PATHOLOGY FLASH POINTS

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1- multiple myeloma, markedly increased level of serum globulins. The renal failure from light chains deposited in the kidneys, and the increased risk for encapsulated bacterial infections is typical. The lytic bone lesions are collections of plasma cells.
2- A monomorphous proliferation of intermediate-sized lymphoid cells is seen with Burkitt lymphoma, often with accompanying macrophages producing starry sky appearance.
3- Atypical lymphocytosis in a young person suggests infectious mononucleosis, which is an infection.
4- The translocation 'Philadelphia chromosome' 9;22 is typical of chronic myelogenous leukemia. With CML, the LAP score is low.
5- Decreased serum B12 or folate leads to a megaloblastic anemia, which may lead to anemia or occasionally leukopenia.
6- The high WBC count with left shift (but no blasts) and the high LAP score are consistent with a leukemoid reaction. The term 'leukemoid' is used because there is a markedly increased WBC count, with immature forms of WBCs, similar to leukemia. In his case, the infection is driving the leukocytosis.
7- Increased numbers of red blood cell inclusions such as nuclear fragments (Howell-Jolly bodies) and degenerated hemoglobin (Heinz bodies) appear following splenectomy.
8- The presence of nucleated RBCs relates more to abnormalities of bone marrow, such as myelophthisic processes. The spleen can be a site for extramedullary hematopoiesis.
9- hodgkin lymphoma is a malignant lymphoproliferative process, usually without tenderness in enlarged lymph nodes. Though many reactive lymphoid cells are present, there should be diagnostic Reed-Sternberg cells
10- high WBC count with the blasts and Auer rods are very characteristic for an acute myelogenous leukemia (AML) that can occur in young adults.
11- CLL is a disease of older adults, and the leukemic cells resemble small mature lymphocytes.
12- Lymphoblastic leukemia/lymphoma is typically a disease of young children, and the lymphoblasts do not have Auer rods or cytoplasmic granules positive for myeloperoxidase.
13- Fanconi anemia is a rare congenital anemia starting in childhood, with underlying defects in DNA repair, with associated anomalies such as organ hypoplasias.
14- The small lymphocytic lymphoma (SLL) component of CLL may involve the lymph nodes, but there will be a diffuse pattern of infiltration by small lymphocytes, not follicles. His normal WBC count is not consistent with CLL.
15- Myeloproliferative disorders, and myelofibrosis in particular, are known to cause massive splenomegaly.
16- As hematopoiesis is reduced in the marrow, it moves elsewhere (extramedullary hematopoiesis) such as the spleen.
17- Metastatic tumor involving marrow, or marrow fibrosis, is a 'myelophthisic' process that reduces normal hematopoiesis and leads to a peripheral 'leukoerythroblastic' picture with immature RBC's

and WBC's in the peripheral blood, as seen here with nucleated RBCs and white cells even more immature than bands (metamyelocytes, myelocytes) on the smear.

18- DIC may produce a microangiopathic hemolytic anemia, but there is widespread activation of coagulation so that microthrombi are formed, with markedly elevated prothrombin time, partial thromboplastin time, and D-dimer. DIC typically follows as a complication of a serious disease process, not de novo.

19- The pentad of fever, mental changes, renal failure, thrombocytopenia, and microangiopathic hemolytic anemia is characteristic of TTP

20- Unlike CML, the leukocyte alkaline phosphatase is high with a leukemoid reaction.

21- With AML there are many immature myeloid forms, particularly blasts.

22- Immature lymphoid forms-- lymphoblasts --are seen with ALL.

23- Most of the circulating cells in CLL are small, mature lymphocytes with CLL. Most persons with CLL are older adults. CLL can have a tissue component called small lymphocytic lymphoma (SLL) with the same small lymphocytes infiltrating organs such as liver and spleen.

24- Reduced beta-globin chain synthesis from beta-thalassemia leads to RBC microcytosis, hypochromia, ineffective erythropoiesis, and excessive iron absorption. There is chronic anemia, because the major hemoglobin A1 is produced insufficiently. The nature of the mutation, typically affecting RNA transcript production, determines the severity of the disease.

25- chronic myelogenous leukemia (CML) which is a form of chronic myeloproliferative process in which one or more cell lines (myeloid, erythroid, megakaryocytic) becomes increased. This is usually driven by an acquired cytogenetic abnormality in a stem cell line, a t(9;22) that forms a BCR-ABL fusion gene with tyrosine kinase activation. Unlike acute myelogenous leukemia, blasts are uncommon in CML.

26- Myelodysplastic syndromes can precede development of AML, as can some cases of myeloproliferative disorders, paroxysmal nocturnal hemoglobinuria, and chemotherapeutic regimens.

27- Auer rods are formed of the cytoplasmic granules of the myeloid blasts of acute myelogenous leukemia (AML) and are a typical finding with AML

28- Wiskott Aldrich syndrome This syndrome is accompanied by thrombocytopenia along with immunodeficiency.

29- Glucose-6-phosphate dehydrogenase (G6PD) deficiency, which can result in a hemolytic anemia on exposure to oxidizing agents including certain drugs such as antimalarials. This is an X-linked disorder. The Heinz bodies within the RBCs are formed from denatured hemoglobin.

30- By teenage years,in sickle cell anemia persons with Hgb SS have undergone 'autosplenectomy' from the multiple infarctions, leading to a very small spleen.

31- The increased MCV seen in macrocytic anemia such as a megaloblastic anemia that can also have hypersegmented PMN's from delayed maturation and reduced numbers of nuclear divisions.

32- Fragmented RBC's, called schistocytes, are indicative of a microangiopathic hemolytic anemia such as DIC.

33- The most probable cause of a hypochromic, microcytic anemia in an older man is iron deficiency anemia.

34- CD10 is a marker for lymphocytes , seen in ALL and CLL.

35- Aplastic anemia : peripheral cytopenias for marked hypocellularity of the bone marrow with aplastic anemia. The spleen is of normal size with aplastic anemia. The 'aplasia' here refers to the three major cell lines: myeloid, and megakaryocytic as well as erythroid.
36- Mycosis fungoides (with no relationship to fungi) is one form of cutaneous T-cell lymphoma. The abnormal CD4 cell proliferation is infiltrating the skin and producing erythroderma. If these neoplastic cells circulate, it is known as Sezary syndrome.
37- Myelodysplastic syndromes lead to an increase in immature myeloid cells in tissues and peripheral blood. There is risk for progression to an acute myeloid leukemia.
38- Increased osmotic fragility is a feature of hereditary spherocytosis.
39- positive Coombs test suggests immune-related hemolysis.
40- Lead poisoning leads to basophilic stippling of rbcs
41- Tear drop shaped RBCs are most likely to appear with myelophthisic processes such as myelofibrosis.
42- Hemophilia A and B are disorders of coagulation factors VIII and IX respectively, and are X-linked recessive. Though it is possible that the girl could have inherited an abnormal gene on each X chromosome, or have random X inactivation, this is unlikely. Hemophilia often leads to hemarthroses with joint problems. Platelets are not affected.
43- CD19 CD 20 are marker for B-lymphocytes.
44- CD68 is a marker for macrophages.
45- CD55 and CD59 are RBC markers
46- Schilling test is done for vitamin b12 deficiency.
47- Reed Sternberg cell markers are CD15 CD 30.
48- LCA CD45 is white blood cell marker.
49- Ham's test is positive in PNH.
50- Reed Sternberg cells are hall mark of Hodgkins lymphoma.

VESSELS KEY POINTS

1. Hemangiomas are very common vascular tumors characterized by increased numbers of normal or abnormal vessels filled with blood. Histologically, these are unencapsulated aggregates of closely packed, thin-walled capillaries, usually blood-filled and lined by flattened endothelium.The vessels are separated by scant connective tissue stroma.
2. Hemangiomas are of three types : capillary , cavernous and lobular capillary hemangiomas.
3. Lobular capillary hemangiomas are traditionally known as pyogenic granuloma , but remember this is a misnomer because u will not find any typical epithelioid cell granulomas in this lesion.
4. Cavernous hemangiomas are a component of von Hippel-Lindau disease , occurring within the cerebellum, brain stem, or retina, along with similar angiomatous lesions or cystic neoplasms in the pancreas and liver.
5. Immunomarkers for vascular lesions / tumors are : CD31 , CD 34 And von willebrand factor.
6. Lymphangiomas are the benign lymphatic analogues of blood vessel hemangiomas. They contain lymph instead of rbc's in their lumina.Two types : capillary and cavernous (cystic hygromas)

7. Glomus tumors are benign, exquisitely painful tumors arising from modified smooth muscle cells of the glomus body, a specialized arteriovenous structure involved in thermoregulation.
8. Glomur tumors show aggregates, nests, and masses of specialized glomus cells intimately associated with branching vascular channels, all within a connective tissue stroma. Individual tumor cells are small, uniform, and round or cuboidal, with scant cytoplasm and ultrastructural features akin to smooth muscle cells
9. Borderline vascular tumors are : Kaposi's sarcoma and hemangioendothelioma.
10. 4 types of kaposi's sarcoma include : Chronic KS (also called classic or European KS) , Lymphadenopathic KS (also called African or endemic KS), Transplant-associated KS and AIDS-associated (epidemic) KS.
11. human herpesvirus-8 (HHV-8) or KS-associated herpesvirus (KSHV) is associated with kapos's sarcoma.
12. Kaposi's sarcoma classical morphological three stages are recognized: Famous 3 P's :patch, plaque, and nodule.
13. Angiosarcoma etiology includes : Thorotrast , polyvinyl chloride and post mastectomy lymphangiosarcoma.
14. In angiosarcoma , all degrees of differentiation can be seen, from plump, anaplastic but recognizable endothelial cells producing vascular channels to wildly undifferentiated tumors with a solid spindle cell appearance and without definite blood vessels
15. Hemangiopericytoma : They consist of numerous branching capillary channels and gaping sinusoidal spaces enclosed within nests of spindle-shaped to round cells with staghorn vasculature.
16. The main immunological mechanisms that initiate noninfectious vasculitis are (1) immune complex deposition, (2) antineutrophil cytoplasmic antibodies, and (3) anti–endothelial cell antibodies.
17. ANCAs are a heterogeneous group of autoantibodies directed against constituents (mainly enzymes) of neutrophil primary granules, monocyte lysosomes, and endothelial cells. These were previously classified according to their intracellular distribution, either cytoplasmic (c-ANCA) or perinuclear (p-ANCA).
18. Although not entirely specific, their new namea are now PR3-ANCAs (c- anca) typical of Wegener granulomatosis and MPO-ANCAs (p anca) are characteristic of microscopic polyangiitis and Churg-Strauss syndrome
19. Giant cell large vessel vasculitis : nodular intimal thickening (with occasional thromboses) that reduces the lumenal diameter. Classic lesions exhibit medial granulomatous inflammation that leads to elastic lamina fragmentation with an infiltrate of T cells (CD4+> CD8+) and macrophages.
20. Polyarteritis nodosa (PAN) is a systemic vasculitis of small or medium-sized muscular arteries (but not arterioles, capillaries, or venules), typically involving renal and visceral vessels but sparing the pulmonary circulation.
21. Fibrinioid necrosis is seen in PAN.
22. Kawasaki disease, is an acute febrile, usually self-limited illness of infancy and childhood (80% are younger than 4 years) associated with an arteritis affecting large to medium-sized, and even small, vessels
23. Kawasaki disease is also known as mucocutaneous lymph node syndrome, because it presents with conjunctival and oral erythema and erosion, edema of the hands and feet, erythema of the palms and soles, a desquamative rash, and cervical lymph node enlargement

24. In some areas (typically post-capillary venules) Of microscopic polyangiitis , only infiltrating and fragmenting neutrophils are seen, giving rise to the term leukocytoclastic vasculitis.
25. Churg-Strauss syndrome (also called allergic granulomatosis and angiitis) is a relatively rare (roughly one in a million people) small-vessel necrotizing vasculitis classically associated with asthma, allergic rhinitis, lung infiltrates, peripheral hypereosinophilia, and extravascular necrotizing granulomas.
26. Wegener granulomatosis is a necrotizing vasculitis characterized by a triad of

- Acute necrotizing granulomas of the upper respiratory tract (ear, nose, sinuses, throat) or the lower respiratory tract (lung) or both
- Necrotizing or granulomatous vasculitis affecting small to medium-sized vessels (e.g., capillaries, venules, arterioles, and arteries), most prominent in the lungs and upper airways but affecting other sites as well
- Renal disease in the form of focal necrotizing, often crescentic, glomerulonephritis

27. Limited" forms of Wegener granulomatosis may be restricted to the respiratory tract.
28. An aneurysm is a localized abnormal dilation of a blood vessel or the heart , it can be congenital or acquired.
29. When an aneurysm involves an intact attenuated arterial wall or thinned ventricular wall of the heart, it is called a true aneurysm.
30. false aneurysm (also called pseudo-aneurysm) is a defect in the vascular wall leading to an extravascular hematoma that freely communicates with the intravascular space ("pulsating hematoma").
31. Weak vessel walls due to defective type III collagen synthesis are also a hallmark of the vascular forms of Ehlers-Danlos syndrome
32. The two most important disorders that predispose to aortic aneurysms are atherosclerosis and hypertension
33. Inflammatory AAAs – abdominal aorti aneurysms are characterized by dense periaortic fibrosis containing abundant lymphoplasmacytic inflammation with many macrophages and often giant cells. Their cause is uncertain.
34. Mycotic AAAs are lesions that have become infected by the lodging of circulating microorganisms in the wall, particularly in bacteremia from a primary Salmonella gastroenteritis.
35. Aortic dissection occurs when blood splays apart the laminar planes of the media to form a blood-filled channel within the aortic wall.
36. Hypertension is the major risk factor in aortic dissection
37. Hypertensive vascular changes include : Hyperplastic Arteriolosclerosis. This lesion occurs in severe (malignant) hypertension; vessels exhibit "onion-skin lesions," characterized by concentric, laminated thickening of the walls and luminal narrowing
38. Another hypertensive vascular benign change is Hyaline Arteriolosclerosis. Arterioles show homogeneous, pink hyaline thickening with associated luminal narrowing . These changes stem from plasma protein leakage across injured endothelial cells, and increased smooth muscle cell matrix synthesis in response to chronic hemodynamic stress.
39. VEGF is linked with vasculogenesis and B-fgf is linked with angionesis.

40. Mönckeberg medial sclerosis is characterized by calcific deposits in muscular arteries in persons typically older than age 50.
41. Atherosclerosis word derived from Greek root words for “gruel” and “hardening.
42. Atherosclerosis is characterized by intimal lesions called atheromas (also called atheromatous or atherosclerotic plaques) that protrude into vessel lumens
43. Endothelial cell injury is the cornerstone of the responseto-injury hypothesis in atherosclerosis.
44. Intimal smooth muscle cell proliferation and ECM deposition convert a fatty streak, the earliest lesion, into a mature atheroma and contribute to the progressive growth of atherosclerotic lesions
45. Fatty streaks are the earliest lesions in atherosclerosis.
46. The key processes in atherosclerosis are intimal thickening and lipid accumulation
47. Atherosclerotic plaques have three principal components: (1) cells, including smooth muscle cells, macrophages, and T cells; (2) ECM, including collagen, elastic fibers, and proteoglycans; and (3) intracellular and extracellular lipid
48. Endothelial cells contain Weibel-Palade bodies, intracellular membrane-bound storage organelles for von Willebrand's factor
49. Glomus tumors are seen mainly in fingers.
50. Hemangiomas remember are the most common benign tumors in children (In adults we all know Lipoma leads)

MALE GENITAL PATHOLOGY FLASH POINTS

1.Undescended testis are characterized by arrested germ cell development associated with marked hyalinization and thickening of the basement membrane of the spermatic tubules.Such testis carries risk of developing testicular neoplasia.

