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 follicles
- Surr by paracortex - T cell area
- Medulla - cords & sinuses.
Lymphoid follicles

- 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
Functions of Lymph node

- Immune Response  T cell & B cells
- Involved in both CMI & humoral immunity.
- Active phagocytosis of particulate matter
Causes of LNopathy

- 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
  - Kikuchi's lymphadenitis
  - Angiofollic. 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 by hypertrophied cells.
Chronic Non Specific Lymphadenitis

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

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

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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

- Dilatation or expansion of the sinuses.
- Lining endothelial cells are hypertrophied.
- Sinuses are packed with HCs.
Sinus Histiocytosis
Sinus Histiocytosis
Specific Reactive conditions of LNs
AIDS Related Lymphadenopathy

- Lymph Node changes
- 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, PI cell prolif
- Necrotising Lymphadenitis **Kikuchi**
- young females
- painless LN pathology
- 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 form 90% 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
- Interfollic. Zone –Inc vascularity
- PI 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

- Exclusion in adults & elderly
- C/F fever, anemia (hemolytic), generalized lymphadenopathy, polyclonal hypergammaglobulinemia
- 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
- Should be regarded as **Atypical Immuno proliferative disorder**
- Ranges --- Reactive & revert to clearly neoplastic & aggregative
Sinus Héosis with massive LNopathy

- Rosai Dorfman Disease
- C/F-painles B/L LN opathy
- Uus 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 & neck region
- Skin CNS GIT etc
- BM & Spleen –spared.
- Etio. Not known
- ? Inf viral
- ? immunological 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 haematopoietic 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

- 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.
- Hemic 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 up to 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.