• ALT is Liver specific (AA-I-608)

• Calcification usually starts from mitochondria in all body tissues except: TBM (Tubular BM) in Kidney

• For determination of sex, a common place for obtaining smear is the buccal mucosa and then study it for X or Y chromosome. Although any nucleated cell could be used for the same purpose.

• Connexin --> Gap junction

• Thymus-
  - Thymoma- *Epithelial cells found (Lymphocytic infiltrates are not neoplastic)
  - Neoplastic variety- T cell lymphoblastic lymphoma

• Alcoholic hyaline inclusions (Mallory bodies) are irregular eosinophilic hyaline inclusions that are found within the cytoplasm of hepatocytes and are *composed of prekeratin intermediate filaments. Hepatic *stellate cell promote collagen deposition and fibrosis in alcoholic cirrhosis.

• Best place to sample cells for hematopoiesis- sternum. Also done in ribs, vertebra, iliac crest, skull and proximal femur.

• Rheumatoid Factors- Antibodies of IgG, IgA, IgM (m/c) class directed against Fc fragment of Immunoglobulin G*.

• Arteriosclerosis-
  - Hyperplastic arteriosclerosis- Malignant hypertension (>200/140) refers to dramatic elevations in systolic and diastolic blood pressure often resulting in early death from cerebral and brainstem hemorrhages. Pathologically the renal vessels demonstrate a concentric obliteration of arterioles by an increase in smooth muscle cells and protein deposition in laminar configuration that includes fibrin material, which leads to total and subtotal occlusion of vessels.
  - Hyaline arteriosclerosis- Seen in DM is presumably caused by leakage of plasma components across the endothelium with or without hypertension.
  - Monckeberg's arteriosclerosis- Medial calcific sclerosis is characterised by dystrophic calcification in the tunica media of muscular arteries. There is no narrowing of the lumen of the affected vessels.

• thymic corpuscle- small spherical bodies of keratinized and usually squamous epithelial cells arranged in a concentric pattern around clusters of degenerating lymphocytes, eosinophils, and macrophages; found in the medulla of the lobules of the thymus.

• Infarcts are localised areas of ischaemic coagulative necrosis.
  - White infarcts- also referred to as pale or anemic infarcts are usually the result of arterial occlusion. They are found in solid organs such as the heart, spleen, and kidneys
  - Red or hemorrhagic- These infarcts are may result from either arterial or venous occlusion. These infarcts are hemorrhagic because there is bleeding into the necrotic area from the adjacent arteries and veins that remain patent. Hemorrhagic infarcts also occur in in organs in which the venous outflow is obstructed (venous occlusion). Examples of this include torsion of testis or ovary. In testis twisting of spermatic cord occludes the venous outflow, but the arterial inflow remains patent because these arterial blood vessels have much thicker walls. This results in *venous infarction. Testicular torsion is usually the result of physical trauma in an individual with a predisposing abnormality such as abnormal development of the gubernaculum testis.

• In congenital dystrophic epidermolysis bullosa defect is seen in--> Collagen type 7 (GT-86 Q-30)

• Thymus contains T-lymphocytes-
  - Mature- Medulla
  - Immature- Cortex
○ Appearance of lymphoid follicles with germinal centers is abnormal and found in thymic hyperplasia.

<table>
<thead>
<tr>
<th>Red Pulp of spleen</th>
<th>White pulp of spleen</th>
</tr>
</thead>
<tbody>
<tr>
<td>The red pulp is the primary site of splenic filtration. Within the red pulp, macrophages destroy old or bad RBC's, microorganisms, and remove particles of debris. The venous sinuses* are distensible areas capable of storing more than 300mL of blood with the help of the red pulp.</td>
<td>The splenic white pulp is primarily lymphoid tissue and is the primary site of immune and phagocytic action. Here, the spleen processes foreign antigen and produces specific immunoglobulin M. It contains Periarteriolar lymphoid sheath, T cells, Antigen Presenting Cells.</td>
</tr>
</tbody>
</table>

- Enlarged tonsil from a 9-yr old girl reveal an increased number of reactive follicles containing germinal centers with proliferating B lymphocytes--> follicular hyperplasia

<table>
<thead>
<tr>
<th>Fibroadenoma</th>
<th>Less cellular stroma and mitotic figures</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phylloides tumor</td>
<td>Increased cellular stroma and mitotic figures</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Heinz bodies</th>
<th>Seen in patients with oxidant stress. These are precipitates of denatured hemoglobin on red blood cells. Common in G6PD patients.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Howell jolly bodies</td>
<td>Seen in asplenia. They are nuclear fragments.</td>
</tr>
</tbody>
</table>