2. Testicular neoplasms span an amazing gamut of anatomic types and can be divided into two major categories: germ cell tumors (95%) and sex cord-stromal tumors

3. Germ cell tumors are subdivided into seminomas and nonseminomas.

4.Most testicular germ cell tumors originate from a precursor lesion called intratubular germ cell neoplasia
(ITGCN).

5. Seminomas are the most common type of germ cell tumor, making up about 50% of these tumors. The peak incidence is the third decade and they almost never occur in infants.

6. The typical seminoma has a homogeneous, graywhite, lobulated cut surface, usually devoid of hemorrhage or necrosis

7. The classic seminoma cell is large and round to polyhedral and has a distinct cell membrane; clear or
watery-appearing cytoplasm; and a large, central nucleus with one or two prominent nucleoli

8. the typical seminoma is composed of sheetsof uniform cells divided into poorly demarcated lobules by delicatefibrous septa containing a lymphocytic infiltrate

9. Approximately 15% of seminomas contain syncytiotrophoblasts.In this subset of patients, serum human chorionicgonadotropin (HCG) levels are elevated

10. Spermatocytic seminomas contain three cell populations, all intermixed: (1) medium-sized cells, the most numerous, containing a roundnucleus and eosinophilic cytoplasm; (2) smaller cells with a narrow rim of eosinophilic cytoplasm resembling secondary spermatocytes; and (3) scattered giant cells, either uninucleate or multinucleate.

11. Histologically the cells In Embryonal carcinoma grow in alveolar or tubular patterns, sometimes with papillary convolutions.More undifferentiated lesions may display sheets of cells.

12.IN approximately 50% of YOLK SAC tumors, structures resembling endodermal sinuses (Schiller-Duval bodies) may be seen; these consist of a mesodermal core with a central capillary and a visceral and parietal layer of cells resembling primitive glomeruli.

13.Seminomas are radiation sensitive.

14.Yolk sac tumor shows raised alpha fetoprotein levels.

15. By immunohistochemistry, seminoma cells stain positively for KIT, (regardless of KIT mutational status), OCT4, and placental alkaline phosphatase (PLAP).

16. Teratomas are composed of a heterogeneous, helter-skelter collection of differentiated cells or organoid structures, such as neural tissue, muscle bundles, islands of cartilage, clusters of squamous epithelium, structures reminiscent of thyroid gland, bronchial or bronchiolar epithelium, and bits of intestinal wall or brain substance, all embedded in a fibrous or myxoid stroma

17. In the child, differentiated mature teratomas usually follow a benign course. In the postpubertal male all teratomas are regarded as malignant, capable of metastatic behavior

18. Because of differing behaviors, tumors of the testis are segregated clinically into two broad categories:
seminoma and nonseminomatous germ cell tumors(NSGCTs).

19.Seminomatous tumors are radiation sensitive and less aggressive and non seminomatous are aggressive , but respond to chemotherapy.

20. Biologic tumor markers include HCG, AFP, and lactate dehydrogenase, which are valuable in the diagnosis and management of testicular cancer.

21. Seminomas remain confined to the testis for a long time and spread mainly to paraaortic nodes-distant spread is rare. Nonseminomatous tumors tend to spread earlier, by both lymphatics and blood vessels.

22. Non-Hodgkin lymphoma is the most common testicular tumor in men older than 60 years.

23. Tumors of Leydig cells are particularly interesting, because they may elaborate androgens and in some cases both androgens and estrogens, and even corticosteroids.

24.Crystalloids of Reinke are seen in a leydig cell tumor.

25. HCG is produced by syncytiotrophoblasts and is always elevated in patients with choriocarcinomas or seminomas containing syncytiotrophoblasts. AFP is elevated when there is a yolk sac tumor component.

26. BPH is the most common benign prostatic disease in men older than age 50 years. It results from nodular hyperplasia of prostatic stromal and epithelial cells and often leads to urinary obstruction.

27. DHT is the main hormone in BPH , It binds to the nuclear androgen receptor (AR) present in both stromal and epithelial prostate cells. DHT is more potent than testosterone because it has a higheraffinity for AR and forms a more stable complex with the receptor.

28. Microscopically, in BPH glandular proliferation takes the form of aggregations of small to large to cystically dilated glands lined by two layers of cells, an inner columnar layer and an outer layer of cuboidal or flattened epithelium.Plus the fibromuscular component is increased.

29. BPH most commonly affects the inner periurethral zone of the prostate, producing nodules that compress the prostatic urethra.

30.Adenocarcinoma of prostate is the most common malignant neoplasm of prostate.

31.Gleason socring is done to grade a prostatic adenocarcinoma.

32. Prostate cancer is graded using the Gleason system, which stratifies prostate cancer into five grades on the basis of glandular patterns of differentiation.

33. Grade 1 represents the most well differentiated tumors, in which the neoplastic glands are uniform and round in appearance and are packed into well-circumscribed nodules

34. In contrast,grade 5 tumors show no glandular differentiation, with tumor cells infiltrating the stroma in the form of cords, sheets, and nests

35. Measurement of serum PSA levels is widely used to assist with the diagnosis and management of prostate cancer.

36.In BPH Double lining epithelial cell layers are seen however in prostate carcinoma basal layer is lost.

LIVER PATHOLOGY FLASH POINTS

1. Acetaminophen is the leading cause of drug-induced acute liver failure.
2. There are three distinctive, albeit overlapping, forms of alcoholic liver disease: (1) hepatic steatosis (fatty liver disease), (2) alcoholic hepatitis, and (3) cirrhosis
3. The fatty change is completely reversible if there is abstention from further intake of alcohol.
4. Mallory bodies in alcoholic hepatitis.Scattered hepatocytes accumulate tangled skeins of cytokeratin intermediate filaments such as cytokeratin 8 and 18, in complex with other proteins such as ubiquitin. Mallory bodies are visible as eosinophilic cytoplasmic clumps in hepatocytes.
5. The manifestations of alcoholic cirrhosis are similar to those of other forms of cirrhosis
6. NAFLD is a group of conditions that have in common the presence of hepatic steatosis (fatty liver), in individuals who do not consume alcohol, or do so in very small quantities (less than 20 g of ethanol/week.NAFLD stands for non alcoholic fatty liver disease.
7. α_1-Antitrypsin deficiency is characterized by the presence of round-to-oval cytoplasmic globular inclusions in hepatocytes, which in routine H&E stains are acidophilic and indistinctly demarcated from the surrounding cytoplasm.Its one of the causes of cirrhosis.
8. Cirrhosis is a condition in which liver lobular architecture is replaced by nodules of variable sizes containing benign hepatoccytes.These nodules are regenerative hepatocytes.
9. Chronic persistent hepatitis means inflammation limited to the portal triads with no acitivity.
10. Chronic active hepatitis means inflammation extending from portal triads into the parenchyma with piecemeal necrosis.
11. piecemeal necrosis is necrosis of the hepatocytes when inflammatory cells cross the limiting plates around the triads and damage the hepatocytes.it shows activity

12. Knodell score and metavir score are used to grade hepatic acitivity in patients with chronic hepatitis.Portal inflammation , piece meal necrosis , lobular necrosis and fibrosis are the main pillars looked to grade such activity.
13. three disorders of intrahepatic bile ducts include: secondary biliary cirrhosis, primary biliary cirrhosis, and primary sclerosing cholangitis
14. The primary feature of this disease is a nonsuppurative, inflammatory destruction of medium-sized intrahepatic bile ducts. It is accompanied by portal inflammation, scarring, and eventual development of cirrhosis and liver failure
15. Antimitochondrial antibodies are present in 90% to 95% of patients. They are highly characteristic of PBC and an essential element for diagnosis, together with the elevation of alkaline phosphatase and γ-glutamyltransferase, which are markers of cholestasis.
16. Right-sided cardiac decompensation leads to passive congestion of the liver
17. Cavernous hemangiomas, blood vessel tumors identical to those occurring elsewhere , are the most common benign liver tumors
18. Benign neoplasms developing from hepatocytes are called hepatic adenomas or liver cell adenomas. Although they may occur in men, hepatic adenomas most frequently occur in young women who have used oral contraceptives;
19. Malignant tumors occurring in the liver can be primary or metastatic
20. Most primary liver cancers arise from hepatocytes and are termed hepatocellular carcinoma (HCC). Much less common are carcinomas of bile duct origin, cholangiocarcinomas.
21. Angiosarcoma of the liver resembles those occurring elsewhere. The primary liver form is of interest because of its association with exposure to vinyl chloride, arsenic, or Thorotrast
22. Hepatoblastoma is the most common liver tumor of young childhood , epithelial and mixed epithelial and mesenchymal types.
23. Four major etiologic factors associated with HCC have been established: chronic viral infection (HBV, HCV), chronic alcoholism, non-alcoholic steatohepatitis (NASH), and food contaminants (primarily aflatoxins).
24. HCC may appear grossly as (1) a unifocal (usually large) mass (2) multifocal, widely distributed nodules of variable size; or (3) a diffusely infiltrative cancer, permeating widely and sometimes involving the entire liver.
25. All patterns of HCCs have a strong propensity for invasion of vascular structures
26. HCCs range from well-differentiated to highly anaplastic undifferentiated lesions. In well-differentiated and moderately differentiated tumors, cells that are recognizable as hepatocytic in origin are disposed either in a trabecular pattern (recapitulating liver cell plates)
27. A distinctive variant of HCC is the fibrolamellar carcinoma , better prognosis than conventional type.
28. Elevated levels of serum α-fetoprotein are found in 50% of persons with HCC
29. Cholangiocarcinoma, the second most common hepatic malignant tumor after HCC, is a malignancy of the biliary tree, arising from bile ducts within and outside of the liver
30. The extrahepatic forms of cholangiocarcinoma include perihilar tumors known as Klatskin tumors, which are located at the junction of the right and left hepatic ducts forming the common hepatic duct, and distal bile duct tumors
31. The liver and lungs share the dubious distinction of being the visceral organs that are most often involved in the metastatic spread of cancers. Although the most common primary sources producing hepatic metastases are those of the colon, breast, lung, and pancreas, any cancer in any site of the body may spread to the liver, including leukemias, melanomas, and lymphomas
32. There are two main types of gallstones. In the West, about 90% are cholesterol stones, containing more than 50% of crystalline cholesterol monohydrate. The rest are pigment stones composed predominantly of bilirubin calcium salts.

33. On histologic examination of chronic cholecystitis the degree of inflammation is variable. In the mildest cases, only scattered lymphocytes, plasma cells, and macrophages are found in the mucosa and in the subserosal fibrous tissue. In more advanced cases there is marked subepithelial and subserosal fibrosis, accompanied by mononuclear cell infiltration.
34. Carcinoma of the gallbladder is the most common malignancy of the extrahepatic biliary tract
35. special stains done for liver include : PAS , PAS-D ,trichrome stain and Reticulin stain.
36. For iron , perls Prussian blue stain is used.

KIDNEY / BLADDER PATHOLOGY FLASH POINTS

1.Special stains for kidney include PAS and methenamine silver to look for thickening of basement membranes of capilliries.

2. The classic diagnostic picture of acute post streptococcal GN is one of enlarged, hypercellular glomeruli . The hypercellularity is caused by (1) infiltration by leukocytes, both neutrophils and monocytes; (2) proliferation of endothelial and mesangial cells; and (3) in severe cases by crescent formation.

3. By immunofluorescence microscopy, there are granular deposits of IgG, IgM, and C3 in the mesangium and along the GBM.

4. The most common histologic picture in Rapidly progressive GN is the presence of crescents in most of the glomeruli (crescentic glomerulonephritis

5. The first type of RPGN is anti-GBM antibody–induced disease, characterized by linear deposits of IgG and, in many cases, C3 in the GBM that are visualized by immunofluorescence

6. The second type of RPGN is the result of immune complex deposition. It can be a complication of any of the immune complex nephritides, including postinfectious glomerulonephritis, lupus nephritis, IgA nephropathy, and HenochSchönlein purpura

7. The third type of RPGN, also called pauci-immune type, is defined by the lack of anti-GBM antibodies or immune complexes by immunofluorescence and electron microscopy. Most patients with this type of RPGN have circulating antineutrophil cytoplasmic antibodies (ANCAs

8.Membranous GN shows silver spikes on special silver stain on diffuse uniform thickened basement membranes of capilliries.

9. Minimal changes”s most characteristic feature is its usually dramatic response to corticosteroid therapy.

10.Focal segmental GN : this lesion is characterized by sclerosis of some, but not all, glomeruli (thus, it is focal); and in the affected glomeruli, only a portion of the capillary tuft is involved (thus, it is segmental

11. Type I MPGN (the great majority of cases) is characterized by the presence of discrete subendothelial electron-dense deposits

12. In dense-deposit disease (type II MPGN) ,a relatively rare entity, the lamina densa of the GBM is transformed into an irregular, ribbon-like, extremely electron-dense structure due to the deposition of dense material of unknown composition in the GBM proper

13. Alport syndrome, when fully developed, is manifest by hematuria with progression to chronic renal failure, accompanied by nerve deafness and various eye disorders, including lens dislocation, posterior cataracts, and corneal dystrophy.

14. Chronic glomerulonephritis is best considered a pool of end-stage glomerular disease fed by several streams of specific types of glomerulonephritis

15. The morphologic changes in the DIABETIC GLOMERULOPATHY - glomeruli include (1) capillary basement membrane thickening, (2) diffuse mesangial sclerosis, and (3) nodular glomerulosclerosis.

16.Diabetic glomerulopathy shows kimmelsteil Wilson nodules , capsular drops and fibrin caps.

17.Special stain to confirm amyloid is CONGO RED.

18.Trichrome stain is done to show fibrosis in glomeruli.

19.Renal biopsy shows classically 4 components :

Glomeruli

Tubules

Interstitium

Blood vessels

20. AKI, a term increasingly favored over the often synonymously used terms acute tubular necrosis (ATN) and acute tubular injury, is a clinicopathologic entity characterized clinically by acute diminution of renal function and often, but not invariably, morphologic evidence of tubular injury

21. By far the most common causes in UTI is Escherichia coli, followed by Proteus, Klebsiella, and Enterobacter. Streptococcus faecalis, also of enteric origin, staphylococci, and virtually every other bacterial and fungal agent can also cause lower urinary tract and renal infection

22. The hallmarks of acute pyelonephritis are patchy interstitial suppurative inflammation, intratubular aggregates of neutrophils, and tubular necrosis.

23. Three complications of acute pyelonephritis are encountered in special circumstances: papillary necrosis , pyonephrosis and perinephric abscess.

24. The hallmarks of chronic pyelonephritis are coarse, discrete, corticomedullary scars overlying dilated, blunted, or deformed calyces, and flattening of the papillae.

25.Microscopically in chronic pyelonephritis , The tubules show atrophy in some areas and hypertrophy or dilation in others. Dilated tubules with flattened epithelium may be filled with colloid casts (thyroidization). There are varying degrees of chronic interstitial inflammation and fibrosis in the cortex and medulla.

26. Xanthogranulomatous pyelonephritis is an unusual and relatively rare form of chronic pyelonephritis characterized by accumulation of foamy macrophages intermingled with plasma cells, lymphocytes, polymorphonuclear leukocytes, and occasional giant cells

27. This is a form of chronic renal disease caused by excessive intake of analgesic mixtures and characterized morphologically by chronic tubulointerstitial nephritis and renal papillary necrosis

28. The main cause of renal dysfunction in multiple myeloma is related to Bence Jones (light-chain) proteinuria. Renal failure correlates well with the presence and amount of such proteinuria and is uncommon in its absence

29. On histologic examination of benign nephrosclerosis , there is narrowing of the lumens of arterioles and small arteries, caused by thickening and hyalinization of the walls (hyaline arteriolosclerosis)

30.Onion skinning is seen in malignant nephrosclerosis.

31. Autosomal-dominant (adult) polycystic kidney disease (ADPKD) is a hereditary disorder characterized by multiple expanding cysts of both kidneys that ultimately destroy the renal parenchyma and cause renal failure

32. There are four main types of calculi: (1) calcium stones (about 70%), composed largely of calcium oxalate or calcium oxalate mixed with calcium phosphate; (2) another 15% are so-called triple stones or struvite stones, composed of magnesium ammonium phosphate; (3) 5% to 10% are uric acid stones; and (4) 1% to 2% are made up of cysteine

33.Benign renal tumors are oncocytoma , renal adenoma and angiomyolipoma

34. Angiomyolipomas are present in 25% to 50% of patients with tuberous sclerosis, a disease caused by loss-of-function mutations in the TSC1 or TSC2 tumor suppressor genes.

35. Because of their gross yellow color and the resemblance of the tumor cells to clear cells of the adrenal cortex, Renal cell carcinoma were at one time called hypernephroma

36. Clear cell carcinoma is the most common type, accounting for 70% to 80% of renal cell cancers. The tumors are made up of cells with clear or granular cytoplasm bcos of lipids and glycogen and are nonpapillary.associated with VHL gene.

37.Papillary carcinoma is associated with MET gene.show foam cells in papillary cores and psammoma bodies at times.

38. Papillary carcinomas are the most common type of renal cancer in patients who develop dialysis-associated cystic disease.

39. Chromophobe renal carcinoma is made up of pale eosinophilic cells, often with a perinuclear halo, arranged in solid sheets with a concentration of the largest cells around blood vessels

40. Collecting duct carcinoma is a rare variant showing irregular channels lined by highly atypical epithelium with a hobnail pattern

41. The three classic diagnostic features of renal cell carcinoma are costovertebral pain, palpable mass, and hematuria, but these are seen in only 10% of cases

42.Paraneoplastic syndromes of RCC are : polycythemia, hypercalcemia, hypertension, hepatic dysfunction, feminization or masculinization, Cushing syndrome, eosinophilia, leukemoid reactions, and amyloidosis.

43.Schistosoma haematobium is associated with squamous cell carcinoma of urinary bladder.

44. Malakoplakia is a peculiar pattern of vesical inflammatory reaction characterized macroscopically by soft, yellow, slightly raised mucosal plaques 3 to 4 cm in diameter , and histologically by infiltration with large, foamy macrophages mixed with occasional multinucleate giant cells and interspersed lymphocytes

45.Urinary bladder WHO grading of urinary bladder tumors ;

Papillomas , Urothelial neoplasm of low malignant potential

Papillary urothelial carcinoma, grade 1

Papillary urothelial carcinoma, grade 2

Papillary urothelial carcinoma, grade 3

46.Staging of urinary bladder carcinoma important thing is PTa – no invasion . PT1 – Lamina propria invasion PT2 – Muscle invasion.

HEAD & NECK FLASH POINTS

1. Histologically, Peripheral giant cell granulomas are distinct. Peripheral giant-cell granuloma is made up of a striking aggregation of multinucleate, foreign body–like giant cells separated by a fibroangiomatous stroma.
2. The term *leukoplakia* is defined by the World Health Organization as "a white patch or plaque that cannot be scraped off and cannot be characterized clinically or pathologically as any other disease."
3. *until it is proved otherwise via histologic evaluation, all leukoplakias must be considered precancerous*.
4. Related to leukoplakia, but much less common and much more ominous, is *erythroplakia*. It represents a red, velvety, possibly eroded area within the oral cavity that usually remains level with or may be slightly depressed in relation to the surrounding mucosa
5. At least 95% of cancers of the head and neck are squamous cell carcinomas (HNSCCs), arising most commonly in the oral cavity
6. Paraganglia are clusters of neuroendocrine cells associated with the sympathetic and parasympathetic nervous systems.Tumors of these cells are called Paragangliomas.
7. While the most common location of paraganglia is within the adrenal medulla, where they give rise to pheochromocytomas
8. Paragangliomas are chiefly composed of nests **(Zellballen)** of round to oval chief cells (neuroectodermal in origin) that are surrounded by delicate vascular septae. The tumor cells contain abundant, clear or granular, eosinophilic cytoplasm and uniform, round to ovoid, sometimes vesicular, nuclei
9. *About 65% to 80% of salivary glands tumors arise within the parotid, 10% in the submandibular gland*, and the remainder in the minor salivary glands, including the sublingual glands
10. Approximately 15% to 30% of tumors in the parotid glands are malignant. In contrast, approximately 40% of submandibular, 50% of minor salivary gland, and 70% to 90% of sublingual tumors are cancerous
11. Because of their remarkable histologic diversity, pleomorphic adenomas have also been called *mixed tumors*. They represent about *60% of tumors in the parotid*
12. Pleomorphic adenomas are benign tumors that consist of a mixture of ductal (epithelial) and myoepithelial cells, and therefore they show both epithelial and mesenchymal differentiation. They reveal epithelial elements dispersed throughout the matrix along with varying degrees of myxoid, hyaline, chondroid (cartilaginous), and even osseous tissue.
13. Warthin tumor is a benign neoplasm with its intimidating histologic name is the second most common salivary gland neoplasm. It arises almost *exclusively in the parotid gland*

14. On microscopic examination warthin tumor shows spaces are lined by a double layer of neoplastic epithelial cells resting on a dense lymphoid stroma sometimes bearing germinal centers . The spaces are frequently narrowed by polypoid projections of the lymphoepithelial elements.
15. Mucoepidermoid carcinoma is a malignant salivary gland tumor.
16. The basic histologic pattern of mucoepidermoid ca is that of cords, sheets, or cystic configurations of squamous, mucous, or intermediate cells. The hybrid cell types often have squamous features, with small to large mucus-filled vacuoles, best seen when highlighted with mucin stains
17. Adenoid cystic carcinoma is another malignant tumor of salivary glands showing small cells having dark, compact nuclei and scant cytoplasm. These cells tend to be disposed in tubular, solid, or cribriform patterns reminiscent of cylindromas arising in the adnexa of the skin. The spaces between the tumor cells are often filled with a hyaline material thought to represent excess basement membrane
18. Acinic cell tumor reveal a variable architecture and cell morphology. Most characteristically, the cells have clear cytoplasm but the cells are sometimes solid and at other times vacuolated. The cells are disposed in sheets or microcystic, glandular, follicular, or papillary patterns

Special stains used :

Mucicarmine : stains mucin as red and rest of the tissues as yellow.

HEART PATHOLOGY FLASH POINTS

1. The most common primary cardiac tumors, in descending order of frequency (overall, including adults and children), are myxomas, fibromas, lipomas, papillary fibroelastomas, rhabdomyomas, angiosarcomas, and other sarcomas.
2. Myxomas are the most common primary tumor of the heart in adults,90% in the atrium.
3. Histologically, myxomas are composed of stellate or globular myxoma cells embedded within an abundant acid mucopolysaccharide ground substance
4. Rhabdomyomas are the most frequent primary tumor of the heart in infants and children
5. The term cardiomyopathy (literally, heart muscle disease) is used to describe heart disease resulting from an abnormality in the myocardium.
6. The term dilated cardiomyopathy (DCM) is applied to a form of cardiomyopathy characterized by progressive cardiac dilation and contractile (systolic) dysfunction, usually with concomitant hypertrophy. It is sometimes called congestive cardiomyopathy.
7. The histologic abnormalities in DCM are nonspecific and usually do not point to a specific etiologic agent
8. Hypertrophic cardiomyopathy (HCM) is characterized by myocardial hypertrophy, poorly compliant left ventricular myocardium leading to abnormal diastolic filling, and in about one third of cases, intermittent ventricular outflow obstruction. It is the leading cause of left ventricular hypertrophy unexplained by other clinical or pathologic causes.
9. Restrictive cardiomyopathy is a disorder characterized by a primary decrease in ventricular compliance, resulting in impaired ventricular filling during diastole.
10. In the United States, viral infections are the most common cause of myocarditis. Coxsackieviruses A and B and other enteroviruses probably account for most of the cases.
11. Nonviral agents are also important causes of infectious myocarditis, particularly the protozoa Trypanosoma cruzi, the agent of Chagas disease.

12. Senile cardiac amyloidosishas a far better prognosis than systemic amyloidosis.
13. Acquired stenoses of the aortic and mitral valves account for approximately two thirds of all cases of valve disease.
14. In mitral valve prolapse (MVP), one or both mitral valve leaflets are “floppy” and prolapse, or balloon back, into the left atrium during systole. The key histologic change in the tissue is called myxomatous degeneration.
15. Uncommonly, MVP is associated with heritable disorders of connective tissue including Marfan syndrome, which is usually caused by mutations in fibrillin-1 (FBN-1)
16. Rheumatic fever (RF) is an acute, immunologically mediated, multisystem inflammatory disease that occurs a few weeks after an episode of group A streptococcal pharyngitis.
17. Distinctive lesions occur in the heart, called Aschoff bodies, which consist of foci of lymphocytes (primarily T cells), occasional plasma cells, and plump activated macrophages called Anitschkow cells (pathognomonic for RF).
18. Aschoff body macrophages have abundant cytoplasm and central round-toovoid nuclei in which the chromatin is disposed in a central, slender, wavy ribbon (hence the designation “caterpillar cells”), and may become multinucleated.
19. Subendocardial lesions, perhaps exacerbated by regurgitant jets, may induce irregular thickenings called MacCallum plaques, usually in the left atrium.
20. Fibrous bridging across the valvular commissures and calcification create “fish mouth” or “buttonhole” stenoses in rheumatic fever.
21. Aschoff bodies are rarely seen in surgical specimens or autopsy tissue from patients with chronic RHD, as a result of the long times between the initial insult and the development of the chronic deformity
22. RF is characterized by a constellation of findings that includes as major manifestations: (1) migratory polyarthritis of the large joints, (2) pancarditis, (3) subcutaneous nodules, (4) erythema marginatum of the skin, and (5) Sydenham chorea, a neurologic disorder with involuntary rapid, purposeless movements.
23. The diagnosis of rheumatic fever is established by the Jones criteria: evidence of a preceding group A streptococcal infection, with the presence of two of the major manifestations listed above or one major and two minor manifestations (nonspecific signs and symptoms that include fever, arthralgia, or elevated blood levels of acute-phase reactants).
24. Infective endocarditis (IE) is a serious infection characterized by colonization or invasion of the heart valves or the mural endocardium by a microbe. This leads to the formation of vegetations composed of thrombotic debris and organisms, often associated with destruction of the underlying cardiac tissues.
25. Traditionally, IE has been classified on clinical grounds into acute and subacute forms
26. Acute infective endocarditis is typically caused by infection of a previously normal heart valve by a highly virulent organism that produces necrotizing, ulcerative, destructive lesions.
27. in subacute IE, the organisms are of lower virulence. These organisms cause insidious infections of deformed valves that are less destructive
28. Endocarditis of native but previously damaged or otherwise abnormal valves is caused most commonly (50% to 60% of cases) by Streptococcus viridans, which is part of the normal flora of the oral cavity.
29. S. aureus is the major offender in intravenous drug abusers with IE
30. The roster of the remaining bacteria of IE includes enterococci and the so-called HACEK group (Haemophilus, Actinobacillus, Cardiobacterium, Eikenella, and Kingella), all commensals in the oral cavity.
31. The hallmark of IE is the presence of friable, bulky, potentially destructive vegetations containing fibrin, inflammatory cells, and bacteria or other organisms on the heart valves
32. Fever is the most consistent sign of IE

33. Noninfected (sterile) vegetations are caused by nonbacterial thrombotic endocarditis and the endocarditis of systemic lupus erythematosus (SLE), called Libman-Sacks endocarditis
34. NBTE is characterized by the deposition of small sterile thrombi on the leaflets of the cardiac valves
35. NBTE is characterized by the deposition of small sterile thrombi on the leaflets of the cardiac valves
36. Mitral and tricuspid valvulitis with small, sterile vegetations, called Libman-Sacks endocarditis, is occasionally encountered in SLE. The lesions are small (1–4 mm in diameter) single or multiple, sterile, pink vegetations that often have a warty (verrucous) appearance
37. The cardiovascular lesions associated with the carcinoid syndrome are distinctive, consisting of firm plaquelike endocardial fibrous thickenings on the inside surfaces of the cardiac chambers and the tricuspid and pulmonary valves
38. The minimal criteria for the diagnosis of systemic HHD are the following: (1) left ventricular hypertrophy (usually concentric) in the absence of other cardiovascular pathology and (2) a history or pathologic evidence of hypertension
39. Microscopically, the earliest change of systemic HHD is an increase in the transverse diameter of myocytes, which may be difficult to appreciate on routine microscopy. At a more advanced stage variable degrees of cellular and nuclear enlargement become apparent, often accompanied by interstitial fibrosis.
40. In more than 90% of cases, the cause of myocardial ischemia is reduced blood flow due to obstructive atherosclerotic lesions in the coronary arteries. Thus, IHD is often termed coronary artery disease (CAD) or coronary heart disease.
41. Most myocardial infarcts are transmural, in which the ischemic necrosis involves the full or nearly full thickness of the ventricular wall in the distribution of a single coronary artery.
42. subendocardial (nontransmural) infarct constitutes an area of ischemic necrosis limited to the inner one third to one half of the ventricular wall
43. The gross and microscopic appearance of an infarct depends on the duration of survival of the patient following the MI.
44. The typical changes of coagulative necrosis become detectable in the first 6 to 12 hours in MI.
45. 12 to 24 hrs changes following MI in heart : Ongoing coagulation necrosis; pyknosis of nuclei; myocyte hypereosinophilia; marginal contraction band necrosis; early neutrophilic infiltrate. Dark mottling on gross.
46. 3 to 7 days MI change : Beginning disintegration of dead myofibers, with dying neutrophils; early phagocytosis of dead cells by macrophages at infarct border.on gross < Hyperemic border; central yellow-tan softening.
47. Scarring following MI occurs in 2 to 8 weeks.
48. The laboratory evaluation of MI is based on measuring the blood levels of proteins that leak out of fatally injured myocytes; these molecules include myoglobin, cardiac troponins T and I, the MB fraction of creatine kinase (CK-MB), lactate dehydrogenase, and many others.Troponin leads the importance .
49. The most sensitive and specific biomarkers of myocardial damage are cardiac-specific proteins, particularly Troponins I and T (proteins that regulate calcium-mediated contraction of cardiac and skeletal muscle).
50. Unchanged levels of CK-MB and troponin over a period of 2 days essentially excludes the diagnosis of MI.
51. The risk of specific postinfarct complications and the prognosis depend primarily on the infarct size, location, and thickness (subendocardial or transmural).
52. The designation chronic IHD is used here to describe progressive heart failure as a consequence of ischemic myocardial damage.
53. Acute myocardial ischemia is the most common trigger for fatal arrhythmias

54. VSD alone does not cause cyanosis.
55. Cyanosis or blue babies are typical of TOF (Tetralogy of fallot)
56. In response to increases in pressure (e.g., hypertension or aortic stenosis), ventricles develop pressure-overload hypertrophy, which usually causes a concentric increase in wall thickness.
57. volume-overload hypertrophy is characterized by ventricular dilation. This is because the new sarcomeres assembled in response to volume overload are largely positioned in series with existing sacromeres.
58. hemosiderin-laden macrophages are telltale signs of previous episodes of pulmonary edema and are often referred to as heart failure cells
59. Most commonly, right-sided heart failure is caused by left-sided heart failure, as any increase in pressure in the pulmonary circulation incidental to left-sided failure inevitably burdens the right side of the heart.
60. Left-sided heart failure is most often caused by (1) ischemic heart disease, (2) hypertension, (3) aortic and mitral valvular diseases, and (4) myocardial diseases.

Special Stains used in Heart lesions :

Masson trichrome stain – it stains collagen , the fibrotic component as blue and rest all tissue including muscles and vessels as red so that we can identify that healed fibrotic part is blue in colour.

MSB is the most commonly used special stain for Fibrin – MSB stands for three dyes : Martius yellow , Scarlet red and mehtylene blue. It stains mature fibrin and muscles as red.Rbc's as yellow and collagen as blue.

Immunostains : No special immunostains required in diagnosis of routine heart lesions.

FEMALE GENITAL SYSTEM PATHOLOGY

1. High oncogenic risk HPVs are currently considered to be the single most important factor in cervical oncogenesis
2. HPV 16 and HPV 18 are the most important. HPV 16 alone accounts for almost 60% of cervical cancer cases, and HPV 18 accounts for another 10% of cases; other HPV types contribute to less than 5% of cases, individually.
3. Viral E6 and E7 proteins are critical for the oncogenic effects of HPV. They can promote cell cycle by binding to RB and up-regulation of cyclin E (E7); interrupt cell death pathways by binding to p53 (E6); induce centrosome duplication and genomic instability (E6, E7); and prevent replicative senescence by up-regulation of telomerase (E6)
4. cervical intraepithelial neoplasia (CIN) classification :mild dysplasia termed CIN I, moderate dysplasia CIN II, and severe dysplasia termed CIN III.
5. the three-tier classification system has been recently simplified to a two-tiered system, with CIN I renamed low-grade squamous intraepithelial lesion (LSIL) and CIN II and CIN III combined into one category referred to as high-grade squamous intraepithelial lesion (HSIL)
6. The diagnosis of SIL is based on identification of nuclear atypia characterized by nuclear enlargement, hyperchromasia (dark staining), presence of coarse chromatin granules, and variation

of nuclear sizes and shapes. The nuclear changes may be accompanied by cytoplasmic halos indicating disruption of the cytoskeleton before release of the virus into the environment

7. Nuclear alterations and perinuclear halo are termed koilocytic atypia.
8. Squamous cell carcinoma is the most common histologic subtype of cervical cancer, accounting for approximately 80% of cases
9. Pap tests are cytologic preparations of exfoliated cells from the cervical transformation zone that are stained with the Papanicolaou method.
10. Chronic endometritis needs the presence of plasma cells to confirm the lesion
11. Endometriosis is the presence of endometrial tissue outside of the uterus. It most commonly consists of both endometrial glands and stroma, but rarely consists only of endometrial stroma.
12. Endometriosis occurs in the following sites, in descending order of frequency: (1) ovaries; (2) uterine ligaments; (3) rectovaginal septum; (4) cul de sac; (5) pelvic peritoneum; (6) large and small bowel and appendix; (7) mucosa of the cervix, vagina, and fallopian tubes; and (8) laparotomy scars.
13. theories of endometriosis include:metastatic and metaplastic theory.
14. There is a profound activation of the inflammatory cascade in endometriosis, characterized by high levels of prostaglandin E2, IL-1β, TNF and IL-6.
15. A histologic diagnosis of endometriosis is readily made when both endometrial glands and stroma are present , with or without the presence of hemosiderin
16. adenomyosis, is defined as the presence of endometrial tissue within the uterine wall
17. Endometrial hyperplasia deserves special attention because of its relationship with endometrial carcinoma
18. A common genetic alteration found in a significant number of hyperplasias and related endometrial carcinomas is inactivation of the PTEN tumor suppressor gene
19. Endometrial hyperplasia is classified as Simple hyperplasia without atypia , simple hyperplasia with atypia , complex hyperplasia without atypia and complex hyperplasia with atypia.
20. Endometrial carcinoma is the most common invasive cancer of the female genital tract and accounts for 7% of all invasive cancer in women, excluding skin cancer
21. type 1 endometrial carcinomas are the most common type, accounting for greater than 80% of all cases. The majority are well differentiated and mimic proliferative endometrial glands and, as such, are referred to as endometrioid carcinomaand show the background of hormone exposure
22. Grading of endometrial carcinoma :

G1. Well-differentiated adenocarcinoma, less than 5% solid growth

G2. Moderately differentiated adenocarcinoma with partly (less than 50%) solid growth

G3. Poorly differentiated adenocarcinoma with predominantly solid growth (greater than 50%)

23. These generally occur in women a decade later than type I carcinoma, and in contrast to type I carcinoma they usually arise in the setting of endometrial atrophy.clear and papillary are variants.P53 gene loss is involved.

24.MMMTs (previously referred to as carcinosarcomas) consist of endometrial adenocarcinomas with malignant changes in the stroma

25. Uterine leiomyomas (commonly called fibroids) are perhaps the most common tumor in women. They are benign smooth muscle neoplasms that may occur singly, but most often are multiple

26. leiomyomas hin the myometrium (intramural), just beneath the endometrium (submucosal) or beneath the serosa (subserosal).

27. On histologic examination, the leiomyoma is composed of whorled bundles of smooth muscle cells that resemble the uninvolved myometrium .sually, the individual muscle cells are uniform in size and shape and have the characteristic oval nucleus and long, slender bipolar cytoplasmic processes.

28.On histologic examination, the leiomyosarcoma range f atypia, from those that are extremely well differentiated to highly anaplastic, pleomorphic lesions The distinction from leiomyomas is based on nuclear atypia, mitotic index, and zonal necrosis.

29. Polycystic ovarian disease (PCOD; formerly termed SteinLeventhal syndrome) affects 3% to 6% of reproductive-age women. The central pathologic abnormality is numerous cystic follicles or follicle cysts, often associated with oligomenorrhea

30. There are three major histologic types of surface epithelial tumors of ovary based on differentiation of the neoplastic epithelium: serous, mucinous, and endometrioid tumors

31. Serous carcinomas account for approximately 40% of all cancers of the ovary and are the most common malignant ovarian tumors.

32. Molecular studies of low- and high-grade serous carcinoma have revealed distinct molecular genetic changes in the two types of carcinoma.Kras and braf in low grade and p53 in high grade.

33. Mucinous tumors of ovary are less common than serous tumors, accounting for about 30% of all ovarian neoplasms. They occur principally in middle adult life and are rare before puberty and after menopause. Eighty percent are benign or borderline, and about 15% are malignant.

34.Serous tumors are unilocular mostly and mucinous multilocular.

35. A clinical condition referred to as pseudomyxoma peritonei is defined by extensive mucinous ascites, cystic epithelial implants on the peritoneal surfaces, adhesions, and frequently mucinous tumor involving the ovaries

36. Endometrioid tumors are distinguished from serous and mucinous tumors by the presence of tubular glands bearing a close resemblance to benign or malignant endometrium

37. Brenner tumors are classified as adenofibromas in which the epithelial component consists of nests of transitional-type epithelial cells resembling those lining the urinary bladder

38. Teratomas are divided into three categories: (1) mature (benign), (2) immature (malignant), and (3) monodermal or highly specialized.

39.Benign dermoid cysts are mature teratomas.

40/.About 1% of the dermoids undergo malignant transformation (e.g., thyroid carcinoma, melanoma, but most commonly, squamous cell carcinoma).

41. The specialized teratomas are a remarkable, rare group of tumors, the most common of which are struma ovarii(thyroid) and carcinoid.

42. The dysgerminoma is best considered as the ovarian counterpart of the seminoma of the testis.

43. On histologic examination the dysgerminoma cells are dispersed in sheets or cords separated by scant fibrous stroma . As in the seminoma, the fibrous stroma is infiltrated with mature lymphocytes and occasional granulomas

44. Similar to the normal yolk sac, the yolk sac tumor is rich in α-fetoprotein and α_1-antitrypsin. Its characteristic histologic feature is a glomerulus-like structure composed of a central blood vessel enveloped by germ cells within a space lined by germ cells (Schiller-Duval body)

45. granulose cell tumor takes one of many histologic patterns. The small, cuboidal to polygonal cells may grow in anastomosing cords, sheets, or strands .In occasional cases small, distinctive, gland-like structures filled with an acidophilic material recall immature follicles (Call-Exner bodies).

46. Tumors arising in the ovarian stroma that are composed of either fibroblasts (fibromas) or plump spindle cells with lipid droplets (thecomas) are relatively common and account for about 4% of all ovarian tumors . Many tumors contain a mixture of these cells and are termed fibromathecomas.

47. The ovarian hilum normally contains clusters of polygonal cells arranged around vessels (hilar cells). Hilus cell tumors (pure Leydig cell tumors) are derived from these cells and are rare, unilateral, and characterized histologically by large lipid-laden cells with distinct borders.

48. A classic example of metastatic gastrointestinal neoplasia to the ovaries is termed Krukenberg tumor, characterized by bilateral metastases composed of mucin-producing, signet-ring cancer cells, most often of gastric origin.

49, Hydatidiform mole is characterized histologically by cystic swelling of the chorionic villi, accompanied by variable trophoblastic proliferation.

50.The most important reason for the correct recognition of moles is that they are associated with an increased risk of persistent trophoblastic disease (invasive mole) or choriocarcinoma

51. Histologically, choriocarcinoma does not produce chorionic villi and consists entirely of a mixed proliferation of syncytiotrophoblasts and cytotrophoblasts

52.Moles are complete and partial.

53.psamomma bodies can be found in papillary serous carcinomas of ovary.

CNS FLASH POINTS:

1. Meningitis refers to an inflammatory process of the leptomeninges and CSF within the subarachnoid space, while meningoencephalitis combines this with inflammation of the brain parenchyma.
2. Infectious meningitis is broadly classified into acute pyogenic (usually bacterial meningitis), aseptic (usually acute viral meningitis), and chronic (usually tuberculous, spirochetal, or cryptococcal) on the basis of the characteristics of inflammatory exudate on CSF examination and the clinical evolution of the illness.Acute cases show neutrophils and chronic cases lymphocytes.
3. In Rabies we find - Negri bodies, the pathognomonic microscopic finding, are cytoplasmic, round to oval, eosinophilic inclusions that can be found in pyramidal neurons of the hippocampus and Purkinje cells of the cerebellum, sites usually devoid of inflammation
4. HIV encephalitis is best characterized microscopically as a chronic inflammatory reaction with widely distributed infiltrates of microglial nodules, sometimes with associated foci of tissue necrosis and reactive gliosis
5. Multiple sclerosis (MS) is an autoimmune demyelinating disorder characterized by distinct episodes of neurologic deficits, separated in time, attributable to white matter lesions that are separated in space.
6. Lesions in multiple sclerosis appear as multiple, well-circumscribed, somewhat depressed, glassy, graytan, irregularly shaped plaques – active , inactive and shadow plaques.
7. The major microscopic abnormalities of Alzeihmer's disease which form the basis of the histologic diagnosis, are neuritic (senile) plaques – amyloid core and neurofibrillary tangles
8. The major classes of primary brain tumors to be considered here include gliomas, neuronal tumors, poorly differentiated tumors, as well as a small collection of other tumors
9. Gliomas, the most common group of primary brain tumors, include astrocytomas, oligodendrogliomas, and ependymomas.
10. The two major categories of astrocytic tumors are infiltrating astrocytomas and non-infiltrating neoplasms, of which the most common are the pilocytic astrocytomas seen in children.
11. Infiltrating astrocytomas account for about 80% of adult primary brain tumors in adults
12. Grading of astrocytomas : Infiltrating tumors range from diffuse astrocytoma (grade II/IV) through anaplastic astrocytoma (grade III/IV) to glioblastoma (grade IV/IV). (The grade I/IV category is limited to pilocytic astrocytoma – non infiltrating.)
13. On microscopic examination, diffuse astrocytomas are characterized by a mild to moderate increase in glial cellularity, variable nuclear pleomorphism, and an intervening feltwork of fine, GFAP-positive astrocytic processes that give the background a fibrillary appearance

14. Anaplastic astrocytomas show regions that are more densely cellular and have greater nuclear pleomorphism; mitotic figures are often observed.
15. The histologic appearance of glioblastoma is similar to anaplastic astrocytoma with the additional features of necrosis (pseudopalisading arrangement also seen) and vascular or endothelial cell proliferation
16. The term gemistocytic astrocytoma is used for tumors in which the predominant neoplastic astrocyte shows a brightly eosinophilic cell body from which emanate abundant, stout processes.
17. Among the alterations that are most common in the low-grade astrocytomas are mutations affecting p53 and overexpression of platelet-derived growth factor α (PDGF-A) and its receptor
18. The transition to higher grade astrocytoma is associated with disruption of two well-known tumor suppressor genes, RB and p16/CDKNaA, and an unknown putative tumor suppressor on chromosome 19q.
19. combinations of mutations that activate RAS and PI-3 kinase and inactivate p53 and RB are present in 80% to 90% of primary glioblastomas
20. On microscopic examination of a pilocytic astrocytoms (presents typically as cystic nodule on gross in kids) the tumor is composed of bipolar cells with long, thin “hairlike” processes that are GFAP-positive and form dense fibrillary meshworks; Rosenthal fibers and eosinophilic granular bodies, are often present. Tumors are often biphasic with a loose microcystic pattern in addition to the fibrillary areas
21. On microscopic examination, the oligodendrogliomas are composed of sheets of regular cells with spherical nuclei containing finely granular chromatin (similar to normal oligodendrocytes) surrounded by a clear halo of cytoplasm.Often show calcification.
22. Tumor cells in ependymomas may form glandlike round or elongated structures (rosettes, canals) that resemble the embryologic ependymal canal, with long, delicate processes extending into a lumen ; more frequently present are perivascular pseudorosettes
23. Medulloblastoma occurs predominantly in children and exclusively in the cerebellum show round blue cells with hyperchromatic nuclei.Poorly differentiated tumors. The tumor may express neuronal (neurosecretory granules or Homer Wright rosettes, as occur in neuroblastoma
24. Medulloblastoma cells have a propensity to form linear chains of cells infiltrating through cerebellar cortex to aggregate beneath the pia, penetrate the pia, and seed into the subarachnoid space. Dissemination through the CSF is a common complication, presenting as nodular masses elsewhere in the CNS, including metastases to the cauda equina that are sometimes termed drop metastases.
25. Meningiomas are predominantly benign tumors of adults, usually attached to the dura, that arise from the meningothelial cell of the arachnoid
26. Most meningiomas have a relatively low risk of recurrence or aggressive growth, and so are considered WHO grade I/IV
27. Variants of grade I meningiomas include syncitial , transitional , fibroblastic , psamommatous , secretory and microcystic.
28. Atypical meningiomas are distinguished from lower grade meningiomas by the presence of either a mitotic index of four or more mitoses per 10 high power fields or at least three atypical features (increased cellularity, small cells with a high nuclear-to-cytoplasmic ratio, prominent nucleoli, patternless growth, or necrosis). Certain histologic patterns (clear cell and chordoid) are also considered to be grade II/IV because of their more aggressive behavior.
29. Anaplastic (malignant) meningioma (WHO grade III/IV) is a highly aggressive tumor with the appearance of a high-grade sarcoma, but retaining some histologic evidence of meningothelial origin. Mitotic rates are often extremely high (>20 mitoses per 10 high power fields).Variants include papillary and rhabdoid.

30. Metastasis : The five most common primary sites are lung, breast, skin (melanoma), kidney, and gastrointestinal tract, accounting for about 80% of all metastases

BREAST PATHOLOGY FLASH POINTS

1.Duct ectasia characterized chiefly by dilation of ducts, inspissation of breast secretions, and a marked periductal and interstitial chronic granulomatous inflammatory reaction

2.Fat necrosis can present as a painless palpable mass, skin thickening or retraction, a mammographic density, or mammographic calcifications

3.There are three principal morphologic changes in fibrocystic disease: (1) cystic change, often with apocrine metaplasia; (2) fibrosis; and (3) adenosis

4. Adenosis is defined as an increase in the number of acini per lobule.

5. Epithelial hyperplasia is defined by the presence of more than two cell layers. The additional cells consist of both luminal and myoepithelial cell types that fill and distend ducts and lobules

6. Sclerosing Adenosis. The number of acini per terminal duct is increased to at least double the number found in uninvolved lobules. The normal lobular arrangement is maintained

7. Papillomas are composed of multiple branching fibrovascular cores, each having a connective tissue axis lined by luminal and myoepithelial cells.They present as nipple discharge complains by the patient.

8. Proliferative disease with atypia includes atypical ductal hyperplasia and atypical lobular hyperplasia

9. Carcinoma of the breast is the most common non-skin malignancy in women

10. The major risk factors for the development of breast cancer are hormonal and genetic.

11. Mutations in BRCA1 and BRCA2 account for the majority of cancers attributable to single mutations and about 3% of all breast cancers

12. The known high-risk breast cancer genes account for only about one quarter of familial breast cancers

13. The major risk factors for sporadic breast cancer are related to hormone exposure: gender, age at menarche and menopause, reproductive history, breastfeeding, and exogenous estrogens

14. Greater than 95% of breast malignancies are adenocarcinomas, which are divided into in situ carcinomas and invasive carcinomas

15.DCIS is ductal carcinoma in situ.

16. Historically, DCIS has been divided into five architectural subtypes: comedocarcinoma, solid, cribriform, papillary, and micropapillary

17. Comedocarcinoma is characterized by the presence of solid sheets of pleomorphic cells with "high-grade" hyperchromatic nuclei and areas of central necrosis.It is high grade DCIS.

18.In paget's disease , Malignant cells (Paget cells) extend from DCIS within the ductal system, via the lactiferous sinuses, into nipple skin without crossing the basement membrane

19. LCIS , lobular carcinoma in situ , is always an incidental biopsy finding, since it is not associated with calcifications or stromal reactions that produce mammographic densities.

20. Palpable tumors are associated with axillary lymph node metastases in over 50% of patients.

21. The term inflammatory carcinoma is reserved for tumors that present with a swollen, erythematous breast. This gross appearance is caused by extensive invasion and obstruction of dermal lymphatics by tumor cells.

22. Gene expression profiling, which can measure the relative quantities of mRNA for essentially every gene, has identified five major patterns of gene expression in the NST group: luminal A, luminal B, normal, basal-like, and HER2 positive

- 23. Estrogen and progesterone receptors are found in breast cancer cells that depend on estrogen and related hormones to grow.
    - 24. All patients with invasive breast cancer or a breast cancer recurrence should have their tumors tested for estrogen and progesterone receptors.

25. If breast cancer cells have estrogen receptors, the cancer is called ER-positive breast cancer. If breast cancer cells have progesterone receptors, the cancer is called PR-positive breast cancer. If the cells do not have either of these two receptors, the cancer is called ER/PR-negative. About two-thirds of breast cancers are ER and/or PR positive.

26. If a patient's tumor expresses ER and/or PR, as seen in approximately 70% of invasive breast cancers, we can predict that this patient will likely benefit from endocrine therapy such as tamoxifen.

27. Her2 expression is associated with a diminished prognosis (e.g., higher risk of recurrence), however, it also predicts that a patient will more likely benefit from anthracycline and taxane-based chemotherapies and directed therapies that target Her2 (trastuzamab), but not to endocrine-based therapies

28.Ductal carcinoma of the breast is the most common type.

29.Lobular carcinomas have been reported to have a greater incidence of bilaterality

30. The histologic hallmark of lobular carcinomais the presence of dyscohesive infiltrating tumor cells, often arranged in single file or in loose clusters or sheets with Indian file pattern

31. Lobular carcinomas have a different pattern of metastasis than other breast cancers. Metastasis tends to occur to the peritoneum and retroperitoneum, the leptomeninges (carcinoma meningitis), the gastrointestinal tract, and the ovaries and uterus.

32. Histologically, the medullary carcinoma is characterized by (1) solid, syncytium-like sheets of large cells with vesicular, pleomorphic nuclei, and prominent nucleoli, which compose more than 75% of the tumor mass; (2) frequent mitotic figures; (3) a moderate to marked lymphoplasmacytic infiltrate surrounding and within the tumor; and (4) a pushing (noninfiltrative) border

33.Tubular,mucinous and papillary are other variants of carcinoma breast

34. “Metaplastic carcinoma” includes a variety of rare types of breast cancer (<1% of all cases), such as matrix-producing carcinomas, squamous cell carcinomas, and carcinomas with a prominent spindle cell component

35. prognosis is determined by the pathologic examination of the primary carcinoma and the axillary lymph nodes.

36. Axillary lymph node status is the most important prognostic factor for invasive carcinoma in the absence of distant metastases.

37. The most commonly used grading system, the Nottingham Histologic Score (also referred to as Scarff-Bloom-Richardson), combines nuclear grade, tubule formation, and mitotic rate to classify invasive carcinomas into three groups that are highly correlated with survival.

38.Fibroadenoma is the most common benign tumor of the female breast

39. Phyllodes tumors, like fibroadenomas, arise from intralobular stroma

40. Phyllodes tumors are distinguished from the more common fibroadenomas on the basis of cellularity, mitotic rate, nuclear pleomorphism, stromal overgrowth, and infiltrative borders.

41.FNAC and biopsy are used to diagnose breast cancer.

BONES , JOINTS , SOFT TISSUES AND MUSCLES FLASH POINTS

1- Osteomyelitis denotes inflammation of bone and marrow, and the common use of the term virtually always implies infection.
2- Staphylococcus aureus is responsible for 80% to 90% of the cases of pyogenic osteomyelitis in which an organism is recovered
3. Brodie abscess is a small intraosseous abscess that frequently involves the cortex and is walled off by reactive bone;
4. Tuberculous osteomyelitis tends to be more destructive and resistant to control than pyogenic osteomyelitis
5. Most common bone tumors are haematopoetic in origin including multiple myeloma and malignant lymphoma involving bones.
6. Multiple osteomas are seen in the setting of Gardner syndrome
7. Osteoid osteoma and osteoblastoma are terms used to describe benign bone tumors that have identical histologic features but differ in size, sites of origin, and symptoms
8. Osteoid osteomas are by definition less than 2 cm in greatest dimension and usually occur in the teens and 20s
9. Osteoblastoma is larger than 2 cm and involves the spine more frequently; the pain is dull, achy, and unresponsive to salicylates, and the tumor usually does not induce a marked bony reaction.
10. The actual tumor in osteoid osteoma and osteoblastoma, known as the nidus, manifests radiographically as a small round lucency that may be centrally mineralized
11. Osteosarcoma is a malignant mesenchymal tumor in which the cancerous cells produce bone matrix. It is the most common primary malignant tumor of bone, exclusive of myeloma and lymphoma, and accounts for approximately 20% of primary bone cancers.
12. RB and p53 Are genes associated with osteosarcoma.
13. The tumor cells in osteosarcoma are oval to spindly in shape , vary in size and shape and frequently have large hyperchromatic nuclei. Bizarre tumor giant cells are common, as are mitoses.
14. The formation of bone or osteoid by the tumor cells is characteristic of osteosarcoma.
15. Variants of osteosarcoma include small cell , chondroblastic , telangiectatic ,parosteal and periosteal.
16. Codman triangle is seen with osteosarcoma on radiological findings.
17. Osteochondroma, also known as an exostosis, is a benign cartilage-capped tumor that is attached to the underlying skeleton by a bony stalk. It is the most common benign bone tumor; about 85% are solitary.
18. Chondromas can arise within the medullary cavity, where they are known as enchondromas, or on the surface of bone, where they are called subperiosteal or juxtacortical chondromas
19. Chondroblastoma is a rare benign tumor that accounts for less than 1% of primary bone tumors.
20. Chondroblastoma is composed of sheets of compact polyhedral chondroblasts that have well-defined cytoplasmic borders, moderate amounts of pink cytoplasm, and nuclei that are hyperlobulatedwith longitudinal grooves
21. Chicken wire calcifications are seen in chondroblastoma.
22. Chondrosarcoma is subclassified according to site as central (intramedullary) and peripheral (juxtacortical and surface). Histologically, they include conventional (hyaline and/or myxoid), clear cell, dedifferentiated, and mesenchymal variants.
23. In contrast to enchondroma, chondrosarcoma rarely involves the distal extremities.
24. Chinese letter bony trabeculae are seen in fibrous dysplasia.
25. Herring bone pattern is seen in a fibrosarcoma

26. The Ewing sarcoma family of tumors encompasses Ewing sarcoma and primitive neuroectodermal tumor (PNET), which are primary malignant small round-cell tumors of bone and soft tissue
27. Ewing sarcoma and PNET together account for approximately 6% to 10% of primary malignant bone tumors and follow osteosarcoma as the second most common group of bone sarcomas in children
28. Ewing's sarcoma is composed of sheets of uniform small, round cells that are slightly larger than lymphocytes . They have scant cytoplasm, which may appear clear because it is rich in glycogen. The presence of Homer-Wright rosettes (tumor cells arranged in a circle about a central fibrillary space) is indicative of neural differentiation.
29. Giant-cell tumor is so named because it contains a mixture of mononuclear cells and a profusion of multinucleated osteoclast-type giant cells, giving rise to the synonym osteoclastoma. This tumor is a relatively uncommon benign but locally aggressive neoplasm
30. Giant-cell tumors in adults involve both the epiphyses and the metaphyses, but in adolescents they are confined proximally by the growth plate and are limited to the metaphysis.
31. Aneurysmal bone cyst is a benign tumor of bone characterized by multiloculated blood-filled cystic spaces that may present as a rapidly growing expansile tumor.
32. Any cancer can spread to bone, but in adults more than 75% of skeletal metastases originate from cancers of the prostate, breast, kidney, and lung. In children, metastases to bone originate from neuroblastoma, Wilms tumor, osteosarcoma, Ewing sarcoma, and rhabdomyosarcoma.
33. Skeletal metastases are typically multifocal; however, carcinomas of the kidney and thyroid are notorious for producing solitary lesions.
34. Rheumatoid arthritis is a chronic systemic inflammatory disorder that may affect many tissues and organs—skin, blood vessels, heart, lungs, and muscles—but principally attacks the joints, producing a nonsuppurative proliferative and inflammatory synovitis that often progresses to destruction of the articular cartilage and ankylosis of the joints.
35. pannus formation is seen in rheumatoid arthritis. The pannus is a mass of synovium and synovial stroma consisting of inflammatory cells, granulation tissue, and synovial fibroblasts, which grows over the articular cartilage and causes its erosion
36. Microscopically Rheumatoid nodules have a central zone of fibrinoid necrosis surrounded by a prominent rim of epithelioid histiocytes (activated macrophages) and numerous lymphocytes and plasma cells
37. RA factor is the main test done to diagnose Rheumatoid arthritis.
38. . In gout , A deficiency of the enzyme hypoxanthine guanine phosphoribosyl transferase (HGPRT) leads to increased synthesis of purine nucleotides through the de novo pathway and hence increased production of uric acid.
39. A complete lack of HGPRT occurs in the uncommon X-linked Lesch-Nyhan syndrome, seen only in males and characterized by hyperuricemia, severe neurologic deficits with mental retardation, self-mutilation, and in some cases gouty arthritis.
40. Tophi are the pathognomonic hallmark of gout. They are formed by large aggregations of urate crystals surrounded by an intense inflammatory reaction of macrophages, lymphocytes, and large foreign body giant cells, which may have completely or partially engulfed masses of crystals
41. For many types of soft-tissue sarcomas the histologic grade is important. Grading, usually I to III, is based on the degree of differentiation, the average number of mitoses per high-power field, cellularity, pleomorphism, and an estimate of the extent of necrosis (presumably a reflection of rate of growth).[
42. In lipsarcoma , the characteristic cells are known as lipoblasts; they mimic fetal fat cells and contain round clear cytoplasmic vacuoles of lipid that scallop the nucleus
43. Myositis ossificans is distinguished from the other reactive fibroblastic proliferations by the presence of metaplastic bone.

44. Histologic examination of fibrosarcoma discloses all degrees of differentiation, from slowly growing tumors that closely resemble cellular fibromatosis and sometimes having spindled cells growing in a herringbone fashion.Hemorrhage and necrosis can be seen
45. Rhabdomyosarcoma is histologically subclassified into embryonal, alveolar, and pleomorphic variants. The rhabdomyoblast—the diagnostic cell in all types—contains eccentric eosinophilic granular cytoplasm rich in thick and thin filaments.
46. Synovial sarcoma : The histologic hallmark of biphasic synovial sarcoma is dual lines of differentiation (i.e., epithelial-like and mesenchymal-like). The epithelial cells are cuboidal to columnar and form glands or grow in solid cords or aggregates. The spindle cells are arranged in densely cellular fascicles that surround the epithelial cells
47. Immunostains for Sarcomas is Vimentin.
48. Immunostain for muscle is Desmin
49. Neural immunomarker is S-100.
50. The muscular dystrophies are a heterogeneous group of inherited disorders of muscle, often beginning in childhood, that lead to progressive weakness and muscle wasting. Histologically, in advanced cases muscle fibers undergo degeneration and are replaced by fibrofatty tissue and collagen
51. The two most common forms of muscular dystrophy are X linked: Duchenne muscular dystrophy (DMD) and Becker muscular dystrophy (BMD).
52. Histopathologic abnormalities common to DMD and BMD include (1) variation in fiber size (diameter) due to the presence of both small and enlarged fibers, sometimes with fiber splitting; (2) increased numbers of internalized nuclei (beyond the normal range of 3% to 5%); (3) degeneration, necrosis, and phagocytosis of muscle fibers; (4) regeneration of muscle fibers; and (5) proliferation of endomysial connective tissue

LUNG PATHOLOGY FLASH POINTS

1.Common obstructive lung diseases include emphysema, chronic bronchitis, asthma, and bronchiectasis, each of which has distinct pathologic features and clinical characteristics.\

2.Emphysema is characterized by irreversible enlargementof the airspaces distal to the terminal bronchiole, accompanied by destruction of their walls without obviousfibrosis.

3.emphysema is classified into four major types: (1) *centriacinar*, (2) *panacinar*, (3) *paraseptal*, and (4) *irregular*. Of these, only the first two cause clinically significant airflow obstruction

4.*Centriacinar (centrilobular) emphysema.* In this type of emphysema the central or proximal parts of the acini, formed by respiratory bronchioles, are affected, whereas distal alveoli are spared

5.*Panacinar (panlobular) emphysema*. In this type, the acini are uniformly enlarged from the level of the respiratory bronchiole to the terminal blind alveoli

6.The idea that proteases are important is based in part on the observation that patients with a genetic deficiency of the anti proteasea1-antitrypsin have a markedly enhanced tendency to develop pulmonary emphysema, which is compounded by smoking.This is called protease antiprotease theory.

7.Smoking and inhaled pollutants cause ongoing accumulations of inflammatory cells, releasing elastases and oxidants, which destroy the alveolar walls.

8.In chronic bronchitis , mild chronic inflammation of the airways (predominantly lymphocytes) and enlargement of the mucus-secreting glands of the trachea and bronchi.themajor change is in the size of mucous glands (hyperplasia). This increase can be assessed by the ratio of the thickness of the mucous gland layer to the thickness of the wall between the epithelium and the cartilage(Reid index). The Reid index (normally 0.4) is increased in chronic bronchitis.

9.*Atopic Asthma.* This is the most common type of asthma which is a classic example of IgE-mediated (type I) hypersensitivity reaction,

10.In bronchianasthma , classically u see Curschmann spirals, which may result from extrusion of mucus plugs from subepithelial mucous gland ducts or bronchioles. Also present are numerous eosinophils and Charcot-Leyden crystals; the latter are composed of an eosinophil protein called galectin.
The other characteristic histologic findings of asthma, collectively called "airway remodeling" which shows fibrosis,hypertrophic glands and smooth muscle hyperplasia.

11.Eosinophils are key inflammatory cells found in almost all subtypes of asthma; other inflammatory cells include mast cells, neutrophils and T lymphocytes.

12.Bronchiectasis is a disorder in which destruction of smooth muscle and elastic tissue by chronic necrotizing infections leads to permanent dilation of bronchi and bronchioles.

13.Obstruction and infection are the major conditions associated with bronchiectasis, and it is likely that both are necessary for the development of full-fledged lesions.

14.Restrictive lung disorders occur in two general conditions:
(1) *chronic interstitial and infiltrative diseases,* such as pneumoconioses and interstitial fibrosis of unknown etiology; and(2) *chest wall disorders* (e.g., neuromuscular diseases such as poliomyelitis, severe obesity, pleural diseases, and kyphoscoliosis),

15.Idiopathic pulmonary fibrosis (IPF) refers to a clinicopathologic syndrome marked by progressive interstitial pulmonary fibrosis and respiratory failure. It is characterized by patchy interstitial fibrosis fibroblastic foci and formation of cystic spaces (honeycomb lung). This histologic pattern is also called usual interstitial pneumonia.

16.The term *pneumoconiosis,* originally coined to describe the nonneoplastic lung reaction to inhalation of mineral dusts encountered in the workplace, now also includes diseases induced by organic as well as inorganic particulates and chemical fumes and vapors.

17.Asbestos is classically linked with mesothelioma.

18.Asbestos causes following lesions :

Localized fibrous plaques or, rarely, diffuse pleural fibrosis
• Pleural effusions, recurrent
• Parenchymal interstitial fibrosis *(asbestosis)*
• Lung carcinoma
• Mesotheliomas
• Laryngeal, ovarian and perhaps other extrapulmonary
neoplasms, including colon carcinomas; increased risk for systemic autoimmune diseases

19.Asbestos bodies are golden brown, fusiform or beaded rods with a translucent center and consist of asbestos fibers coated with an iron-containing proteinaceous material

20.Silicosis is linked with Tuberculosis.

21.Sarcoidosis is a systemic granulomatous disease of unknown cause that may involve many different tissues and organs. Sarcoidosis presents in many clinical patterns, but bilateral hilar lymphadenopathy or lung involvement is most common, occuring 90% of cases.

22.Insarcoidosis , Intra-alveolar and interstitial accumulation of CD4+ T cells, resulting in CD4/CD8 T-cell ratios ranging from 5 : 1 to 15: 1, suggesting pathogenic involvement of CD4+ helper T cells.

23.Noncaseating granulomas , schaumann bodies and asterioid bodies are seen in Sarcoidosis.

24.The vast majority (60% to 80%) of pulmonary emboli are clinically silent, a minority (5%) cause acute corpulmonale, shock, or death (typically from large "saddle emboli"), and the remaining cause pulmonary infarction.

25.*Streptococcus pneumoniae,* or *pneumococcus,* is the most common cause of community-acquired acute pneumonia.

26.*Staphylococcus aureus*is an important cause of secondary bacterial pneumonia in children and healthy adults following viral respiratory illnesses (e.g., measles in children and influenza in both children and adults).

27.In **lobar pneumonia,** four stages of the inflammatory response have classically been described: congestion, red hepatization, gray hepatization, and resolution.

28.The term *atypical organisms* is used for *Mycoplasma pneumoniae, Chlamydophilapneumoniae, Coxiellaburnetii,* and viruses (influenza viruses types A and B, human metapneumovirus) sincethey are not detectable on Gram stain nor do they grow on the standard bacteriologic culture media.

29.Lung abscess is often caused by anaerobic organisms or by mixed infections and frequently occur in debilitated individuals following aspiration of oral flora.

30.Other common causes of acute pneumonias in the community include*H. influenzae*and *M. catarrhalis*(both associated with acute exacerbations of COPD), S. *aureus* (usuallysecondary to viral respiratory infections), *K. pneumoniae* (observed in patients who are chronic alcoholics), *P. aeruginosa* (seen in persons with cystic fibrosis, in burn victims, and in patients with neutropenia), and *L. pneumophila,* seen particularly in organ transplant recipients.

31.The term *pulmonary abscess* describes a local suppurative process that produces necrosis of lung tissue.

32.The cardinal histologic change in all abscesses is suppurative destruction of the lung parenchyma within the central area of cavitation.

33.Pneumonia in immunocompromised patients include causes :
1) bacteria (P. *aeruginosa, Mycobacterium* species, *L. pneumophila,* and *Listeria monocytogenes),* (2) viruses (cytomegalovirus [CMV] and herpesvirus), and (3) fungi (P. *jiroveci, Candida* species, *Aspergillus* species, the

34.About 80% of lung cancers occur in active smokers or those who stopped recently.

35.*Squamous cell carcinoma* is highly associated with exposure to tobacco smoke and harbors diverse genetic aberrations, many of which are chromosome deletions involving tumor suppressor loci.

36.Squamous cell carcinoma shows the highest number of *TP53* mutations of all histologic types of lung carcinoma.

37.*Small cell carcinoma* shows the strongest association with smoking and despite its divergent histologic features shares many molecular features with squamous cell carcinoma. This includes frequent loss-of-function aberrations involving *TP53* (75% to 90% of tumors), *RB* (close to 100% of tumors), and chromosome 3p deletions.

38.*Precursor (Preinvasive) Lesions.* Four types of morphologic precursor epithelial lesions are recognized: (1) squamous dysplasia and carcinoma in situ, (2) atypical adenomatous hyperplasia, (3) adenocarcinoma in situ, and (4) diffuse idiopathic pulmonary neuroendocrine cell hyperplasia.

39.Adenocarcinoma (38%)
• Squamous cell carcinoma (20%)
• Small cell carcinoma (14%)
• Large cell carcinoma (3%)
• Other (25%)

40.**Adenocarcinoma in situ** (formerly called bronchioloalveolar carcinoma) is a lesion that is less than 3 em and is composed entirely of dysplastic cells growing along preexisting alveolar
septae. The cells have more dysplasia than atypical adenomataus hyperplasia and may or may not have intracellular mucin (mucinous and non mucinous, respectively)

41.Small cell carcinoma (oat cell) is comprised of relatively small cells with scant cytoplasm, ill-defined cell borders, finely granular nuclear chromatin (salt and pepper pattern), and absent or inconspicuous nucleoli . The cells are round, oval, or spindle shaped, and nuclear molding is prominent.

42.Paraneoplastic syndromes are linked with small cell carcinoma of lung including cushings , adh like secretions .Hypercalcemia is linked with squamous cell carcinoma of lung.

43.Smallcell lung carcinomas are best treated by chemotherapy, because almost all are metastatic at presentation.
44.Histologically, the Lung carcinoid tumor is composed of organoid, trabecular, palisading, ribbon, or rosette-like arrangements of cells separated by a delicate fibrovascularstroma. In common with the lesions of the gastrointestinal tract, the individual cells are quite regular and have uniform round nuclei and a moderate\amount of eosinophilic cytoplasm.

45.Chromogranin and synaptophysins are immunomarkers for carcinoid.

46.Neurosecretory granules are seen in small cell carcinoma too showing positive chromogranin and synaptophysins.

47.The lung is the most common site of metastatic neoplasms. Both carcinomas and sarcomas arising anywhere in the body may spread to the lungs via the blood or lymphatics or by direct continuity.

48.Malignant mesotheliomas, although rare, have assumed great importance in the past few decades because of their increased incidence among people with heavy exposure to asbestos

49.Mesotheliomas can be benign or malignant.

50.Ghon focus is a primary tuberculous lesion in lung and Ghon complex is lesion with lymph node showing granulomatous pathology.

ENDOCRINE PATHOLOGY FLASH POINTS

1. Hashimoto thyroiditis is the most common cause of hypothyroidism in areas of the world where iodine levels are sufficient
2. Hashimoto thyroiditis is caused by a breakdown in self-tolerance to thyroid auto-antigens. This is exemplified by the presence of circulating autoantibodies against thyroglobulin and thyroid peroxidase in the vast majority of Hashimoto patients.Cell death occurs due to cytotoxic T cells ,cytokine mediated cell death and antibody dependent cell mediated cytotoxicity.
3. Microscopic examination of Hashimoto thyroiditis reveals extensive infiltration of the parenchyma by a mononuclear inflammatory infiltrate containing small lymphocytes, plasma cells, and well-developed germinal centers.Hurthle cells are also seen which are actually follicular epithelial cells undergone metaplasia.
4. Subacute thyroiditis, which is also referred to as granulomatous thyroiditis or De Quervain thyroiditis, occurs much less frequently than does Hashimoto disease
5. Subacute lymphocytic thyroiditis, which is also referred to as painless thyroiditis, usually comes to clinical attention because of mild hyperthyroidism, goitrous enlargement of the gland, or both.
6. Graves disease is characterized by a breakdown in self-tolerance to thyroid auto-antigens, most importantly the TSH receptor. The thyroid gland is usually symmetrically enlarged because of diffuse hypertrophy and hyperplasia of thyroid follicular epithelial cells
7. Multiple autoantibodies in Graves disease include thyroid stimulating immunoglobulins ,thyroid growth stimulating immunoglobulins and thyroid binding inhibitor immunoglobulins.
8. Two phases can be identified in the evolution of diffuse nontoxic goiter: the hyperplastic phase and the phase of colloid involution
9. Multinodular goiter is characterized by nodules of variable sizes separated by thin fibrous tissue septae.
10. The solitary thyroid nodule is a palpably discrete swelling within an otherwise apparently normal thyroid gland
11. Solitary thyroid nodules, in general, are more likely to be neoplastic than are multiple nodules.
12. Thyroid Nodules in younger patients are more likely to be neoplastic than are those in older patients.
13. Adenomas of the thyroid are typically discrete, solitary masses, derived from follicular epithelium, and hence they are also known as follicular adenomas. Clinically, follicular adenomas can be difficult to distinguish from dominant nodules of follicular hyperplasia or from the less common follicular carcinomas.
14. Somatic mutations of the TSH receptor signaling pathway have been found in toxic adenomas, as well as in toxic multinodular goiter
15. The typical thyroid adenoma is a solitary, spherical, encapsulated lesion that is well demarcated from the surrounding thyroid parenchyma.Compressed normal thyroid tissue is seen at the periphery of an adenoma of thyroid.

16. Careful evaluation of the integrity of the capsule is therefore critical in distinguishing follicular adenomas from follicular carcinomas, which demonstrate capsular and/or vascular invasion.Capsular and vascular invasion are seen in follicular carcinomas.
17. Papillary carcinoma is the most common carcinoma of thyroid .
18. RET PTC gene rearrangements are seen in papillary carcinoma thyroid.
19. Papillary carcinomas can contain branching papillae having a fibrovascular stalk covered by a single to multiple layers of cuboidal epithelial cells. Concentrically calcified structures termed psammoma bodies are often present within the lesion, usually within the cores of papillae
20. The diagnosis of papillary carcinoma is made based on these nuclear features. The nuclei of papillary carcinoma cells contain finely dispersed chromatin, which imparts an optically clear or empty appearance, giving rise to the designation ground-glass or Orphan Annie eye nuclei. (orphan annie name is after a comic character annie who has optically clear eyes)
21. Variants of papillary carcinoma thyroid include Follicular , encapsulated variant , tall cell variant and diffuse sclerosing variant.
22. Anaplastic carcinoma thyroid , microscopically theese neoplasms are composed of highly anaplastic cells, with variable morphology, including: (1) large, pleomorphic giant cells, including occasional osteoclast-like multinucleate giant cells; (2) spindle cells with a sarcomatous appearance; and (3) mixed spindle and giant cells.
23. Medullary carcinomas of the thyroid are neuroendocrine neoplasms derived from the parafollicular cells, or C cells, of the thyroid, and account for approximately 5% of thyroid neoplasms
24. Microscopically, medullary carcinomas are composed of polygonal to spindle-shaped cells, which may form nests, trabeculae, and even follicles. Small, more anaplastic cells are present in some tumors and may be the predominant cell type. Acellular amyloid deposits, derived from altered calcitonin polypeptides, are present in the adjacent stroma in many cases
25. The most common cause of primary hyperparathyroidism is a solitary parathyroid adenoma arising in the sporadic (nonfamilial) setting
26. Aggregates of osteoclasts, reactive giant cells, and hemorrhagic debris occasionally form masses that may be mistaken for neoplasms (brown tumors of hyperparathyroidism).
27. Renal failure is by far the most common cause of secondary hyperparathyroidism
28. The three most common and distinctive clinical syndromes associated with functional pancreatic endocrine neoplasms are (1) hyperinsulinism, (2) hypergastrinemia and the Zollinger-Ellison syndrome, and (3) MEN
29. The most important diseases of the adrenal medulla are neoplasms, which include neoplasms of chromaffin cells (pheochromocytomas) and neuronal neoplasms (neuroblastic tumors).
30. 10% rule fits on pheochromocytoma – 10% are extraadrenal ,biologically malignant , no hypertension in 10%.
31. The histologic pattern in pheochromocytoma is quite variable. The tumors are composed of polygonal to spindle-shaped chromaffin cells or chief cells, clustered with the sustentacular cells into small nests or alveoli (zellballen) by a rich vascular network
32. the definitive diagnosis of malignancy in pheochromocytomas is based exclusively on the presence of metastases
33. The MEN syndromes are a group of genetically inherited diseases resulting in proliferative lesions (hyperplasia, adenomas, and carcinomas) of multiple endocrine organs.
34. MEN-1 is characterized by abnormalities involving the parathyroid, pancreas, and pituitary glands; thus the mnemonic device, the 3Ps
35. MEN-2 is subclassified into three distinct syndromes: MEN-2A, MEN-2B, and familial medullary thyroid cancer.

36. MEN-2A, or Sipple syndrome, is characterized by pheochromocytoma, medullary carcinoma, and parathyroid hyperplasia
37. In MEN 2 B ,Patients develop medullary thyroid carcinomas, which are usually multifocal and more aggressive than in MEN-2A, and pheochromocytomas
38. Thyroid neoplasms with follicle like arrangement include :
    Follicular adenoma

    Follicular carcinoma

    Follicular variant of papillary carcinoma thyroid

    39.Thyroid function tests include : TSH , T3,T4.

    40.Amyloid in medullary carcinoma thyroid is confirmed by a special stain congo red.

GASTROINTESTINAL TRACT PATHOLOGY FLASH POINTS

1. Barrett esophagus is a complication of chronic GERD that is characterized by intestinal metaplasia within the esophageal squamous mucosa.
2. The greatest concern in Barrett esophagus is that it confers an increased risk of esophageal adenocarcinoma.
3. Diagnosis of Barrett esophagus requires both endoscopic evidence of abnormal mucosa above the gastroesophageal junction and histologically documented intestinal metaplasia. Villous formations and Goblet cells, which have distinct mucous vacuoles that stain pale blue by H&E and impart the shape of a wine goblet to the remaining cytoplasm, define intestinal metaplasia and are necessary for diagnosis of Barrett esophagus
4. Two morphologic variants of esophageal carcinoma comprise the majority of esophageal cancers: adenocarcinoma and squamous cell carcinoma
5. The most common cause of chronic gastritis is infection with the bacillus Helicobacter pylori
6. Autoimmune gastritis, the most common cause of atrophic gastritis, represents less than 10% of cases of chronic gastritis and is the most common form of chronic gastritis in patients without H. pylori infection.

7. Four features are linked to H. pylori virulence:

- Flagella, which allow the bacteria to be motile in viscous mucus
- Urease, which generates ammonia from endogenous urea and thereby elevates local gastric pH
- Adhesins that enhance their bacterial adherence to surface foveolar cells
- Toxins, such as cytotoxin-associated gene A (CagA), that may be involved in ulcer or cancer development by poorly defined mechanisms

7. Intraepithelial neutrophils and subepithelial plasma cells are characteristic of H. pylori gastritis.Neutrophils in the glandular epithelia indicate active gastritis.
8. MALT is mucosa associated lymphoid tissue, that has the potential to transform into lymphoma.
9. Warthin-Starry silver and Giemsa stains are special stains used to demonstrate and confirm H.Pylori.
10. Peptic ulcer disease (PUD) is most often associated with H. pylori–induced hyperchlorhydric chronic gastritis, which is present in 85% to 100% of individuals with duodenal ulcers and in 65% with gastric ulcers
11. Malignant transformation of peptic ulcers is very rare, and reports of transformation probably represent cases wherein a lesion thought to be benign was actually an ulcerated carcinoma from the start.
12. Ménétrier disease is a rare disorder caused by excessive secretion of transforming growth factor α (TGF-α)
13. Approximately 75% of all gastric polyps are inflammatory or hyperplastic polyps
14. Similar to other forms of gastric dysplasia, adenomas almost always occur on a background of chronic gastritis with atrophy and intestinal metaplasia.
15. Adenocarcinoma is the most common malignancy of the stomach, comprising over 90% of all gastric cancers.
16. Chemical carcinogens linked to gastric carcinoma are nitrates and preservatives.
17.e loss of E-cadherin function seems to be a key step in the development of diffuse gastric cancer.

18.Most gastric adenocarcinomas involve the gastric antrum; the lesser curvature is involved more often than the greater curvature

19.Laurens classification of gastric carcinoma :

Gastric tumors with an intestinal morphology tend to form bulky tumors composed of glandular structures , while cancers with a diffuse infiltrative growth pattern (see are more often composed of signet-ring cells.Later carries bad prognosis.

20.In gastric carcinmoma , When there are large areas of infitration, diffuse rugal flattening and a rigid, thickened wall may impart a leather bottle appearance termed linitis plastica\

21. the remarkable decrease in gastric cancer incidence applies only to the intestinal type, which is most closely associated with atrophic gastritis and intestinal metaplasia.

22. In advanced cases gastric carcinoma may first be detected as metastases to the supraclavicular sentinel lymph node, also called Virchow's node.

23. Gastric tumors can also metastasize to the periumbilical region to form a subcutaneous nodule, termed a Sister Mary Joseph nodule, after the nurse who first noted this lesion as a marker of metastatic carcinoma

24.H.Pylori is linked to peptic ulcer , chronic gastritis , gastric carcinoma and gastric lymphoma

25. Carcinoid tumors arise from the diffuse components of the endocrine system. The majority are found in the GI tract, and more than 40% occur in the small intestine

26. Carcinoid tumors are best considered to be well-differentiated neuroendocrine carcinomas.

27. GI stromal tumor (GIST) is the most common mesenchymal tumor of the abdomen, and more than half of these tumors occur in the stomach

28. Approximately 75% to 80% of all GISTs have oncogenic, gain-of-function mutations of the gene encoding the tyrosine kinase c-KIT, which is the receptor for stem cell factor

29.Immunostain for GIST is CD117.

30.Immunostain for lymphoma in gastric region include LCA , CD19,CD20 AND T cell markers.

31.In celiac disease , Some gliadin peptides induce epithelial cells to express IL-15, which in turn triggers activation and proliferation of CD8+ intraepithelial lymphocytes that are induced to express NKG2D, a natural killer cell marker.

32. The histopathology of celiac disease is characterized by increased numbers of intraepithelial CD8+ T lymphocytes (intraepithelial lymphocytosis), crypt hyperplasia, and villous atrophy

33. The most common celiac disease–associated cancer is enteropathy-associated T-cell lymphoma, an aggressive lymphoma of intraepithelial T lymphocytes.

34. Inflammatory bowel disease (IBD) is a chronic condition resulting from inappropriate mucosal immune activation. The two disorders that comprise IBD are Crohn disease and ulcerative colitis.

35. Ulcerative colitis is a severe ulcerating inflammatory disease that is limited to the colon and rectum and extends only into the mucosa and submucosa. In contrast, Crohn disease, which has also been referred to as regional enteritis (because of frequent ileal involvement) may involve any area of the GI tract and is typically transmural

36. Differences very important !

MACROSCOPIC	Crohn's	Ulcerative colitis
Bowel region	Ileum ± colon	Colon only
Distribution	Skip lesions	Diffuse
Stricture	Yes	Rare
Wall appearance	Thick	Thin

MICROSCOPIC		
Inflammation	Transmural	Limited to mucosa
Pseudopolyps	Moderate	Marked
Ulcers	Deep, knife-like	Superficial, broad-based
Lymphoid reaction	Marked	Moderate
Fibrosis	Marked	Mild to none
Serositis	Marked	Mild to none
Granulomas	Yes (~35%)	No
Fistulae/sinuses	Yes	No

37. One of the most feared long-term complications of ulcerative colitis and colonic Crohn disease is the development of neoplasia.

38. Microscopic colitis encompasses two entities, collagenous colitis and lymphocytic colitis

39. The majority of juvenile polyps are located in the rectum and most present with rectal bleeding

40. Peutz-Jeghers syndrome is associated with an increased risk of several malignancies, including cancers of the colon, pancreas, breast, lung, ovaries, uterus, and testicles, as well as other unusual neoplasms, such as sex cord tumors

41. Cowden syndrome is characterized by macrocephaly, intestinal hamartomatous polyps, and benign skin tumors, typically trichilemmomas, papillomatous papules, and acral keratoses

42. the most common and clinically important neoplastic polyps of colon are colonic adenomas, benign polyps that are precursors to the majority of colorectal adenocarcinomas.

43. Colorectal adenomas are characterized by the presence of epithelial dysplasia.

44. Adenomas can be classified as tubular, tubulovillous, or villous based on their architecture

45. Although most colorectal adenomas are benign lesions, a small proportion may harbor invasive cancer at the time of detection. Size is the most important characteristic that correlates with risk of malignancy

46. Familial adenomatous polyposis (FAP) is an autosomal dominant disorder in which patients develop numerous colorectal adenomas as teenagers. At least 100 polyps are necessary for a diagnosis of classic FAP, and as many as several thousand may be present

47. Colon cancers in HNPCC patients tend to occur at younger ages than sporadic colon cancers and are often located in the right colon.(Hereditary non-polyposis colorectal cancer (HNPCC), also known as Lynch syndrome)

48. The dietary factors most closely associated with increased colorectal cancer rates are low intake of unabsorbable vegetable fiber and high intake of refined carbohydrates and fat.

49.2 genetic pathways in carcinoma of the colon include : the APC/β-catenin pathway, which is associated with WNT and the classic adenoma-carcinoma sequence; and the microsatellite instability pathway, which is associated with defects in DNA mismatch repair

50. The classic adenoma-carcinoma sequence, which accounts for as much as 80% of sporadic colon tumors, typically includes mutation of APC early in the neoplastic process

51.APC,K-RAS,p53,SMAD 2 and SMAD 4 are common genetic alterations in carcinoma of the colon.

52. Tumors in the proximal colon often grow as polypoid, exophytic masses that extend along one wall of the large-caliber cecum and ascending colon; these tumors rarely cause obstruction. In contrast, carcinomas in the distal colon tend to be annular lesions that produce “napkin-ring” constrictions and luminal narrowing

53. Cecal and other right-sided colon cancers are most often called to clinical attention by the appearance of fatigue and weakness due to iron deficiency anemia.

54. Left-sided colorectal adenocarcinomas may produce occult bleeding, changes in bowel habits, or cramping left lower quadrant discomfort.

55. Although poorly differentiated and mucinous histologies are associated with poor prognosis, the two most important prognostic factors are depth of invasion and the presence or absence of lymph node metastases in carcinoma of the colon , which are mostly adenocarcinomas.

56.Dukes stage C is lymph node involvement in carcinoma colon.

57. Diagnosis of acute appendicitis requires neutrophilic infiltration of the muscularis propria. Although mucosal neutrophils and focal superficial ulceration are often present, these are not specific markers of acute appendicitis

58. The most common tumor of the appendix is the carcinoid.arising mostly from the tip.

59. In the most advanced cases of appendiceal carcinoma ,the abdomen fills with tenacious, semisolid mucin, a condition called pseudomyxoma peritoneii

60.Immunomarkers for carcinomas are Cytokeratin.

200 KEY POINTS IN SPECIAL PATHOLOGY 4TH YEAR MBBS

1.Key processes in atherosclerosis are intimal thickening and lipid accumulation.

2.Atherosclerotic plaque consists of 3 components: a)cells ,including SMCs,macrophages,and leukocytes,b)ECM and c)Intracellular and extracellular lipid.These components are arranged in central core and fibrous cap.

3.The two most important causes of aortic aneurysms are atherosclerosis and cystic medial degeneration of the arterial media.

4.ANCA are antineutrophil cytoplasmic antibodies seen in patients with vasculitis.c-ANCA are seen in Wegener's granulomatosis and p-ANCA seen in Microscopic polyangiitis and Churg-Straus syndrome.

5.Vasculitis are divided into three types : large vessel including giant cell and takayasu's (granulomas),medium vessel including PAN(fibrinioid necrosis) and Kawasaki's disease and small vessels including Wegener's,ChurgStrauss syndrome and Microscopic polyarteritis.

6.Lobular capillary hemangioma (Pyogenic granuloma) is a common oral benign tumor occurring as polypoidal form attached to mucosa.

7.Vascular immunomarkers include: CD31,CD34 and vWF.

8.Kaposi's sarcoma is the most common AIDS associated cancer in US.The lesion is caused by KSHV and shows three morphological stages : Pact,Plaque and nodule.(PPN).

9.In Myocardial infarction,coagulation necrosis starts in 4 to 12 hrs and collagen deposition starts in 10 to 14 days.

10.Myxoma is the most common primart tumor of heart in adults and Rhabdomyoma is most commonest in children.

11.Aschoff bodies ,pathognominic of Rheumatic fever,are foci of swollen eosinophilic collagen surrounded by lymphocytes ,occasional plasma cells and plump macrophages called Anitschkow cells.

12.The thalassemia syndromes are a heterogenous group of inherited disorders caused by genetic lesions leading to decreased synthesis of either the alpha or beta globin chain of HbA.

13.Causes of enlarged lymph node include : Reactive lymph node enlargement,Infections – commonest is Tuberculosis and Malignancies which may be primary (Lymphoma) or metastatic.

14.Papillary carcinoma is the commonest thyroid malignancy,linked with history of childhood radiation.Diagnosis rests on nuclear features ,the nuclei typically are optically clear (empty)-Orphan Annie appearance,show intranuclear inclusions and intranuclear grooves.

15.Scharff Bloom Richardson system grades the Breast carcinoma.It includes : a) tubule formation ,

b) no of mitoses and c)atypia and pleomorphism.

16.Medullary Carcinoma is linked with amyloid deposition.

17.Meningiomas are divided into 3 categories:a) simple benign type (WHO grade 1),b)atypical –show 4 to 19 mitoses / 10HPF or show 3 of following 5 features: hypercellularity,macronucleoli,necrosis,patternless sheet like arrangement and small cell components with high N/C ratio.,c) Malignant type – more than 20 mitoses per 10 HPF or loss of differentiating features.

18.Syncitial,transitional,fibroblastic,psamommatous and secretory types fall into type 1,Clear cell and chordoid types of meningiomas fall into atypical variety and Papillary and Rhabdoid types fall into malignant variety.

19.Dukes stage C refers to involvement of lymph nodes in carcinoma of the large bowel.

20.Rosenthal fibers,eosinophilic granular bodies and microcysts are seen in low grade Pilocytic astrocytoma.

21.WHO grading of Astrocytoma include : Grade I: low grade , Grade II: Diffuse fibrillary,Grade III: Anaplastic (mitoses and endothelial proliferation) and Grade IV : Glioblastoma Multiforme (necrosis).

22.Leiomyomas are differentiated from leiomyosarcoma on three basis ; atypia , necrosis and number of mitoses (< 5/10HPF: Leiomyoma,5-9/10HPF : Borderiine STUM P and > 10/10HPF: Leiomyosarcoma).

23.Synovial Sarcoma shows dual line of differentiation (both epithelial like and spindly mesenchymal cells are seen).

24.Reed Sternberg cell is the hall mark of Hodgkin’s Lymphoma .Immunomarker is CD 15 and CD 30.The thought of origin is post germinal center B-Cell.

25.Giant cell tumor of bone consists of Mononuclear stromal cells (the main neoplastic element) and evenly placed multinucleated giant cells.

26.Tophi are pathognomonic hallmark of gout,consisting of large aggregates of urate crystals surrounded by an intense inflammatory reaction of macrophages,lymphocytesand large foreign body giant cells.

27.Indian file pattern of tumor cells with monomorphic morphology is seen in Infiltrating lobular carcinoma of breast.

28.Rheumatoid nodules consist of central zone of fibrinioid necrosis surrounded by a prominent rim of epithelioid histiocytes and numerous lymphocytes and plasma cells.

29.Ewing Sarcoma (PNET) of bone consists of malignant round to oval tumor cells with cytoplasm containing glycogen.Immunomarker is CD99(MIC-2)

30.Osteosarcoma is confirmed by the presence of malignant lace like osteoid surrounded by tumor cells.

31.The histologic hall mark of Paget's disease of bone is mosaic pattern of lamellar bone.

32.Peripheral palisading is seen in nests of tumor cells in Basal cell carcinoma (Rodent ulcer).It is locally aggressive tumor, which rarely metastasize. Immunomarker is BER-EP4.

33.Lupus nephritis is of 6 classes : Class 1: no change , class II:

34.Malignant Melanoma shows two patterns of growth : radial and vertical.The nature and extent of the vertical growth phase determine the biologic behaviour of malignant melanoma.

35.Follicular carcinoma of thyroid is confirmed by capsular and vascular invasion by tumor cells.

36.Laurens classification of gastric carcinoma include diffuse (signet ring cells) and intestinal types.

37.Adenoma is the commonest tumor Pituitary gland.

38.Fibroadenoma is commonest benign tumor of breast in young females.

39.Invasive ductal carcinoma of breast is the commonest malignant tumor of breast.

40.Carcinoid tumors common sites include small intestine and tip of appendix.The cells are monomorphic with abundant granular cytoplasm.Immunomarkers include Chromogranin and synaptophysins.

41.Chronic cholecystitis is the inflammation of gall bladder revealing subepithelial fibrosis and chronic inflammation in the lamina propria.

42.Hydatidiform mole is characterized by cystically dilated avascular chorionic villi with trophoblastic proliferation.

43.Choriocarcinoma shows no chorionic villi.

44.Panacinar emphysema is linked with alpha 1 antitrypsin deficiency.

45.Reid index is the ratio of thickness of the mucous gland layer to the thickness of the wall between the epithelium and cartilage.It is increased in Chronic bronchitis.

46.Bronchiectasis is a disease characterized by permanent dilation of bronchi and bronchioles caused by destruction of the muscle and elastic tissue resulting from or associated with chronic necrotizing infections.

47.Asbestos is linked with Mesothelioma and Bronchogenic Carcinoma.

48.Barett's esophagus is linked with esophageal adenocarcinoma,characterized by goblet cells and villous morphology.

49.Pleomorphic adenoma is the commonest benign tumor of salivary gland.

50.Seminoma is the commonest malignant tumor of testis in adults.It is the most radiosensitive.

51.Helicobacter pylori is linked with chronic gastritis,peptic ulcer disease,gastric carcinoma and gastric MALT lymphoma.

52.Active inflammation is signified by the presence of neutrophils within the glandular and surface epithelial layer.

53.Ulcerative colitis is characterized by pseudololyps,superficial ulcers,cryptitis,crypt destruction,crypt distortion and crypt abscess formation.

54.Crohn's disease is characterized by strictures,transmural inflammation,deep linear ulcers,sinuses and granulomas.

55.Cirrhosis of liver is characterized by nodules of variable sizes composed of benign hepatocytes and separated by thin fibrous tissue septae infiltrated by chronic inflammatory cell infiltrate.Active cirrhosis means the presence of piecemeal necrosis.

56.Knodell Score is done to evaluate the grade and stage of hepatitis activity.it comprises of : portal inflammation,piecemeal necrosis,intralobular inflammation and fibrosis.Total score is 24.

57.Metavir score is another criteria of grading and staging of liver activity.It comprises of A for activity and F for fibrosis.

58.Gleason Score is done for the grading of Prostatic adenocarcinomas.

59.Perineural invasion is depicted by prostatic and pancreatic adenocarcinomas.

60.Furrhman Grading is done for Renal cell carcinoma.

61.All patterns of Hepatocellular carcinomas have a strong propensity for invasion of vascular channels.

62.Crescents are seen in rapidly progressive glomerulonephritis,diffuse thickening of the glomerular capillary wall with silver spikes are seen in membranous glomerulonephritis and focal thickening of glomerular basement membrane with silver tram track appearance is seen in membranoproliferative glomerulonephritis.

63.Diabetic glomerulosclerosis is characterized by capillary basement membrane thickening,diffuse mesangial sclerosis ,kimmelstiel-wilson nodules (PAS positive),capsular drops and fibrin caps

64.Chronic pyelonephritis is characterized on gross examination by a corticomedullary scar and on microscopy by tubular atrophy and dilation,thyroidization and chronic interstitial inflammation.

65.Acute pyelonephritis is characterized by patchy interstitial suppurative inflammation,intratubular aggregates of neurtophils and tubular necrosis.Three complications include three P's: Papillary necrosis,pyonephrosis and perinephric abscess.

66.Multiple Myeloma is characterized by bence jones proteinuria,cast nephropathy,hypercalcemia,hyperuricemia and light chain deposition disease.

67.Hyaline arteriosclerosis is benign hypertensive vascular change showing thickening and hyalinization of the walls with narrowed lumina of small vessels.Hyperplastic arteriosclerosis is malignant vascular hypertensive change showing fibrosis with onion skin type layering of wall.

68.Renal cell carcinoma is of 4 major types: clear cell – the most common type,papillary ,chromophobe and bellini duct carcinoma.

69.Renal cell carcinoma has the tendency to metastasize widely before giving rise to any local symptoms or signs.

70.WHO grades Transitional cell tumors into Urothelial papilloma,urothelial neoplasm of low malignant potential,low grade papillary urothelial carcinoma and high grade papillary urothelial carcinoma.

71.PT1 is for lamina propria and PT2 is for muscle wall invasion in Transitional cell tumors.

72.Seminomatous germ cell tumors are radiosensitive and chemo responsive while non seminomatous tumors are vice versa.

73.Yolk sac tumor shows Schiller Duval bodies,Granulosa cell tumor shows Call exner bodies.

74.Immature or malignant teratomas show sheets of undifferentiated cells,not seen in a benign teratoma.Teratomas in males are malignant,unless proved otherwise.

75.Alpha fetoprotein is the tumor marker of HCC and CEA is the tumor marker of carcinoma of the colon.

76.Human Papilloma Virus is the etiologic agent of Squamous cell carcinoma of the cervix ,the most common cervical carcinoma.(types 16,18,31,33).

77.Bethesda system divided into ASCUS- atypical suamous cells of undetermined significance,LSIL-low grade squamous intraepithelial lesion(Koilocytosis and mild dysplasia) and HSIL – High grade squamous intraepithelial lesion (Moderate and severe).

78.Dysgerminoma is the counterpart of seminoma in females.

79.Tubular carcinoma of breast carries the best prognosis.

80.ER – PR postivie breast carcinomas carry better prognosis and are responsive to antiestrogen drugs.HER-2/neu positive tumors carry poor prognosis.

81.Squmaous cell carcinoma is characterized by sheets and groups of pleomorphic malignant squamous epithelial cells with hyperchromatic nuclei and pale cytoplasm.Individual cell keratinization.intracellular bridges and keratin epithelial pearls are seen.

82.Cytokeratin is the immunomarker for epithelial tumors and Vimentin is the marker for mesenchymal tumors.Desmin is positive in skeletal muscle tumors. Melan-A and HMB45 is positive for Melanomas.LCA is the universal leukocyte marker.

83.CD19,CD20 AND CD79a is positive in B-cells and CD 2,3,4,5 in T cells.

84.Adenocarcinomas are characterized by back to back glandular structures lined by pleomorphic malignant epithelial cells with hyperchromatic nuclei and eosinophilic cytoplasm.Glandular wall sharing is one of the hall mark sign of adenocarcinomas.

85.Acute appendicitis is depicted by the presence of neutrophils in the muscularis layer.

86.Small cell carcinoma of the lung shows maximum paraneoplastic syndromes – most common cushing like picture and ADH like morphology.Hypercalcemia is shown by squamous cell carcinoma of lung.

87.Hodgkin's Lymphoma is divided into five types : one set include Lymphocyte rich,lymphocyte depletion,nodular sclerosis and mixed cellularity (CD 15 +,CD 30 +).The separate set includes nodular lymphocyte predominance (CD45 +).

88.Burkitt's lymphoma carries 100% mitotic index.

89.Benign prostatic hyperplasia includes two components : Glandular and fibromuscular.DHT is the source of hyperplasia.

90.Endometriosis is the presence of normal endometrial glands,stroma and hemosiderin laden macrophages outside uterus.

91.Calretenin is the immunomarker for mesothelioma.Epithelioid and sarcomatoid types are noted in a mesothelioma.

92.Squamous cell carcinoma of lung is strongly associated with smoking.

93.PTEN is the antioncogene altered in endometrial hyperplasia and adenocarcinoma.Simple and atypical are two types of endometrial hyperplasia.EIN is the new term assoiated with endometrial hyperplasia.Endometrial carcinoma arises on two backgrounds: hormone dependent with good prognosis and vice versa with poor prognosis(clear and papillary types).

94.The primary feature of primary biliary cirrhosis is the non suppurative inflammatory destruction of medium sized intrahepatic bile ducts.90% patients are positive for antimitochondrial antibodies.

95.Hashimoto's thyroiditis shows lymphoid follicles with germinal centers and hurthle cells.DeQuervain's type shows presence of granulomas.

96.Pheochromocytoma is composed of polygonal to spindle shaped chromaffin cells clustered with the sustentecular cells into small nests of alveoli (Zell ballen appearance) by a rich vascular network.

97.MEN TYPE 1 or Wermer syndrome shows abnormalities in 3 P's : Parathyroid,pancreas and pituitary.MEN -2A or Sipple syndrome shows pheochromocytoma,medullary carcinoma thyroid and parathyroid hyperplasia.MEN2-B is same as 2A,with the exception of parathyroid involvement.

98.Round blue cell tumors of childhood include tumors with blue cell morphology with scanty cytoplasm,e.g Lymphoma,Neuroblastoma,Medulloblastoma and Retinoblastoma etc.

99.Chicken wire calcification is seen in chondroblastoma.

100.Antoni A cellular areas and Antoni B myxoid areas are seen in Schwanommas.

Few Characteristics Exclusive to Lesions - Rapid revision:

101.Peripheral Palisading – Basal cell carcinoma.

102.Pseudopalisading in tumor – GBM.

103.Whorls like arrangement – Meningioma.

104.Signet ring cells – Poorly differentiated mucin secreting adenocarcinoma.

105.Keratin pearls – Squamous cell carcinoma.

106.Verocay bodies (Antoni A and B) – Schwanomma.

107.Call exner bodies – Granulosa cell tumor.

108 .Psamomma bodies – Papillary carcinoma thyroid,Papillary serous ovarian tumors,Meningioma.

109.Capsular / vascular invasion – Follicular carcinoma thyroid.

110.Amyloid – Medullary carcinoma thyroid.

111.Osteoid formation – Osteosarcoma.

112.Chicken wire calcifications – chondroblastoma.

113.Schiller Duval bodies – Yolk sac tumor.

114.Indian file pattern – Lobular carcinoma breast.

115.Piecemeal necrosis – Chronic active hepatitis.

116.Mallory bodies – Alcoholic cirrhosis.

117.Stellate cells – Cardiac myxoma.

118.Oat cells – Small cell carcinoma lung.

119.Hair in tumor – Benign cystic teratoma (dermoid cyst)

120.Reed Sternberg cells – Hodgkin's Lymphoma.

Typical descriptions / Key Points of Few common major Lesions :

121.Carcinoid tumor : nests,aggregates and packets of monomorphic cells with round to oval nuclei and granular cytoplasm.Chromogranin positive.

122.Adenocarcinoma : Back to back closely packed gland like structures lined by pleomorphic malignant epithelial cells with hyperchromatic nuclei and eosinophilic cytoplasm.Wall sharing is noted.

123.Squamous cell carcinoma : sheets and clusters of pleomorphic malignant squamous epithelial cells with hyperchromatic nuclei and pale cytoplasm.Keratin epithelial pearls,intercellular bridges and individual cell keratinization are seen.

124.Hemangioma : Aggregates of closely arranged thin walled vascular channels lined by benign endothelium and separated by scanty fibrous stroma.

125.Angiosarcoma: Sheets of malignant oval to spindly cells with hyperchromatic nuclei.Mitoses are noted.

126.Sarcoma : Sheets of malignant oval to spindly cells with hyperchromatic nuclei .Abnormal mitoses and focal necrosis is noted in high grade sarcomas.

127.Papillary carcinoma thyroid : Papillary structures with fibrovascular cores lined by pleomorphic malignant epithelial cells with Orphan Annie nuclei.Nuclear Grooves are seen.Psamomma bodies are present.

128.Seminoma : Sheets and groups of seminoma cells with hyperchromatic vesicular nuclei and water clear cytoplasm.These tumor cells arranged in lobules separated by fibrous tissue septae infiltrated by lymphocytes.

129.Yolk sac tumor : sheets and nests of pleomorphic malignant oval to polygonal cells forming schiller duval bodies.Eosinophilic secretions are noted.

130.Meningioma : whorls and bundles of oval to fusiform to polygonal cells with regular nuclei.Psamomma bodies may also be seen.

131.Non Hodgkin''s Lymphoma : Sheets of monotonous population of atypical lymphoid cells with hyperchromatic nuclei and scanty cytoplasm.Mitoses are noted.

132.Hodgkin's Lymphoma : Heterogenous population of cells including lymphocytes,plasma cells,macrophages and eosinophils (Characteristic feature).Reed Sternberg cells are hall mark.

133.Basal cell carcinoma (Rodent ulcer) ; Nests and groups of pleomorphic basaloid cells with hyperchromatic nuclei and scanty cytoplasm.Peripheral palisading is noted at the periphery of nests.Mitoses can be seen.

134.Malignant melanoma : Sheets of malignant oval to polygonal cells with hyperchromatic nuclei and prominent pink nucleoli.Pigment deposition is noted.

135.Ductal carcinoma breast : Ductal structures,sheets and groups of pleomorphic malignant epithelial cells with hyperchromatic nuclei and eosinophilic cytoplasm .

136.Liver cirrhosis : Nodules composed of benign hepatocytes separated by thin fibrous tissue septae.

137.Teratoma : Cystic lesion lined by benign stratified squamous epithelium and cyst wall showing hair follicles,sebaceous glands.Other types of tissue like nervous,thyroid tissue can be seen.

TYPICAL immunomarkers :

138. Cytokeratin – Carcinomas.

139.Vimentin – Sarcomas.

140.S-100 – Melanoma , nEural marker.

141.Melan – A.Malignant melanoma.

142.Desmin – Muscle marker.

143.LCA – Leukocyte common antigen – Lymphomas.

144.Chromogranin – Neuroendcrine marker ,carcinoid.

145.CD 15 & CD 30 – Reed Sternberg cells / Hodgkin's.

146.CD19,CD20,CD79a – B cell Lymphoma Marker.

147.CD3,4,5 ,8 – T cell Lymphoma Marker.

Famous GRADING / STAGING to be Learnt by heart:

148.Bloom Richardson grading : Breast carcinoma.

149.Gleason's Score : Carcinoma Prostate.

150.Duke's Staging : Carcinoma colon .

151.TNM Staging of Lung.

152.Fuhrmann's Grading : Renal cell carcinoma.

153.Knodell score : Chronic hepatitis / cirrhosis / liver activity.

154.FIGO grading / staging : Carcinoma endometrium and cervix.

155.Meningioma : WHO grading.

156.WHO grading of Astrocytoma.

157.Lauren's Classification : carcinoma stomach.

158.Hodgkin's Lymphoma staging. (Ann Arbor)

Important DIFFERENTIAL DIAGNOSIS in special pathology "

159. Cold nodule thyroid : