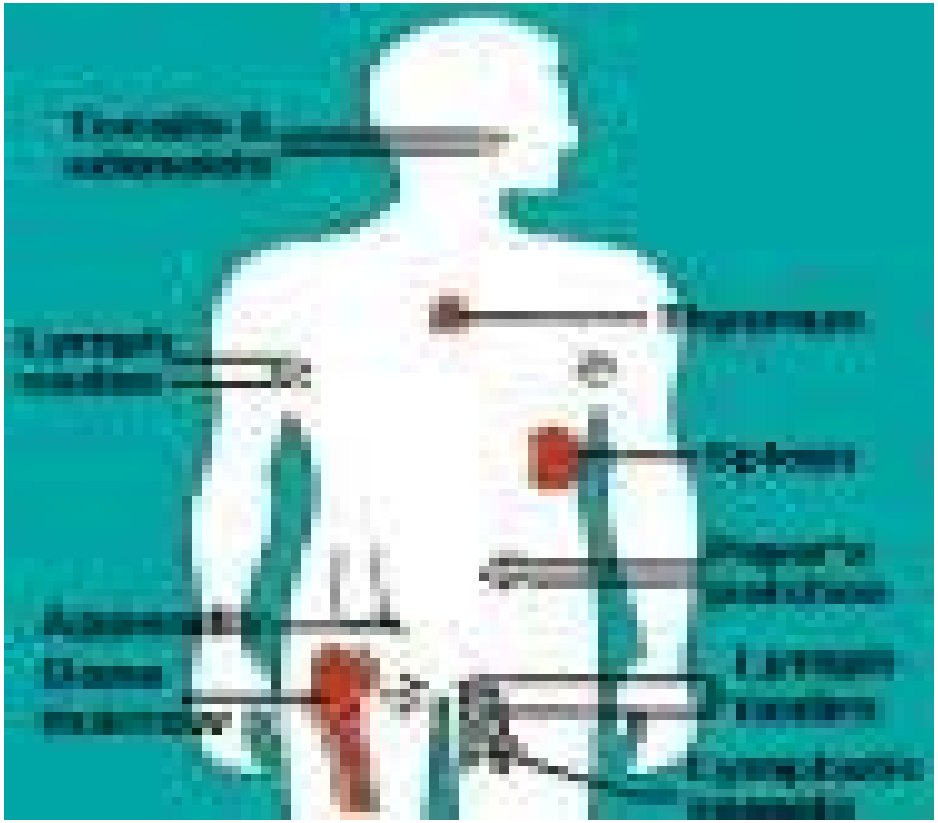
- **Lymph Nodes & Spleen**

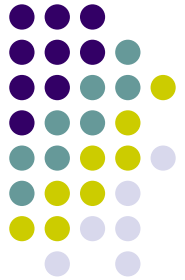
LYMPH NODE



- Imp component of lympho reticular system
- Others- thymus
- spleen
- tonsils inc adenoids
- Paeyer s patches
- Less organised –Bone Marrow
- MALT-lungs GIT



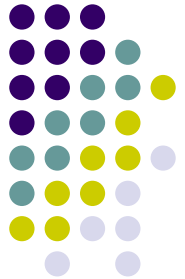
Structure of LN



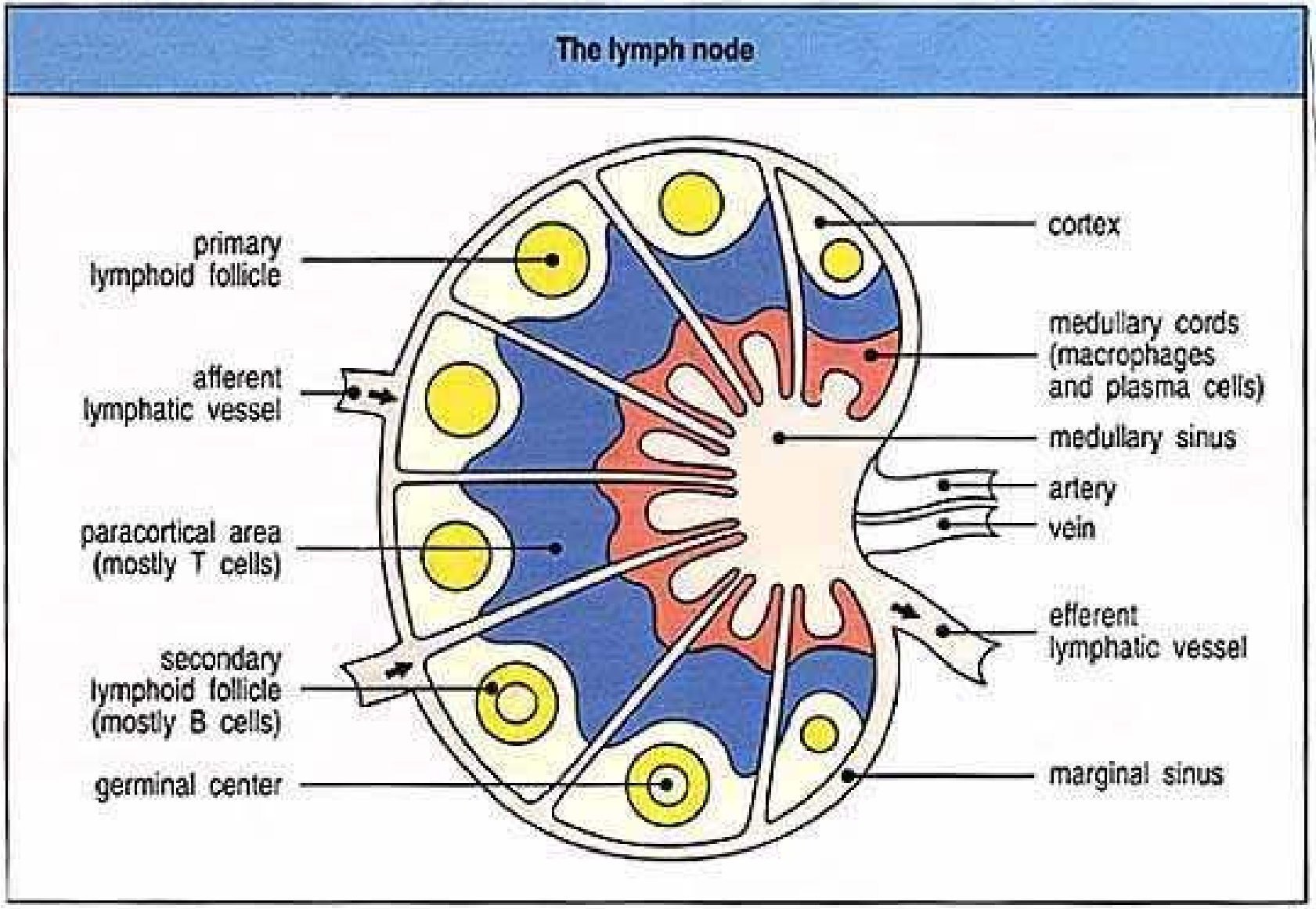
- Capsule-perforated by afferent lymphatic channels
- Enter into sub capsular sinus
- Branch
- Terminate at concavity or hilum as efferent lymphatic vessel

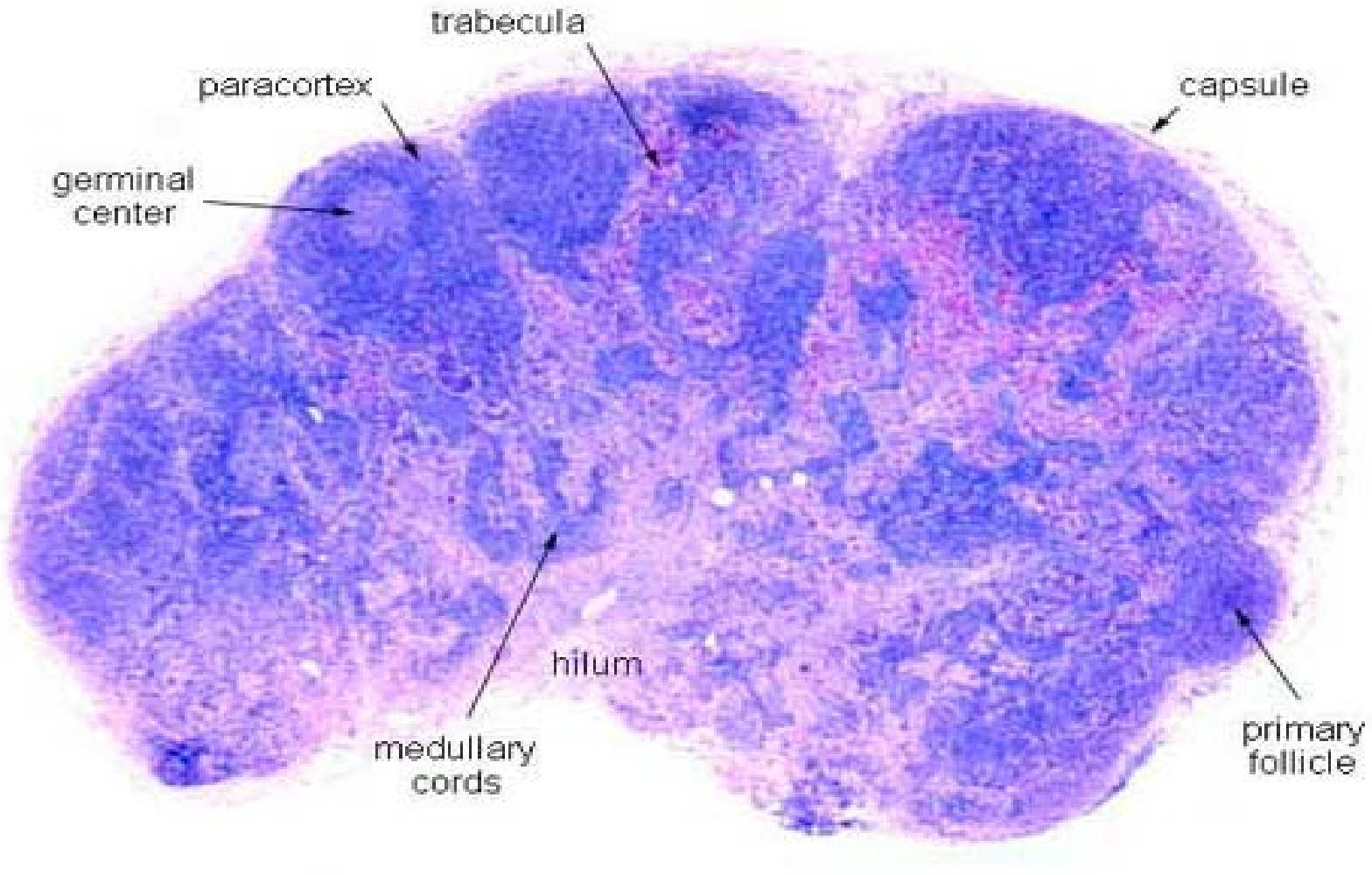
- C/S outer cortex ,inner medulla
- Cortex-Lymphoid folls
- Surr by paracortex- T cell area
- Medulla –cords & sinuses.

Lymphoid folls

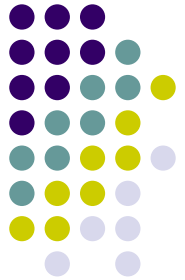


- B cell areas
- Ag.ic stim –dev G. C.
- Composed of FCC
- Surr by small LCs(mantle zone)
- Outside mantle zone –marginal zone.


The lymph node
primary lymphoid follicle
afferent lymphatic vessel
paracortical area (mostly T cells)
secondary lymphoid follicle (mostly B cells)
germinal center
cortex
medullary cords (macrophages and plasma cells)
medullary sinus
artery
vein
efferent lymphatic vessel
marginal sinus

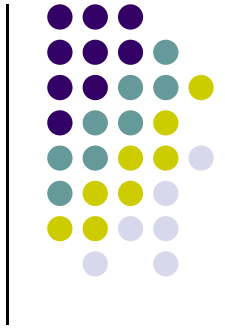

trabecula
paracortex
capsule
germinal
center
hilum
medullary
cords
primary
follicle

Functions of Lymph node



- Immune Response T cell & Bcells
- Involved in both CMI & humoral immunity.
- Active phagocytosis of particulate matter

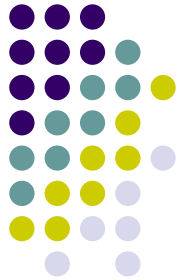
Causes of LN opathy



- Non neoplastic & neoplastic

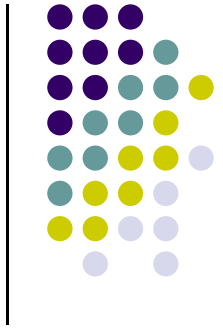
- Non Neo-Inflammatory
- Immune response
- Neoplastic –primary
- Secondary(Metastatic deposits)
- Primary Lymphoma
- Plasma cell disorders
- Langerhans cell histiocytosis

Reactive Ln adenitis



- Acute -Supp. &non Supp.
- Chronic-Specific Or Non Specific
- Non Sp.-Reactive H plasia
- Sp- Granulomatous

LNopathy



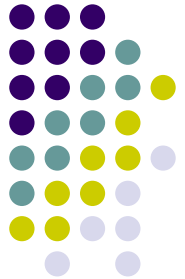
- **Specific conditions –**
- AIDS related
- Kikuchis lymphadenitis
- Angiofoll. H plasia
- Angio immunoblastic
- Dermatopathic
- Sinus HCosis with massive LNopathy
- Lnopathy in autoimmune dis

Acute Non specific Lymphadenitis

- Lymph nodes undergo reactive changes due to
- Microbial inf
- Their breakdown products
- Cell debris
- Foreign matter
- All types of ac. Infl. –cause this in LNs draining the area of infl.eg cervical -oral cavity inf.,inguinal – lower limb infs.

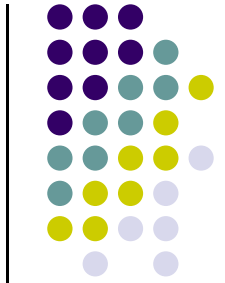
Morphology

- Gross : Mild & transient process.
- LN enlarged &tender
- maybe fluctuant
- skin –red & hot



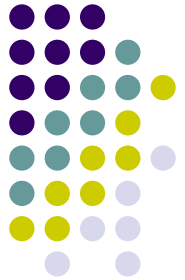
- M/ E Prominence of lymphoid follicles with large germinal centres.
- Active phagocytosis & mitosis in the centre
- Many show necrosis ,NPs & abscess form.
- Sinusoids –dilated & congested,contain NPs
- lined byhypertrophied cells.

Chronic Non Specific Lymphadenitis



- Reactive Hyperplasia
- 3 types
- Follicular hyperplasia
- Paracortical Hyperplasia
- Sinus histiocytosis

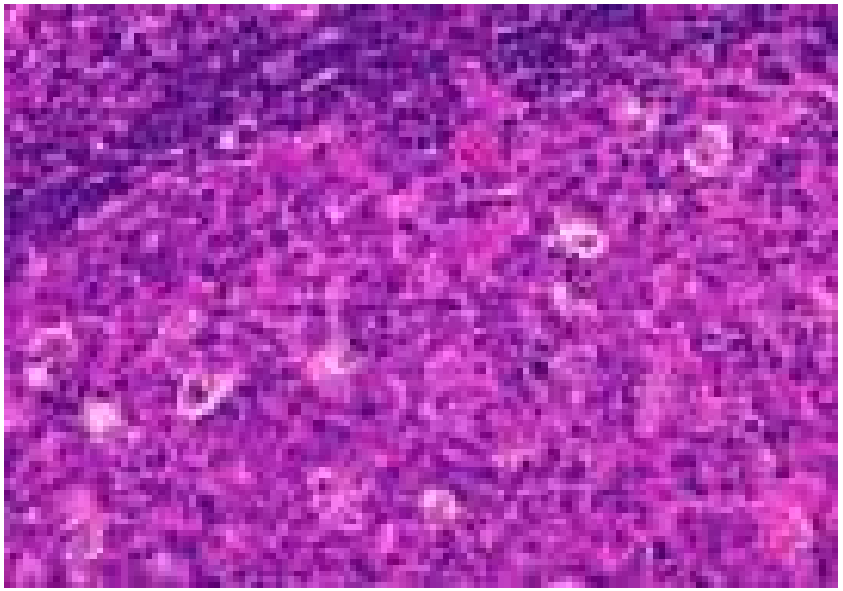
Follicular Hyperplasia



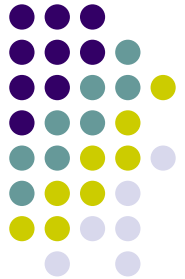
- Commonest pattern
- Causes Chronic infs non sp
- R.A.
- Toxo
- AIDS

Reactive Hyperplasia

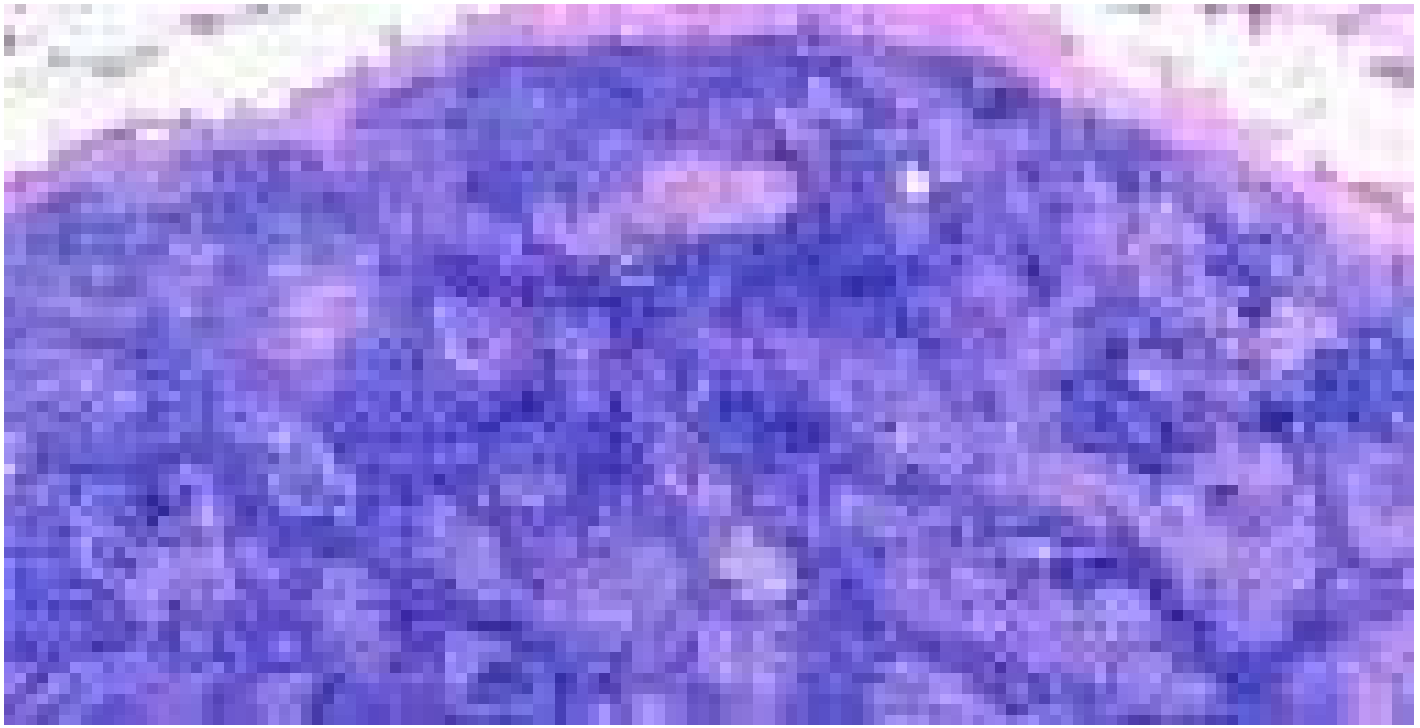
Reacive Hyperpasia-G. C.



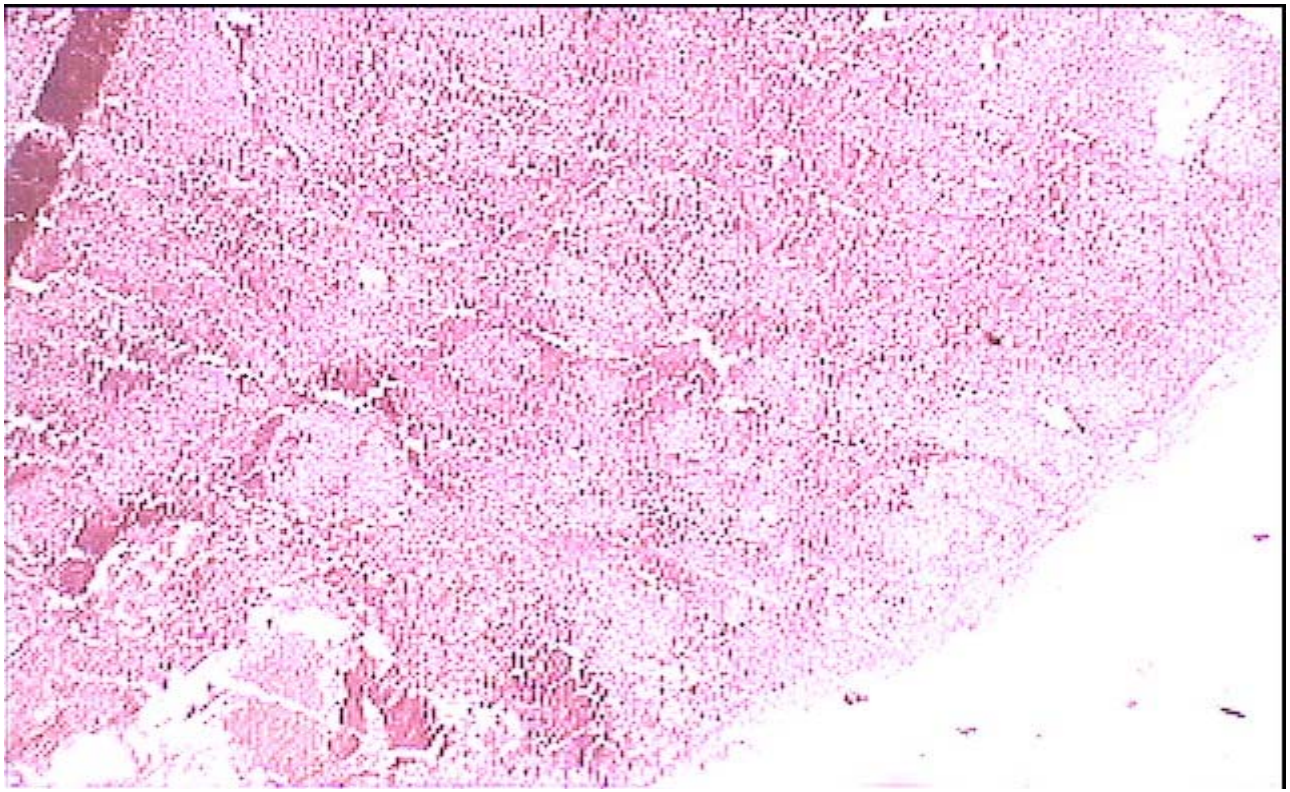
Morphology



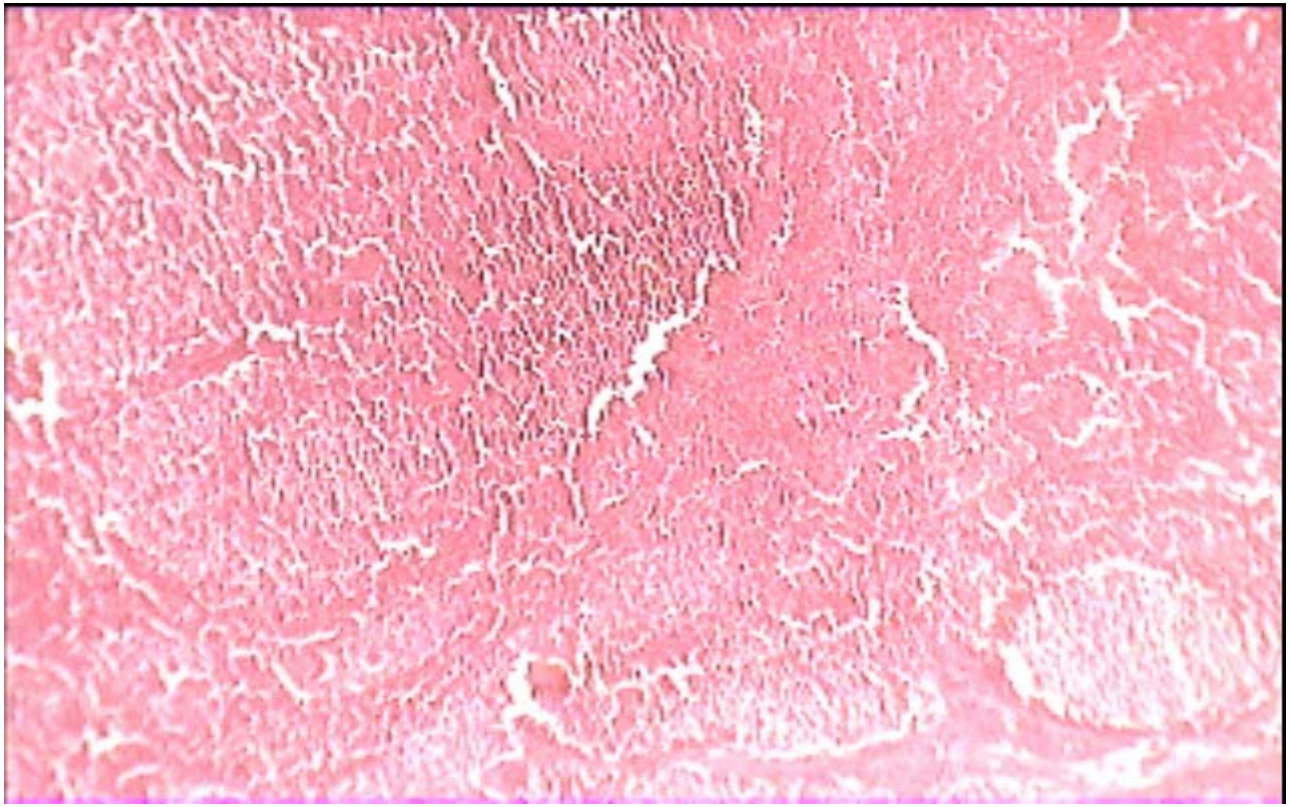
- Large follicles with prominent germinal centres –appear to bulge against a collar of small B lymphocytes.
- Germinal centres-lympho in varying stages blast transform.
- Mitotic activity
- HCs containing phagocytosed material
- Parafollicular zones and medulla-cellular
- LCs and HCs ,few EPs& NPs
- H plasia of cells lining lymphatic sinuses
-
-



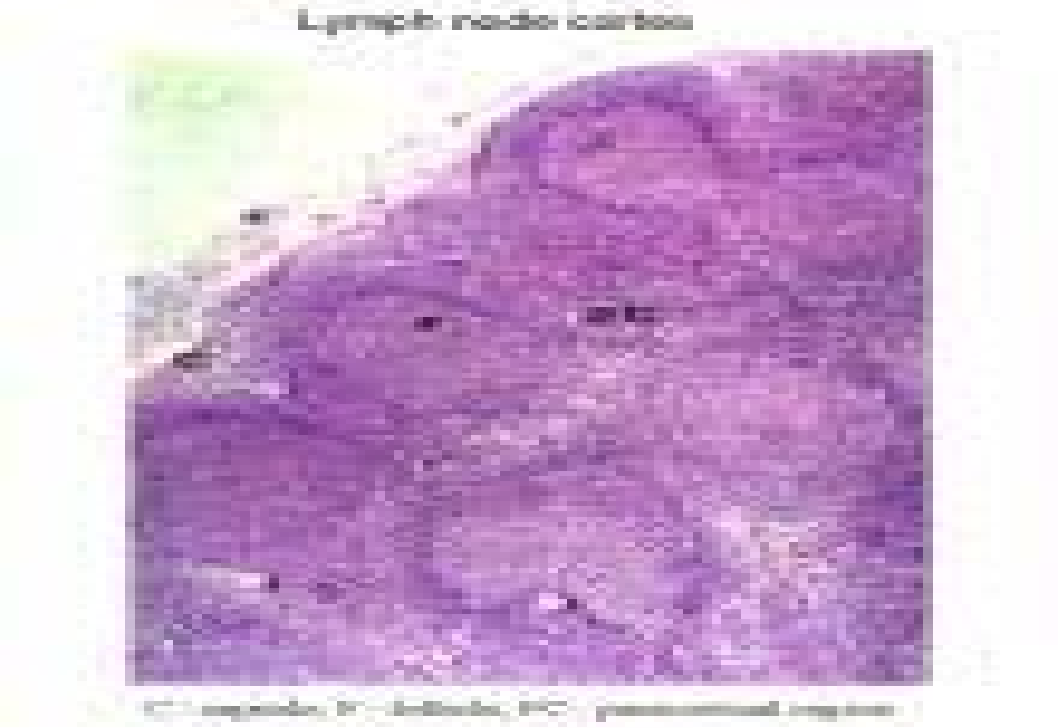
Reactive Hyperplasia



Reactive Hyperplasia



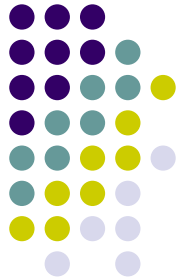
Lymph reactive hyperplasia



Points to Remember

- Preservation of Nodal arch.
- Variation in size & shape of follicles
- Mixed population of LC & HC in GC
- Prominent phagocytic activity in G. C.

Paracortical Lymphoid Hyperplasia

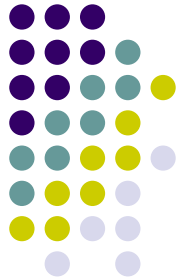


- Reactive changes in T cell region
- Encroaches upon & effaces the G.C.
- T cells undergo blastic trans. to immunoblasts
- Hypertrophy of sinusoidal & vascular endoth. Cells
- Mixed cellular infiltrate-MPs & EPs
- Mottled T cell appearance

Causes

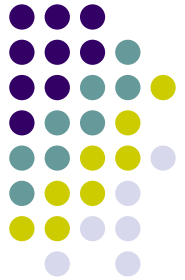
- Drugs-Dilantin
- Foll. Small pox vaccination
- Other vaccines
- Viral infs.
- Auto immune disorders

Sinus Histiocytosis



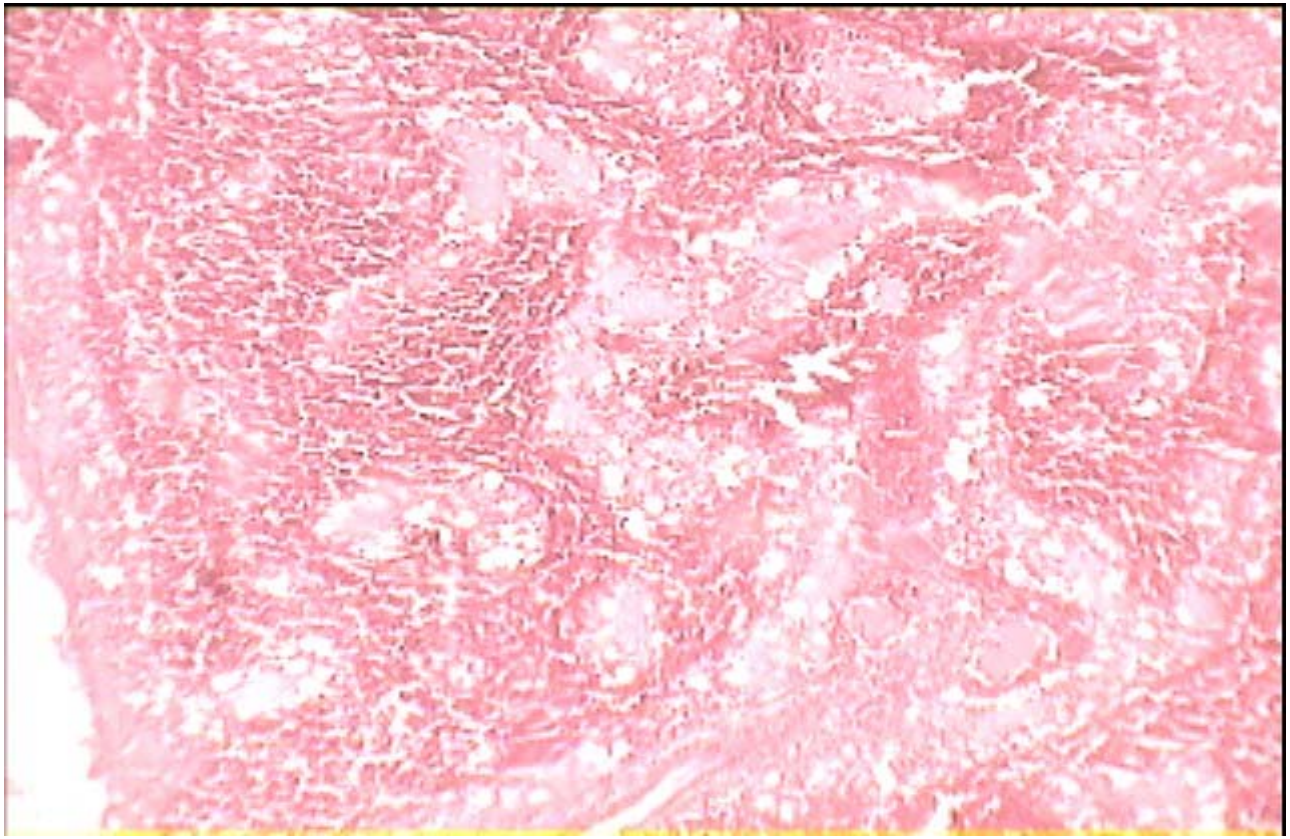
- Sinus H plasia
- Causes –
- In regional LNs draining malignant tumors
- In LNs draining inflammatory lesions.

Morphology

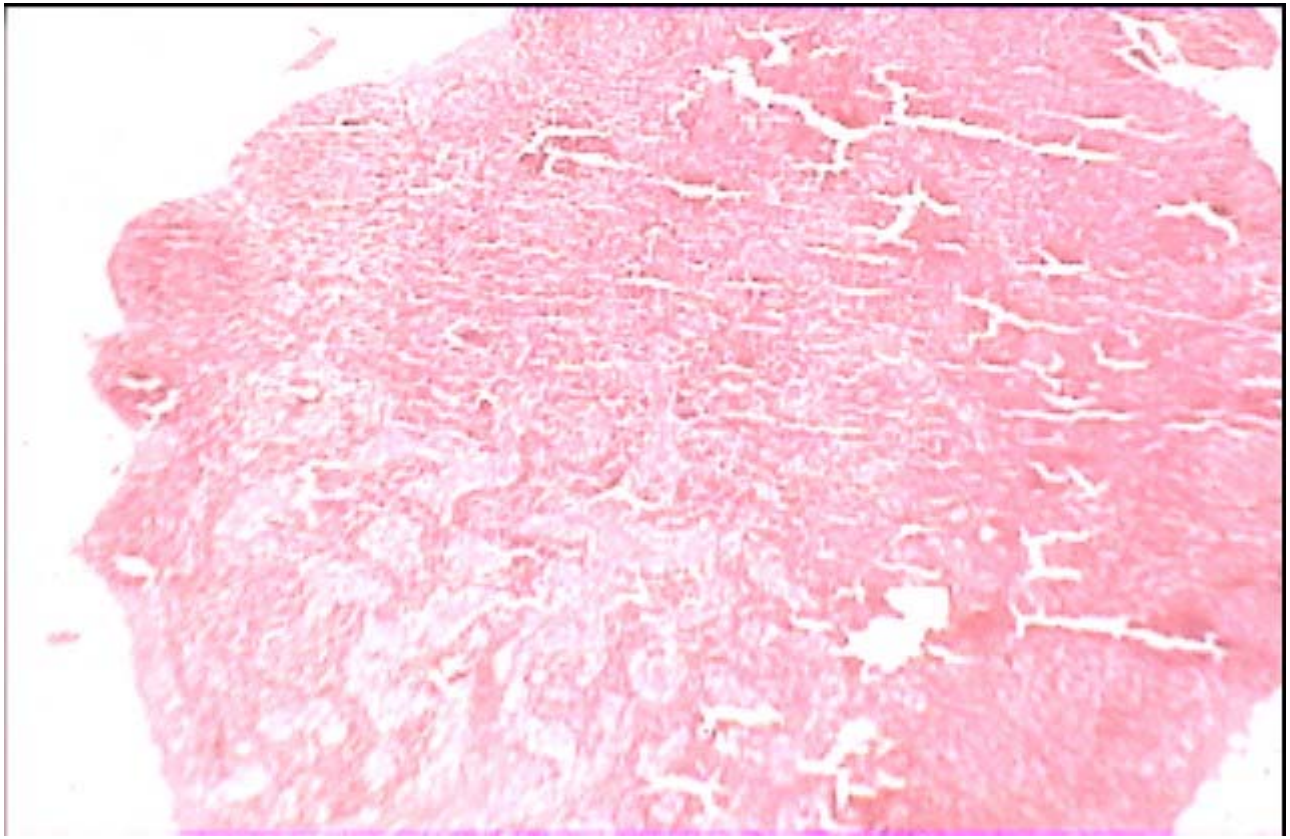


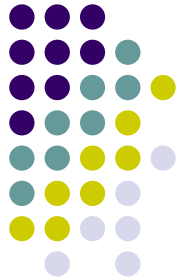
- Diste ntion ,dilatation or expansion of the sinuses.
- lining endoth. Cells are hypertrophied.
- Sinuses packed with HCs

Sinus Histiocytosis



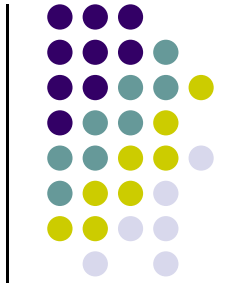
Sinus Histiocytosis





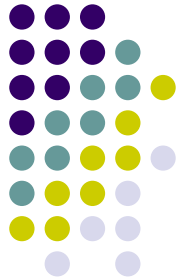
- Specific Reactive conditions of LNs

AIDS Related Lymohadenopathy



- Lymph Node changes variable
- Opp. Infs
- Kaposi sarcoma
- Lymphomas
- Florid reactive H plasia

Florid Reactive Hyperplasia



- Reactive follicles
- Collection of monocytoid B cells in sinuses & interfollicular areas
- Reactive germinal centres may show follicular lysis
- Invagination of mantle zone into germinal centres
- Follicular involution– Moth eaten app.

-

- Lymphocyte depletion –some
- Burnt out LN
- Small & atrophic
- Seat of nu. Opp.infs.

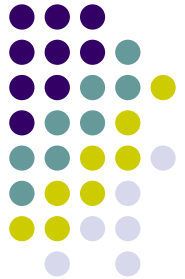
Infectious mononucleosis

- Confused with lymphoma
- -Effacement of architecture
- Capsular infilt.
- Prolifer of immunoblasts ,immature & mature plasma cells.
- Follicular Hyperplasia
- Diag. clinched- PBF
- Serology

Post vaccinial –Pseudolymphoma

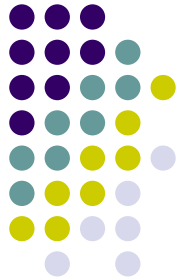
- May mimic lymphoma
- Diffuse or nodular paracortical expansion
- Mixed cellular prolif
- EP,Plasma cells,immunoblasts

Dermopathic lymphadenitis

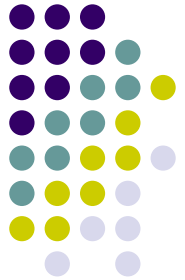


- Lipomelanotic LNpathy
- Nodal H plasia sec to gen dermatitis
- T cell response to skin Ag –processed & presented by interdigitating cells
- C/S –LN Bulging pale yellow
- Linear black streaks –melanin
- M/E T cell paracortical zone widened
- histiocytes,interdigitating cells langerhans cells.
- HCs-contain melanin & fat.

Drug Related

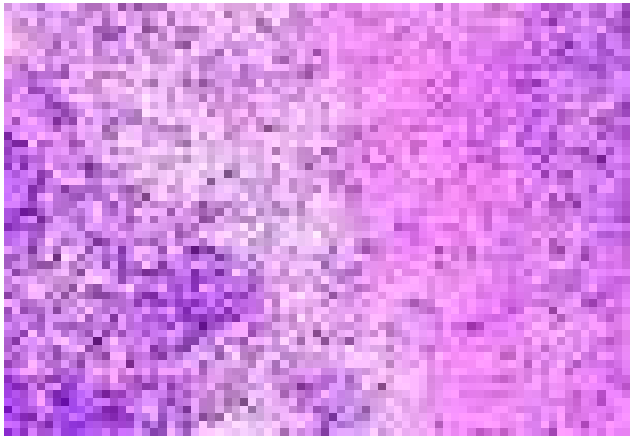


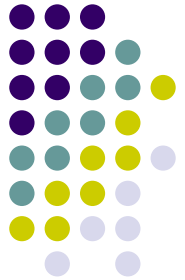
- Esp seen with Antiepileptic dilantin
- Partial eff of arch.by polymorphic cell pop.
- immunoblasts
- EP
- plasma cells
- Some IBs –mimic RS cells



- SLE
- RH Arthritis -- FH ,Pl cell prolif
- Necrotising Lymphadenitis Kikuchi
- young females
- painless LNpathy
- fever

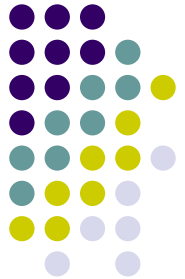
kikuchi





- M/E focal well circumscribed paracortical necrotising lesions.
- HCs
- Karyorrhectic debris
- Fibrin deposits
- Mononu.& NPs scanty
- PLasmacytoid LC
- C/F-Benign & self limited.

Castleman Dis.

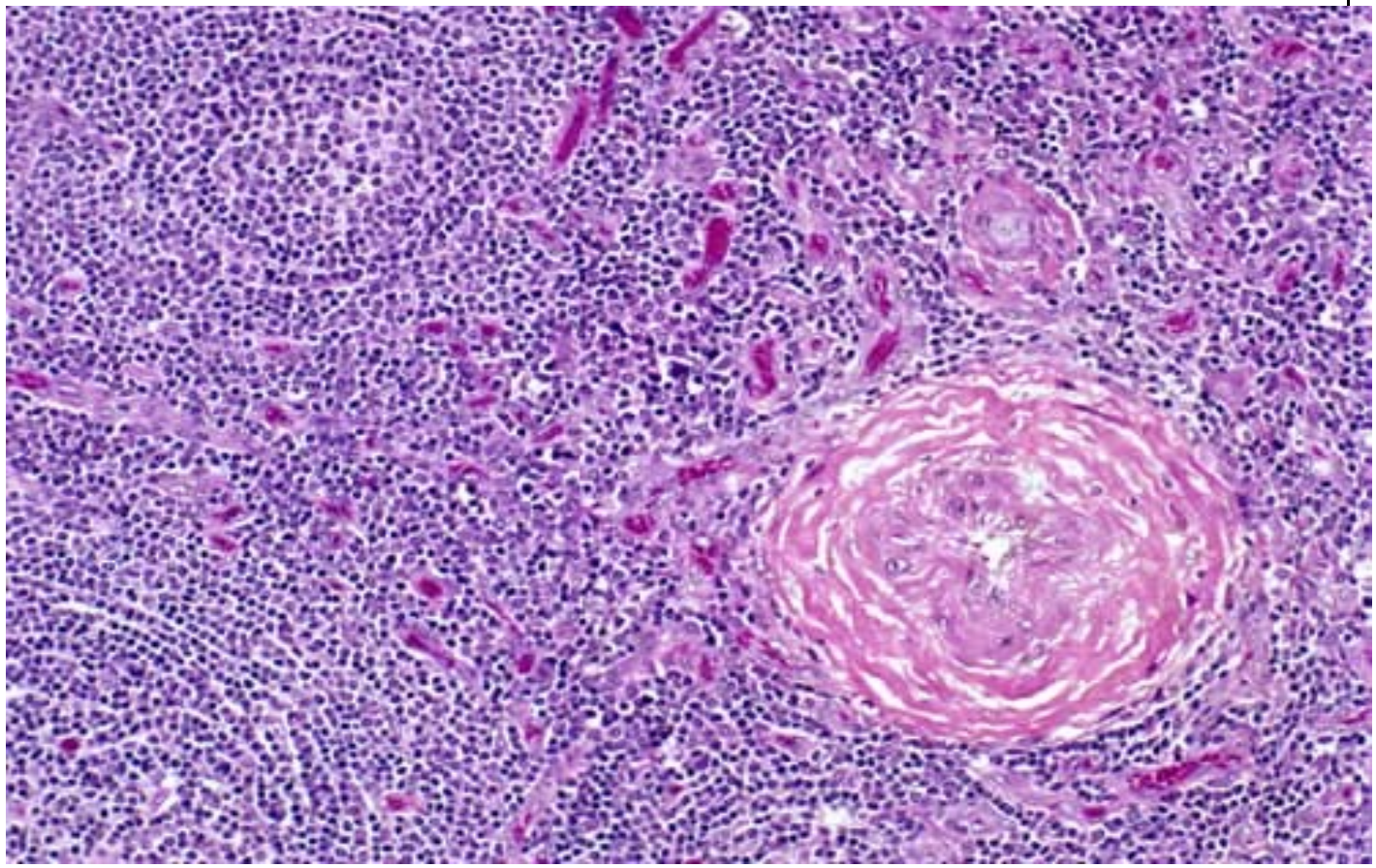


- Giant LN Hplasia
- 2 cat.—Hyaline Vasc.(Angio foll)
- Plasma cell type
- C/F-Solitary form90% HV type
- Systemic form -always Pl cell type
- Gen LNpathy
- Spleen involv.

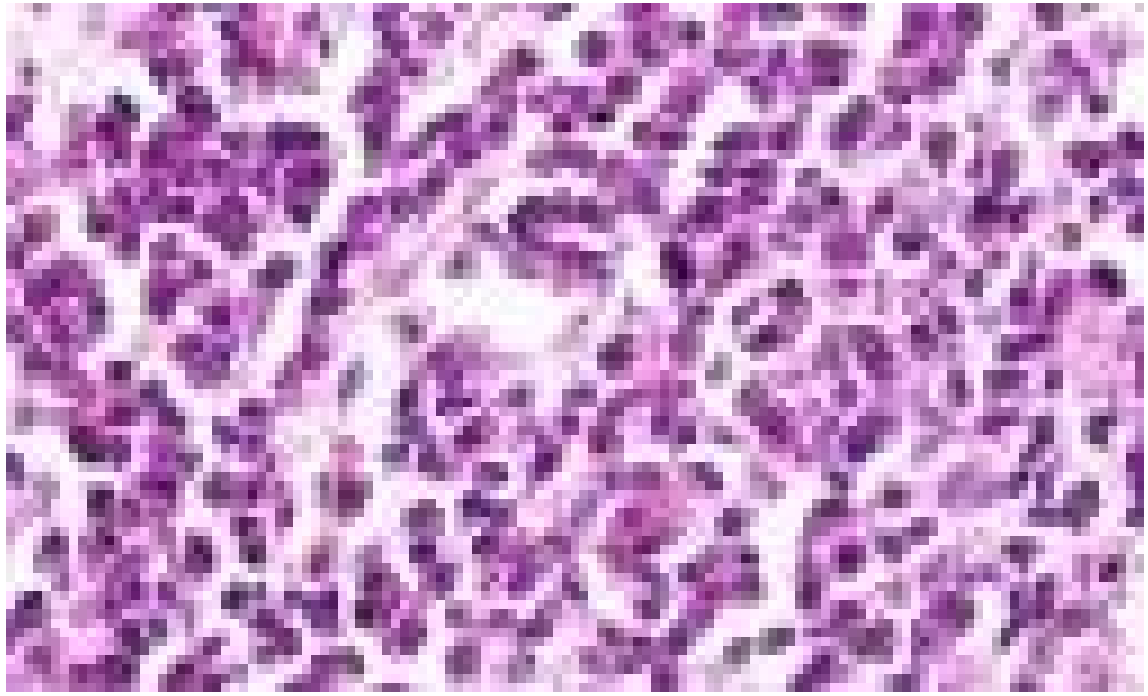
Hyaline Vascular Type

- Large follicles
- Marked vascular proliferation & hyalinisation in G. C.
- Concentric layering of LCs at peri.-
- Onion skin appearance
- Interfoll. Zone –Inc vascularity
- Pl cells & immunoblasts

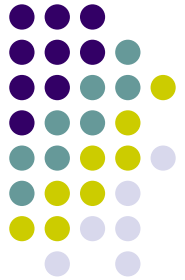
Castleman HV



Castleman dis

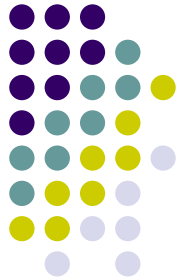


Plasma cell type

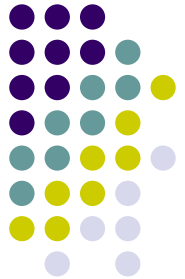


- Diffuse prolif of pl cells in I/F zone with /without Russel bodies
- Hyaline vasc changes in foll.—minimal
- Foll normal or enlarged
- Prominent G. C. containing PAS +ve material

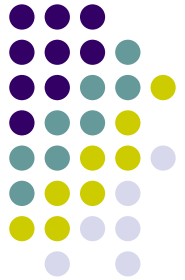
Angio immunoblastic Lymphadenopathy



- Exc. In adults & elderly
- C/ F fever,an (haemolytic),gen LNpathy,Polyclonal hyper gammaglobulinemia
- Lesions seen in LN
- spleen liver
- thymus
- BM



- M/E Oblit. Of LN arch
- Polymorphic cell infiltrate(LC, EP ,pl cells,immunoblasts)
- Prolif of finely arborising vessels
- Germinal centres burnt out-
- Contain HC ,epith cells



- Nature –Unclear
- shd be regarded as Atypical Immuno proliferative disorder
- Ranges ---Reactive & rev to
- clearly neoplastic & agg.

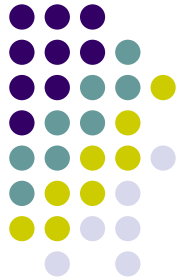
Sinus HCosis with massive LNopathy

- Rosai Dorfman Disease
- C/F-painles B/L LN opathy
- Us cervical
- Fever ,raised ESR
- TLC inc.
- Polyclonal hyper gamma globulinemia

- Gross Matted LNs
- Perinodal fibrosis
- M/E Pronounced dil. Of lymph sinuses
- Partial or total eff.
- Sinuses occ by LC ,pl cells,,HC –containing lipid
- Lymphophagocytosis HCs with engulfed Lc.
- Capsular & pericap fibrosis

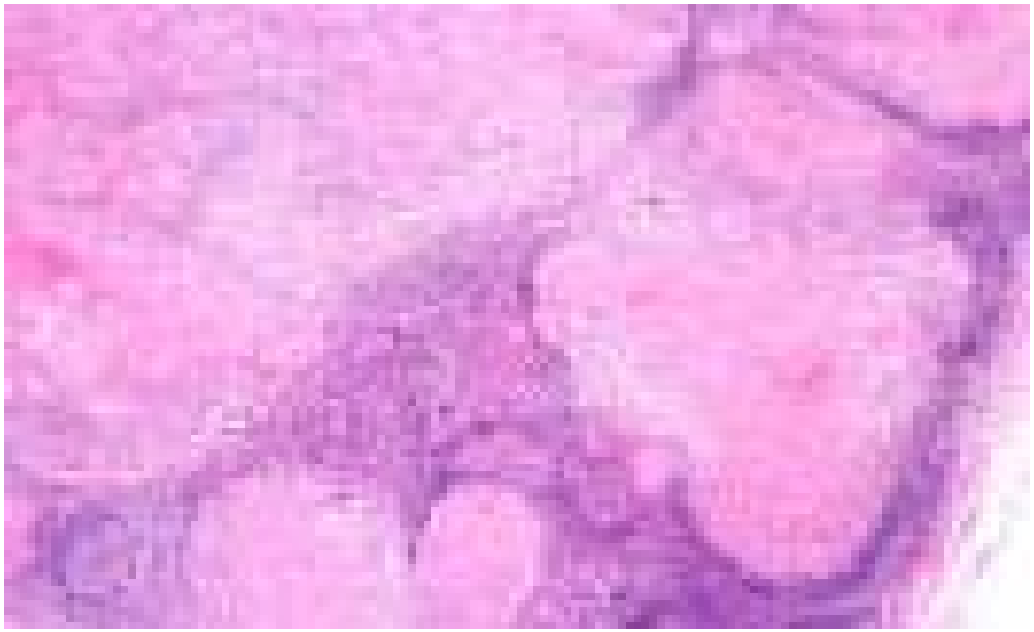
- Extra nocal involv -1/4 cases.
- eyes,head & nneck region
- Skin CNS GIT etc
- BM & Spleen –spared.
- Etio. Not known
- ? Inf viral
- ?immunulogical defect
- May-spont resolution
- Protracted course
- unaffected by therapy

Granulomatous LNpathy



- Multiple causes bact
- fungal
- viral
- foreign body
- tms-HD etc
- Common causes TB Sarcoid Leprosy LGV Cat scratch Dis Histopl Toxo

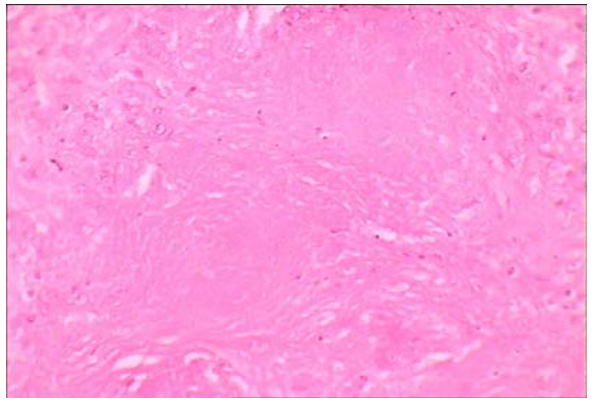
granulomas



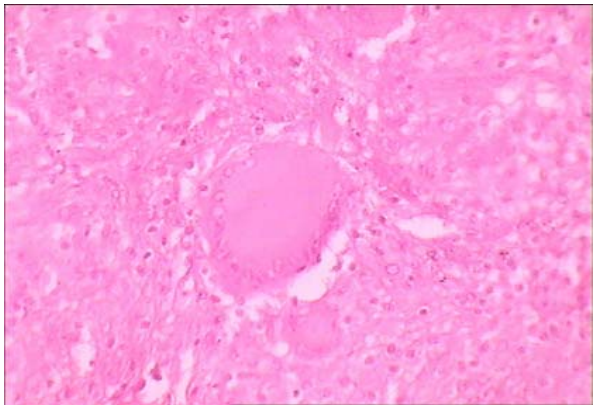
Tb lymph node

- Identification of tissue
  - Capsule
  - Lymphoid tissue
  - Lymphoid follicles with germinal centers
- Histological diagnosis

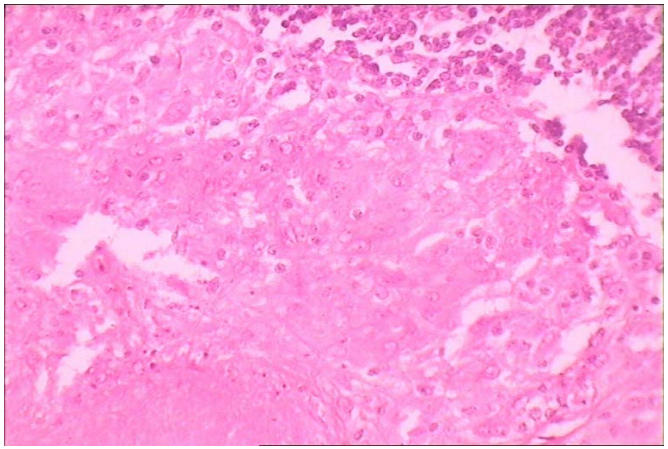
- Epitheloid cell granuloma with central caseation necrosis
  - Granular, amorphous, eosinophilic debris



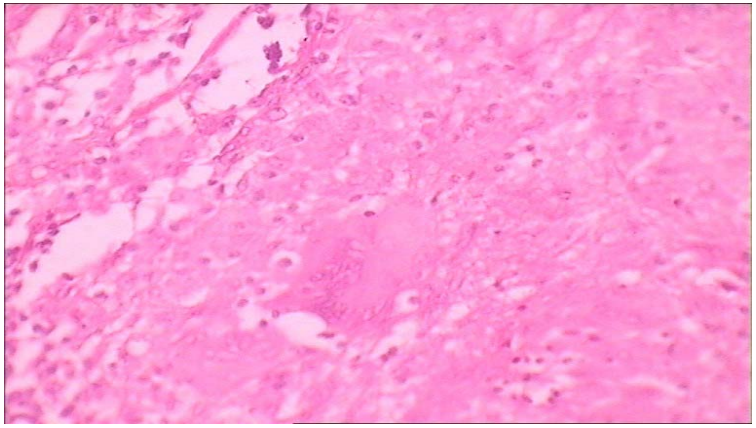
- Another focus showing a giant cell with peripheral nuclei forming a horseshoe



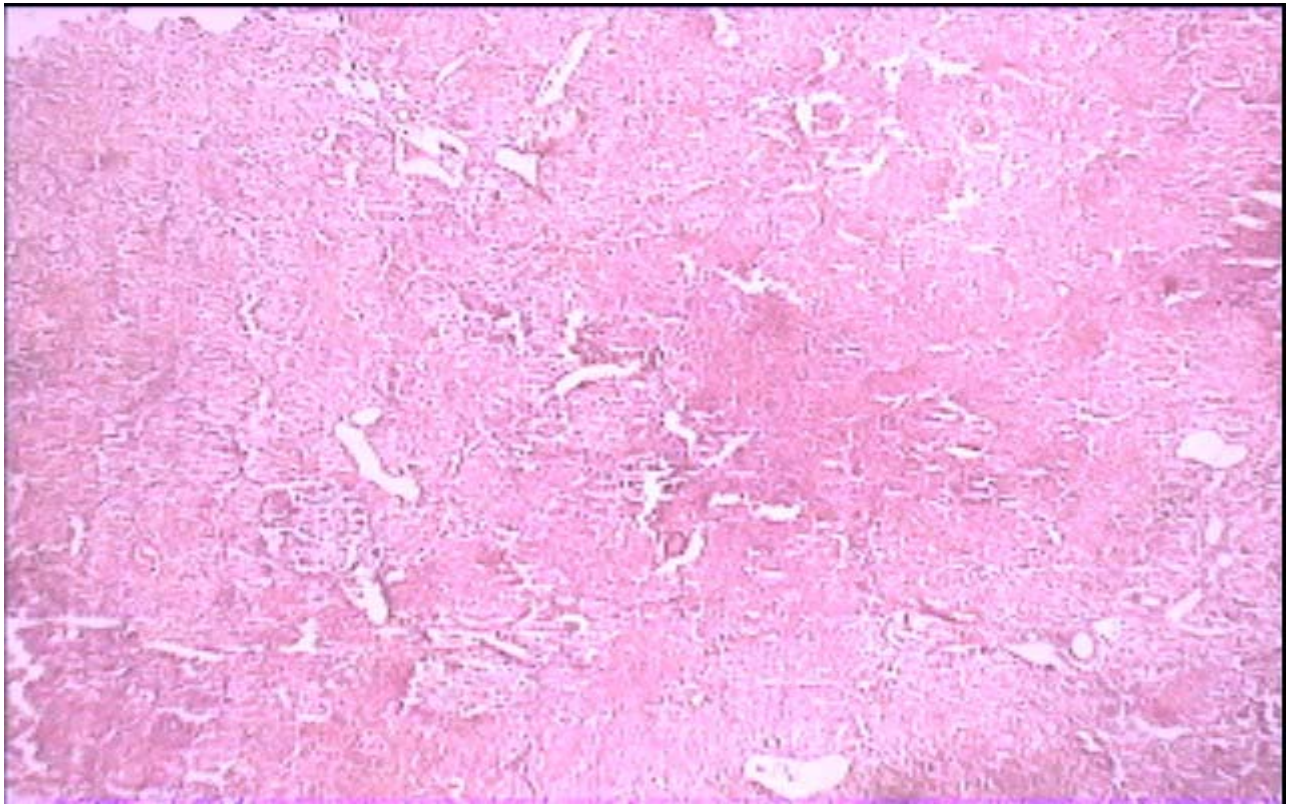
- Epitheloid cells with peripheral lymphocytes



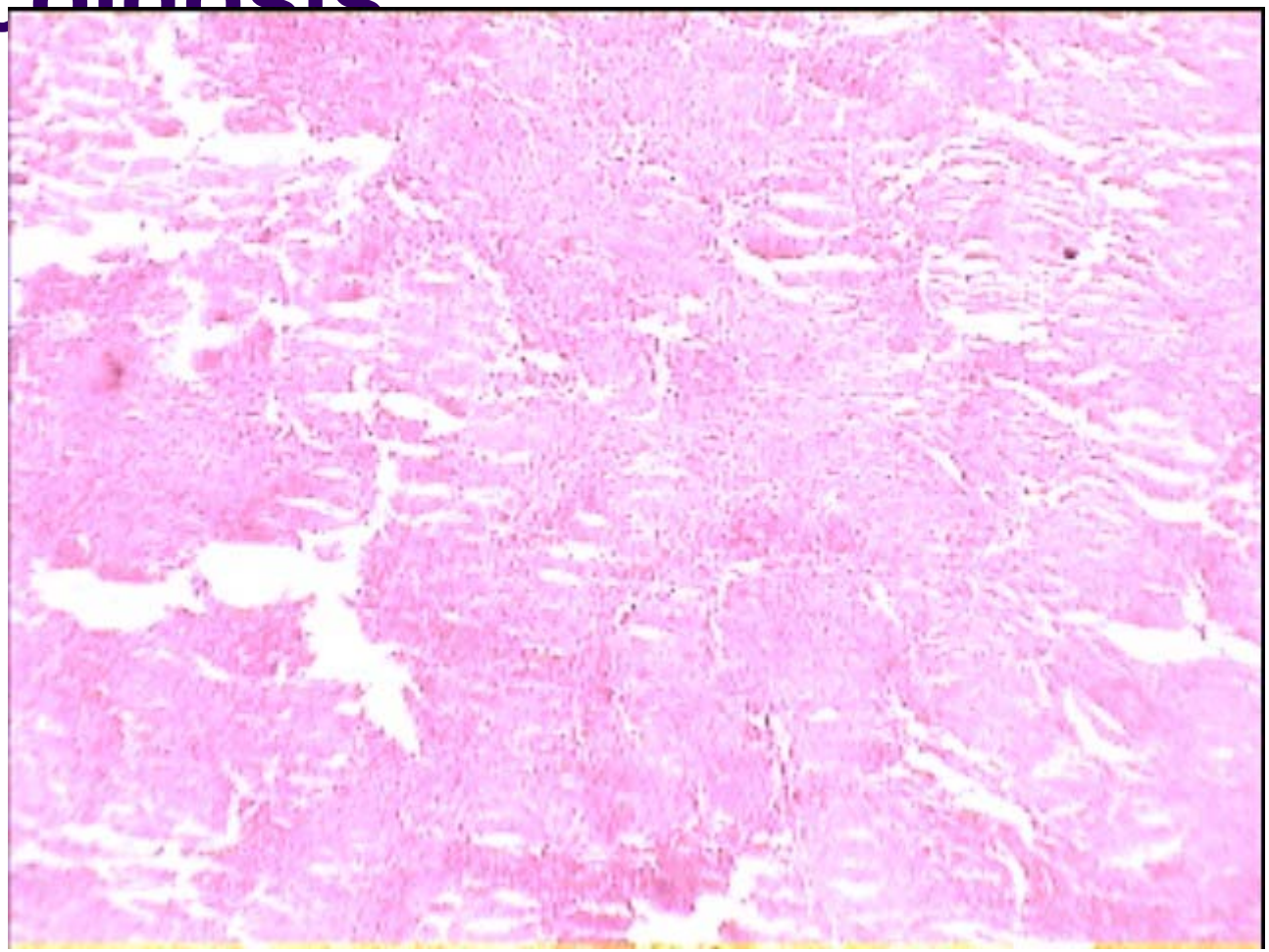
- High power showing nature of Epitheloid cells – Large cells with abundant cytoplasm, indistinct cell boundaries forming syncytium and oval vesicular nucleus
- Also seen is a giant cell – large cell with numerous nuclei pushed towards periphery- Langhans giant cell



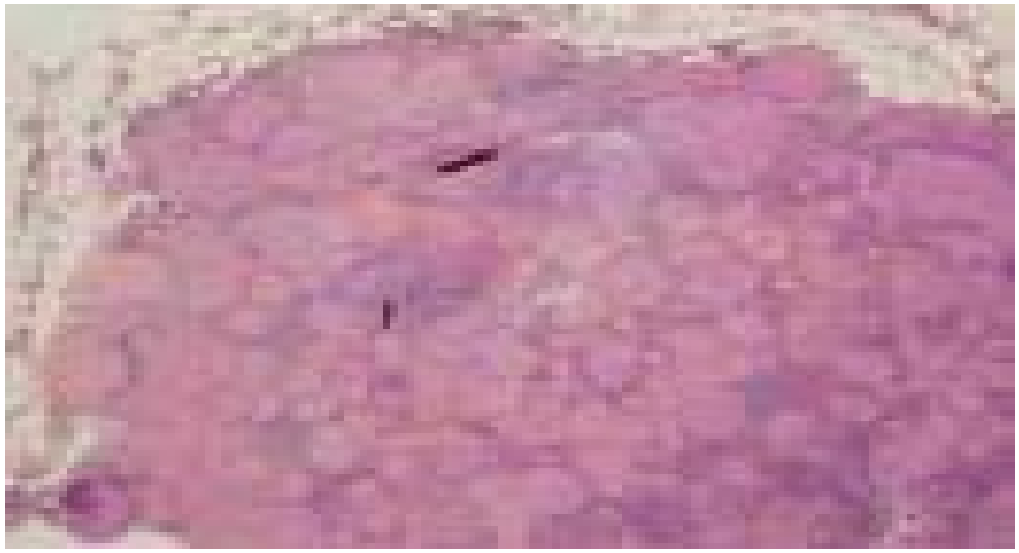
Sarcoidosis



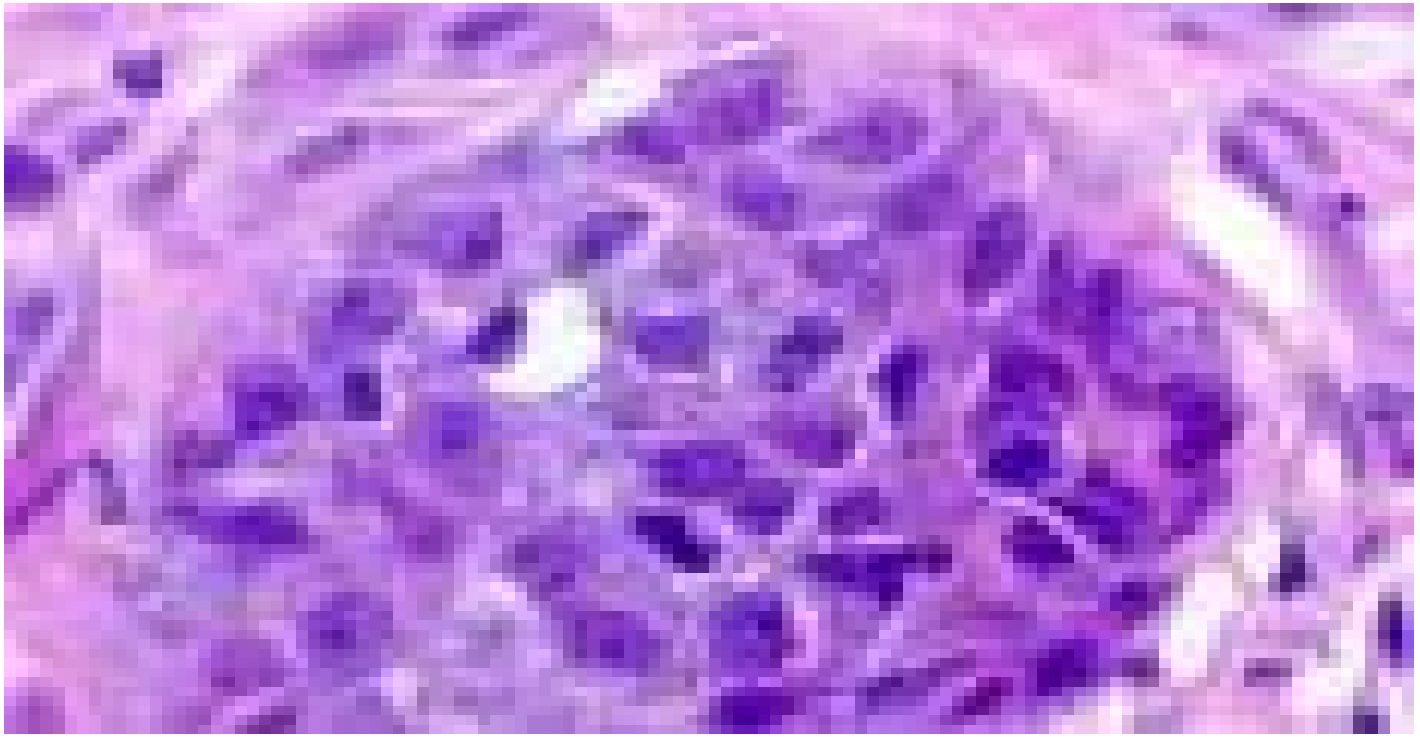
Sarcoidosis



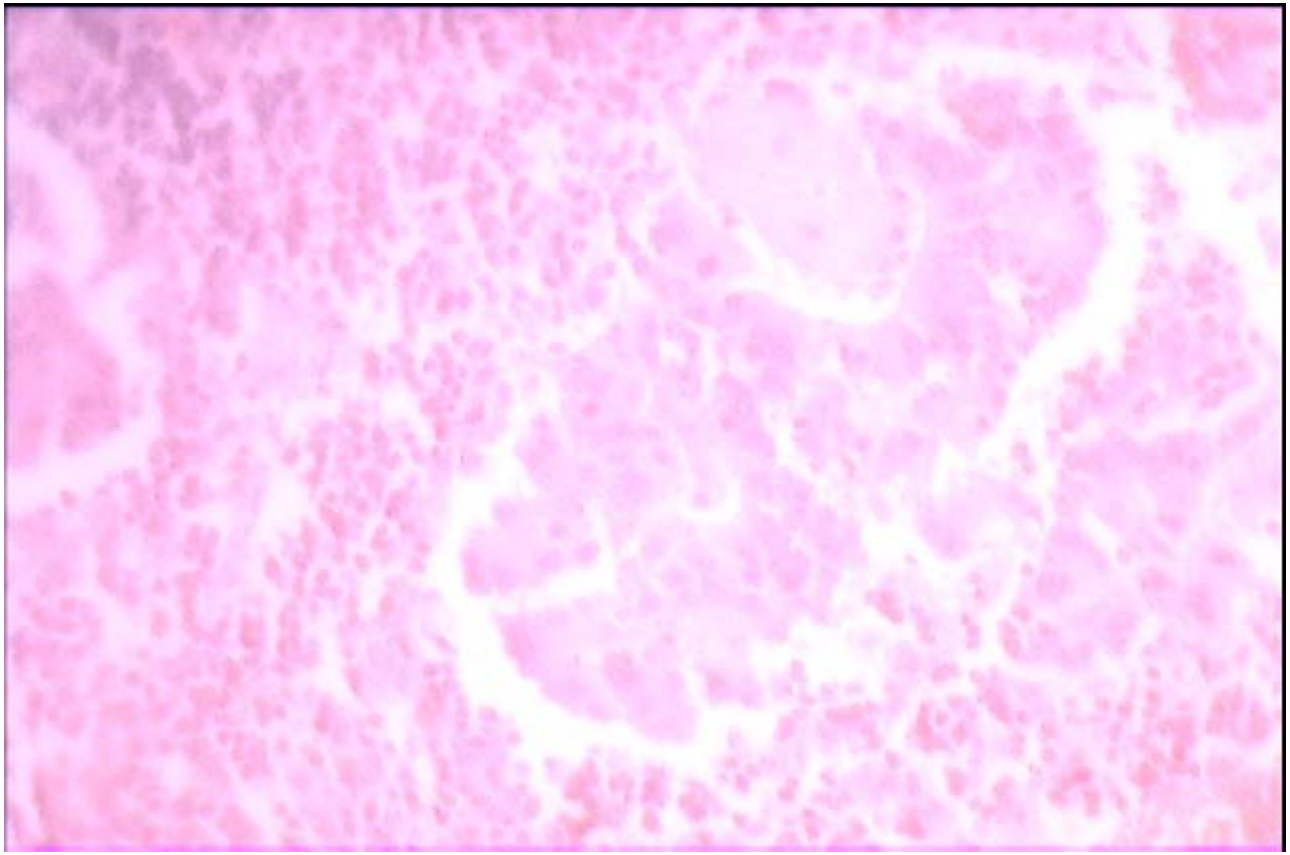
granulomas



Metastatic Deposits

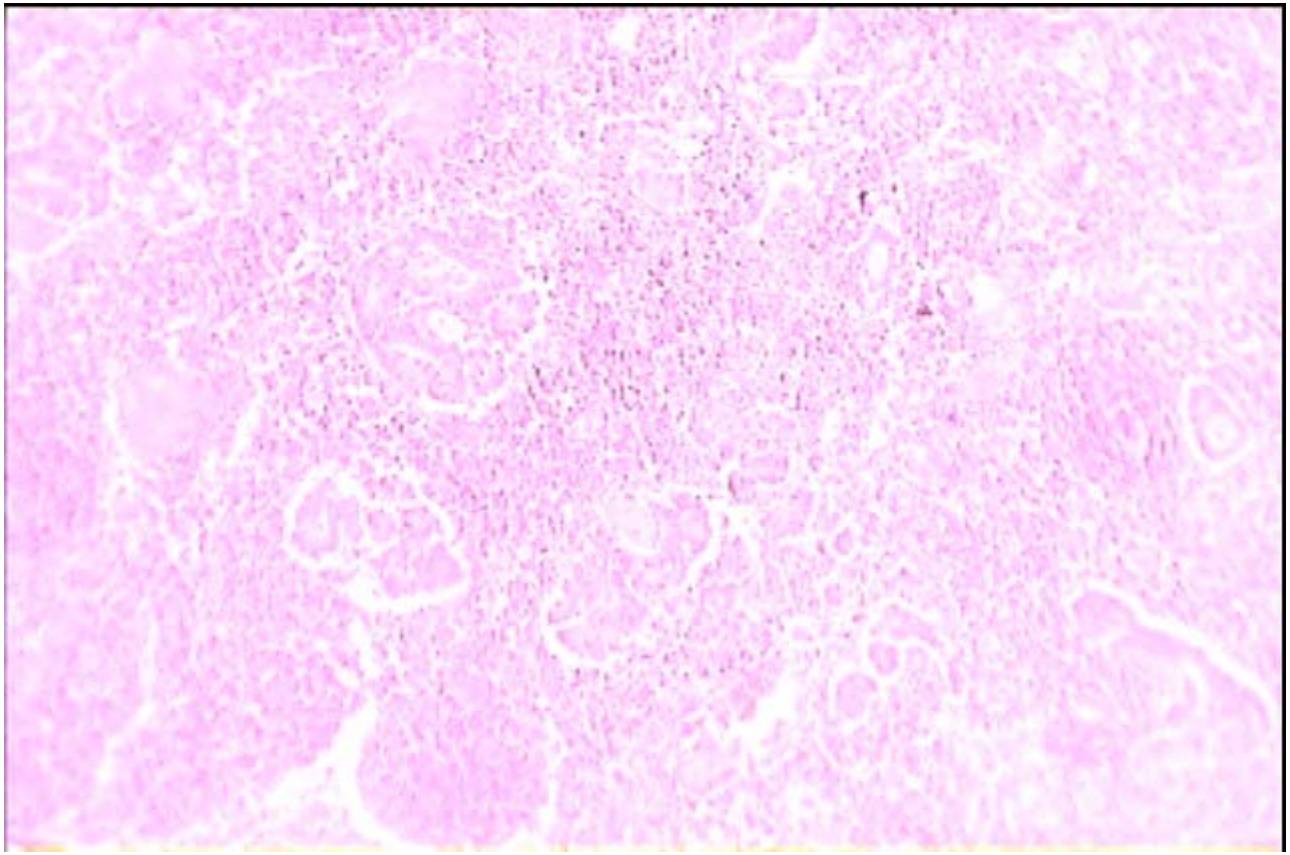


Deposits



deposits

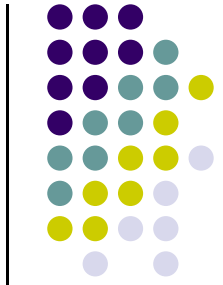
Deposits



Spleen

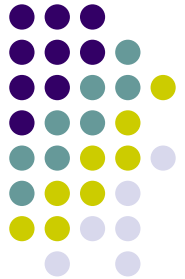
- Secondary lymphoid organ
- Fetal life: Active blood formation
- Adults –mainly filtration organ
- filters bl.stream of all foreign elements

Functions of spleen



- Filtration of blood –Culling destroys abn RBC + old RBCs
- -Pitting –removal of granular inclusions from RBCs eg HJ bodies
- Immune function: contributes to CMI & humoral immunity
- Source of lymphoreticular cells & sometimes haematopoeitic cells

Splenomegaly



- Inflammatory
- Congestive states
- Hyperplastic
- Infiltrative & storage disorders
- Cysts & neoplasms

Mild enlargement (upto 5 cm) occurs in

CVC of spleen in CHF,

acute malaria

typhoid fever

bacterial endocarditis,

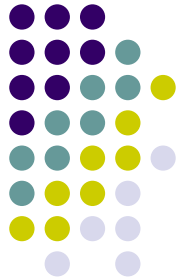
SLE, rheumatoid arthritis and thalassaemia minor.

Moderate enlargement (upto umbilicus) occurs in hepatitis,cirrhosis, lymphomas, infectious mononucleosis, haemolytic anaemia, splenic abscesses and amyloidosis

- *Massive enlargement (below umbilicus)* occurs in CML, myeloid metaplasia with myelofibrosis, storage diseases,

  thalassaemia major, chronic malaria, leishmaniasis and portal vein obstruction.

Splenomegaly



- Inflammatory
- Congestive states
- Hyperplastic
- Infiltrative & storage disorders
- Cysts & neoplasms

Inflammatory

Acute & subacute :

Acute splenic tumor

- septicemia
- typhoid
- IM
- SABE
- Abscess

Chronic

- Ch malaria
- Kala azar
- Syphilis
- TB
- Sarcoid

Congestive splenomegaly

- Cirrhosis
- Portal or splenic V thrombosis
- Cardiac failure

Infiltrative

- Storage of N & abnormal metabolic products

- Gaucher dis
- Neimann pick dis
- Amyloidosis

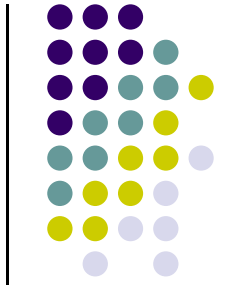
Cysts & Tumors

- True cysts—Parasitic
  - Hydatid
  - Dermoid
  - Lymphangioma
- False Cysts
  - Haemorrhagic
  - Inflammatory
  - Hamartomas

Tumors

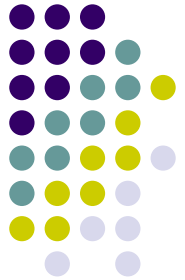
- Leukemia
- Lymphoma
- Histiocytosis
- Metastasis

Massive enlargement of Spleen



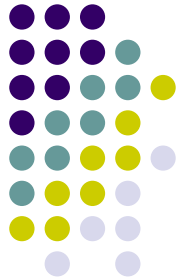
- Chronic Myeloid Leukemia
- CLL
- Lymphomas
- Ch Malaria
- Kala Azar
- CVC Spleen
- Myelofibrosis
- Hydatid cyst
- Gaucher Disease

Acute splenic tumor



- Common in ac. Blood borne systemic infs.
- Reaction can occur to –microbial agents
- products of infl.
- Mild to mod enlargement
- Caused by- true reactive H plasia of myeloid & lymphoid cells
- congestion with RBCs.

Congestive splenomegaly



- CVC spleen –seen in
- Cardiac failure
- Intrahepatic derangement of portal venous drainage
- Obst. To portal/ splenic vein

- All these ult –portal HT

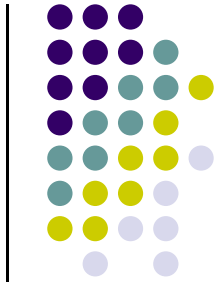
- Gross Marked enlargement
  Firm
  capsule thickened & fibrotic.
- C/S Meaty appearance.
- M/E early cases-red pulp suffused with RBCs

Later more fibrous & cellular

Long standing cases thickening of trabeculae

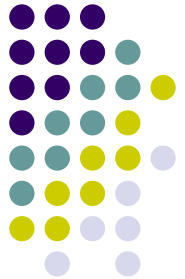
Fibrosis of red pulp

Atrophy of lymphoid tissue



- Gamma gandy bodies –Sidero fibrotic nodules – Ca & haemosiderin deposits over organised Hges.

Infiltrative

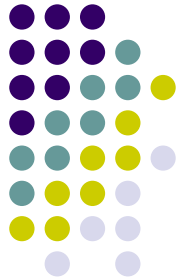


- Excessive storage of n & abn metabolic products
- Gaucher s disease
- Def. of beta glucocerebrosidase
- Glucocerebrosides-not metabolised
- Deposited in HCs.
- Splenic cords & sinusoids infilt. By Gaucher cells.

- Gaucher cell HC with glucocerebroside in cyto.
- Small nu.
- Cyto.-faintly eosino.

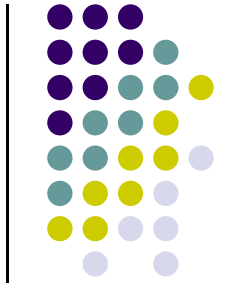
  Striated or fibrillar.

Neimann Pick disease



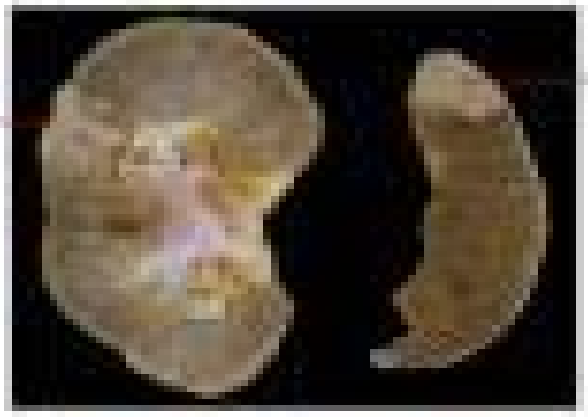
- Accum of sphingo myelins in HCs
- Appear as foam cells having vacuolated cyto.

Splenic Infarcts

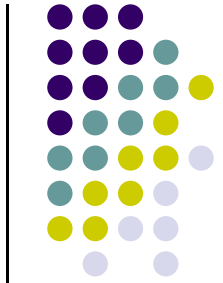


- Fairly common
- Causes Us due to emboli from heart
- local thrombosis
- Infarct –Wedge shaped
- base at periphery.
- H gic first & then pale.
- Later replaced by fibrous tissue-depressed scar.

Infarct



Hypersplenism



- Ill effects produced thru exaggeration of its N functions.
- Criteria for diagnosis—
- 1.haematological findings-Anemia
  Leucopenia
  Thrombocytopenia
  Or a combin.
- 2. Cellular BM
- 3.Splenomegaly
- 4.Improvement after splenectomy

Amyloid Spleen

- Sago spleen –Amyloid limited to follicles
- Lardaceous spleen- Diffuse involv. Of splenic sinuses in form of sheet like waxy deposits.

THYMUS

- The thymus gland is a complex lymphoreticular organ lying within the mediastinum.
- At birth, the gland weighs 10-35 gm and grows in size upto puberty, following which there is progressive involution in the elderly.

In the adult, thymus weighs 5-10 gm.

- The gland consists of right and left encapsulated lobes, joined together by fibrous connective tissue.
- Connective tissue septa pass inwards from the capsule and subdivide the lobe into large number of lobules

- The histologic structure of the lobule shows *outer cortex* and *inner medulla.*
- Both cortex and medulla contain two types of cells:
- epithelial cells : Polygonal in cortex & spindle shaped in medulla
- lymphocytes (thymocytes).

- **Epithelial cells** have elongated cytoplasmic processes forming network in which thymocytes and macrophages are found.
- *Hassall's corpuscles* are distinctive structures within the medulla composed of onion skin-like concentrically arranged

epithelial cells having central area of keratinisation

- **Thymocytes** are predominantly present in the cortex.
- These cells include immature T lymphocytes in the cortex and mature T lymphocytes in the medulla

- Most common primary tumour present in the anterosuperior mediastinum is thymoma.
- Most of the patients are adults.

- Most of the patients are adults.
- In about half the cases, thymoma remains asymptomatic and is accidentally discovered in X-rays.
- Other patients have associated conditions like myasthenia gravis or local symptoms such as cough, dyspnoea and chest pain

thymoma

- ***Grossly,*** the tumour is spherical, measuring 5-10 cm in diameter with an average weight of 150 gm.
- C/S: soft, yellowish, lobulated and may be either homogeneous or contain cysts due to the presence of haemorrhage and necrosis.

- ***Microscopically,*** the tumour has a thick fibrous capsule from which extend collagenous septa into the tumour dividing it into lobules

- **Benign thymoma** is more common.

  It consists of epithelial cells which are similar to the epithelial cells in the medulla of thymus and hence also called as medullary thymoma (spindle shaped) or a mixture of medullary- and cortical-type epithelial cells. There is usually a sparse infiltrate of thymocytes

- **Malignant thymoma** is less common and is further of 2 types:
- *Type 1* is cytologically benign looking but aggressive and invades the mediastinal structures locally.

- *Type 2* is also called thymic carcinoma and has cytologic features of cancer.
- Further subtypes of epithelial malignancy may be squamous cell type (most common) and lymphoepithelial type.