**SARCOIDOSIS-**:
- Schaumann calcifications
- Asteroid bodies/ACE increase/ Anergy
- Respiratory complications/Restrictive lung disease/ Restrictive cardiomyopathy/ Renal calculi
- Calcium increase in serum and urine/ CD4 helper cells
- Ocular lesions
- Immune mediated noncaseating granulomas/ [Ig] increase
- Diabetes insipidus/D vit. increase/ Dyspnea Osteopathy
- Skin (Subcutaneous nodules, erythema nodosum)
- Interstitial lung fibrosis/ IL-1
- Seventh C.N. palsy

- **Migratory thrombophlebitis or Trousseau’s syndrome** - It is most strongly associated with Pancreatic carcinoma, but most commonly seen in patients with Lung Carcinoma, as latter is much more common.
- Carcinomas having tendency to metastasize to bone--> Particular Tumours Love Killing Bone-
  - Prostate
  - Thyroid
  - Lung
  - Kidney
  - Breast

- The essential feature of HCM is massive myocardial hypertrophy, usually without ventricular dilation. The classic pattern is disproportionate thickening of the ventricular septum as compared with the free wall of the left ventricle (with a ratio greater than 1 : 3), frequently termed asymmetric septal hypertrophy. The most important histologic features of the myocardium in HCM are-
  1. Extensive myocyte hypertrophy to a degree unusual in other conditions
2. Haphazard disarray of bundles of myocytes, individual myocytes, and contractile elements in sarcomeres within cells (termed myofiber disarray)

3. Interstitial and replacement fibrosis

- **Flocculent densities** are characteristically seen in irreversibly injured myocardial cells that undergo reperfusion soon after ischaemic injury.

- **Growth factors**
  - Basic FGF (Fibroblast growth Factor) - capable of inducing all* of the stages of angiogenesis
  - Epidermal Growth Factor Family - includes EGF and TNF-alpha. These substances can cause proliferation of many types of epithelial cells and fibroblasts.
  - PDGF - cause migration and proliferation of fibroblasts, smooth muscle cells and monocytes

- Liver does not produce following clotting factors 3, 4, 6, 8, 11. Stored blood is deficient in factor 5 and 8. Only way to correct deficiency of **factor V** is addition of thrombin.

- Bleeding due to Warfarin --> Give **FFP** (contains Prothrombin and factors 7, 9, 10 all vit. K dependent clotting factors)

<table>
<thead>
<tr>
<th>t(11;14)</th>
<th>Mantle cell lymphoma(Cyclin D-1--&gt;Most specific; CD5 +ve, CD23-ve)</th>
</tr>
</thead>
<tbody>
<tr>
<td>t(14;18)</td>
<td>Follicular lymphoma(Bcl-2 +ve)</td>
</tr>
<tr>
<td>t(11;18)</td>
<td>Marginal zone lymphoma(Maltoma)---&gt;only lymphoma cured by antibiotics, a/w H.Pylori</td>
</tr>
<tr>
<td>8;14;10;14</td>
<td>T-cell acute lymphoblastic leukaemia</td>
</tr>
<tr>
<td>11;22</td>
<td>Ewing sarcoma</td>
</tr>
<tr>
<td>15;17</td>
<td>APML</td>
</tr>
<tr>
<td>15;30</td>
<td>Hodgkin’s lymphoma</td>
</tr>
<tr>
<td>t(8;14)</td>
<td>Burkitt's lymphoma</td>
</tr>
</tbody>
</table>

Q. Ectrodactyly is an autosomal dominant trait that causes missing middle fingers. A grandfather and grandson both have ectrodactyly, but the intervening father has normal hands by X-Ray. Which term applies to this family-

1. Incomplete penetrance -----------ans
2. New mutation
3. Variable expressibility
4. Germinal mosaicism

**Discussion**

<table>
<thead>
<tr>
<th>Incomplete penetrance*</th>
<th>Normal individual who is known to have an abnormal allele for autosomal dominant trait</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Variable expressibility</strong></td>
<td>Refers to family members who exhibit the signs of autosomal dominant disorder that vary in severity. When this severity seems to worsen with progressive generations, it is called anticipation</td>
</tr>
</tbody>
</table>

**Q-All of the following chromosomal disorders are associated with advanced maternal age except:**

1. Down’s syndrome
2. Cri-du-chat syndrome----ans
3. Edward’s syndrome
4. Patau syndrome

**Explanation:** Cri-du-chat syndrome occurs d/t deletion of short arm of fifth chromosome. Others are due to nondisjunction and have strong association with increased maternal age

**Some conditions with very high (>100 mm/hr) ESR:**

1. Multiple myeloma
2. Connective tissue disorders - SLE, RA and other autoimmune diseases
3. Tuberculosis
4. Malignancies
5. Severe anemia*

**Some conditions with low ESR:**
1. Polycythemia
2. Severe Leukocytosis
3. Sickle cell disease (anemia)*
4. Hereditary spherocytosis
5. Congestive cardiac failure
6. Corticosteroid use
7. Hypofibrinogenemia

*Note that sickle cell anemia and spherocytosis have low ESR unlike other anemias. This is due to reduced rouleaux formation owing to the abnormally shaped RBCs in this condition.*

Q. Low Complement Levels associated with a/e-
1. Lupus Nephritis
2. PSGN
3. Mesangiocapillary GN
4. HUS

Explanation- Lupus Nephritis, PSGN, Mesangiocapillary GN are all associated with low complement levels. Decreased complement levels not seen in HUS. HUS is characterized by microangiopathic hemolytic anaemia, thrombocytopenia & renal failure due to microangiopathy.

**Low Complement Levels are seen in-**
1. Idiopathic glomerulonephritis
2. Crescentic glomerulonephritis
3. MPGN (Membranoproliferative glomerulonephritis)
4. Lupus nephritis
5. Poststaphylococcal glomerulonephritis

**Normal Complement Levels-**
1. Anti GBM disease- Good posture’s syndrome
2. Immune complex mediated- IgA nephropathy/*Henoch schonlein purpura.
3. Pauci immune glomerulonephritis- Wegner’s granulomatoses/Microscopic polyarteritis nodosa

(Ref Harrison)

- **Carcinoid tumor**- Nerve-endocrine secretory granules present. **Cardiac** lesion in the carcinoid syndrome are typically right-sided due to inactivation of serotonin and bradykinin in the blood during passage through the lungs by MAO enzyme. However bronchial carcinoids can produce left sided cardiac valvular lesion.
- **Function of Cadherin** is cell adhesion. It’s mutation leads to cancer associated with loss of cell adhesion
- **Kaposi’s sarcoma** is the most common neoplasm in AIDS patients. It’s cells express markers of both endothelial and smooth muscle lineages. CNS is the most common extranodal site affected.
- **Anti-alpha-fodrin antibody**- detected in patients with **Sjogren Syndrome** and in those with SLE. It seems to be more valuable for diagnosis of Sjogren syndrome than anti-SS-A(Ro)
- **Oncosis:** Pre-lethal changes preceding necrotic cell death.

**Turner’s syndrome-**
Persons whose red blood cells are "Duffy negative" (a genetic trait) are resistant to *P. Vivax* infection...

<table>
<thead>
<tr>
<th>Anti-inflammatory Mediators</th>
<th>IL-4,10,13, PGE-2; TNF-beta</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pro-inflammatory Mediators</td>
<td>IL-1(alpha+beta),2,6,8; IFN; TNF-alpha; PAF</td>
</tr>
</tbody>
</table>

92. Nasal and URT involvement is seen in-
   1. SLE
   2. Churg strauss syndrome
   3. Wegener’s granulomatatis---------ans
   4. PSS

64. Reversible ischemic change includes all except-
   1. Cell swelling
   2. Lysosomal swelling-------------ans
   3. Blebs at cell surface
   4. Dissociation of polysomes to monosomes

65. All are irreversible ischemic changes except-
   1. Mitochondrial vacuolisation
   2. Plasma membrane damage
   3. Massive calcium influx into cell after reperfusion
   4. Detachment of ribosomes from endoplasmic reticulum---------ans

Discussion-
Irreversible damage-
   1. Damage of cell membrane
   2. Ruptured lysosome-->Inflammation
   3. Mitochondrial dysfunction--> Calcium influx--> Flocculant deposition

66. The earliest change in the nucleus in necrosis-

Madelung deformity (short ulna with V-Shaped angle between distal radius and ulna). Flattening of the medial tibial condyle with enlarged adjacent femoral condyle (blount’s disease). Renal anomalies like horseshoe kidneys.
1. Karyolysis
2. Pyknosis
3. Clumping of chromatin
4. Karyorrhexis

**Discussion**
Remember the order-
1. Pyknosis- d/t homogenous condensation of chromatin
2. Karyorrhexis- Fragmentation of nucleus
3. Karyolysis- Dissolution of nucleus

**68. Non-inflammatory oedema will occur when there is-**
1. An increase in intravascular hydrostatic pressure
2. A fall in colloid osmotic pressure of the plasma
3. An impairment in the flow of lymph
4. All

**Discussion**

<table>
<thead>
<tr>
<th>Oedema</th>
<th>Inflammatory---&gt; Exudate</th>
<th>Non-Inflammatory---&gt; Transudate</th>
</tr>
</thead>
</table>

**69. Granular pick coagulate in alveolar spaces is seen in-**
1. Respiratory distress syndrome
2. Hyaline membrane disease
3. Mycoplasma infection
4. Pulmonary edema

**Discussion**
Respiratory distress syndrome- DAD (Diffuse alveolar damage)
Granular pink coagulate-
- PCP*(Pneumocytosis carinii--->Characteristic)
- Amyloidosis
- PAP
also found in RDS

**70. Lines of Zahn in a thrombus are found when thrombus forms in-**
1. Peripheral veins
2. Heart
3. Brain
4. Muscular arteries

**Discussion**
Alternate layers of platelets and RBCs(Wiki- Alternating pale layers of platelets mixed with fibrin and darker layer containing red blood cells. Their presence implies thrombosis at a site of rapid blood flow that happened before death.)

**71. Diagnosis of Neimann-Pick is by-**
1. Sphingomyelinase estimation in blood
2. Biopsy of liver and bone marrow
3. Detection of specific mutations
4. Sphingomyelin accumulation in skin fibroblasts

**Discussion**
NP Cells (Niemann Pick Cells) in BM. Stained by Oil-Red-O. They classically have a small nucleus. Abundant vacuolated* cytoplasm d/t lipid/fat. Such cells are also seen in Cholesterol ester deficiency/lipoprotein lipase deficiency. Now sphingomyelin estimation is done in lekocytes for diagnosis(Not availbe in India).

**72. Gaucher’s cells have which feature-**
1. Rarely vacuolated
2. Cytoplasm resembles crumpled tissue paper
3. In cerebral Gaucher’s disease gaucher cells are found in Virchow-Robin space
4. All--------ans

Discussion:

**Gaucher's disease**: M/C lysosomal storage disease* with *beta-glucosidase deficiency. Types-
- **Non-neuropathic**: Type 1 (M/C)
- **Neuropathic**: Type 2,3 (Incompatible with life)

C/F:
- Hematology-
  - Anemia
  - Thrombocytopenia
- Organomegaly
- Bony lesion
- Histopathology-
  - Gaucher's cell-
    - Blend nucleus
    - *Wrinkled paper like cytoplasm*
    - PAS +ve
    - Oil-Red-O negative

Enzyme replacement therapy available

---

73. Most common nuclear fluorescence in SLE is-
1. Peripheral
2. Speckled--------ans
3. Diffuse
4. Nucleolar

Discussion-
SLE--> ANA test--> Fluorescent ANA test-
- Speckled pattern: SLE(M/C)
- Nucleolar pattern: Scleroderma

74. In SLE, histological change in skin is-
1. Subepithelial thickening
2. Pickle cell layer proliferation
3. Liquefactive degeneration of basal layer--------ans
4. Parakeratosis and hyperkeratosis

76. Centriacinar emphysema usually affects-
1. Upper lobes--------ans
2. Middle lobe
3. Lower lobes
4. Lower zone of upper lobes

77. Earliest feature of chronic bronchitis is-
1. Hypersecretion of mucus in large airways--------ans
2. Goblet cell increase in small airways
3. Obstruction of small bronchi and bronchioles
4. Hypertrophy of bronchial smooth muscle

Discussion-
Reids index = Gland/Wall
- Gland- Mucous gland layer thickness
- Wall-thickness of wall b/n epithelium and cartilage

Chronic bronchitis = 0.6-0.7 (N-0.4)

78. All are found in bronchial asthma except-
1. Hypertrophy of bronchial muscle wall
2. Thickened basement membrane of bronchial infiltration
3. Eosinophilic infiltration in bronchial walls
4. Submucosal glands hyperplasia----------ans

Discussion: Hypertrophy not hyperplasia

Airway remodelling in bronchial asthma-
- Increased airway wall thickness that involves both smooth muscle and collagen tissue
- Increased mucous glands and mucus production
- Increased vascularity, or blood supply, in the airways

79. The characteristic morphology of PAP is-
1. Patchy pneumonitis
2. Localised inflammation of alveolar septa------ans (PAP- Pulmonary Alveolar Proteinosis)
3. Suppurative destruction of lung parenchyma
4. Lipid laden foamy macrophages

80. The most common chronic occupational disease is-
1. Coal worker’s pneumoconiosis
2. Silicosis----------ans
3. Asbestosis
4. Berylliosis

Discussion: Silicosis- Fibrotic nodule, Onion scarring appearance

81. The most prevalent asbestos is-
1. Crocidolite
2. Chrysotile--------ans
3. Amosite
4. Atinolyte

Discussion: Mesothelioma- Most specific marker--> WT-1(from Wilm's Tumor). But most commonly done--> Calretinin

82. Acanthocytic RBC’s or “burr cells” are found in-
1. Disaccharidase deficiency
2. Irritable bowel disease
3. Postinfections sprue
4. Abetalipoproteinemia--------ans

83. Creeping fat or mesenteric fat wrapped around the bowel surface is found in-
1. Terminal ileitis----------------ans(Crohn’s d/e = terminal ileitis)
2. Ulcerative colitis
3. Pseudomembranous colitis
4. Irritable bowel syndrome

84. All are true about Crohn’s disease except-
1. Skip lesions
2. String sign
3. Creeping fat
4. Pseudopolyps----------ans

85. Which worm usually causes appendicular obstruction-
1. Ascaris
2. T solium
3. Ancylostoma
4. Enterobios vermicularis----------ans

86. Argenataffin cells are stained by-
1. MASSON FONTANA--------ans
2. VON KOSSA
3. GMELIN’S
4. PAS
Discussion - Silver stain--> Reduced--> Black colour deposition

**87. MAST CELLS ARE STRAINED BY-**
1. SAFFRANIN--------ans
2. PTAH
3. ATPASE
4. LUXOL FAST BLUE

**88. Nissl substance is stained by-**
1. ORANGE G
2. PAS
3. H&E
4. TOLUIDINE BLUE----------ans
Discussion - Nissl substance--> large granular body found in neuron
Metachromatic stains- Stains which impart different colour than original colour eg. Toluidine
Frozen biopsy--> Stained with--> Rapid H&E or Toluidine

**98. Grimelius stains-**
1. Endocrine granules-------ans
2. Glycogen
3. Fibrin
4. Mast cells
Discussion - Was used earlier to diagnose pheochromocytoma.

**99. Swank Davenport stains-**
1. Nerve cells and axons
2. Neuroglial cells
3. Myelin degenerate--------ans
4. Paneth cell granules

**Q. PAS stains the following except-**
a. glycogen
b. lipids....................ans
c. fungal cell wall
d. basement memb of bacteria
Discussion- it is given as [d] in AA, however dey hv also given dat pas is useful in
basement memb,& they hv given glycolipids n nt simply lipids--> galat diya hai .. aa
main..

**92. Heyman’s antigen is involved in immune complex formation in-**
1. Crescentic GN
2. Mesangioproliferative GN
3. Membranous GN-----------ans
4. Lipoid nephrosis

**93. In RPGN, accumulation of cells to form crescents occurs in-**
1. Basement membrane of glomerulus
2. Subendothelial
3. Bowman’s space--------ans
4. Mesangium

**94. In sickle cell nephropathy which is NOT found-**
1. Hematuria
2. Polyuria
3. Glycosuria---ans
4. Proteinuria

95. The most common salivary tumour is-
1. Pleomorphic adenoma--ans(Biphasic tumor--> Epithelial, Myoepithelial)
2. Monomorphic adenoma
3. Mucoepidermoid tumours
4. Acinic cell tumours

- Angiosarcoma-
  - Histopathology-
    - Prominence of endothelial cells
    - Arborising vascalate path
  - Marker- CD34

- Synovial sarcoma- It has no business with synovium(misnomer). Seen in youg adults and arises from proximal part of jt. Biphasic tumor (Sarcomatous/Carcinomatous)

<table>
<thead>
<tr>
<th>Rhabdomyosarcoma</th>
<th>Embryonal(M/C)</th>
<th>Alveolar</th>
<th>Pleomorphic</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2-3 yrs of age involves Head/Trunk region or female groin--&gt; Bobroids tumor*--Bunch of grapes. Nicholson's cambium layer is diagnostic.</td>
<td>2nd decade. Hand/leg involvement. Classical MSRCT. Tumor giant cells seen</td>
<td>Elderly. Poor prognosis</td>
</tr>
<tr>
<td>Stain- PAS, PTAH</td>
<td></td>
<td></td>
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<tr>
<td>Markers- Desmin, Myogenin</td>
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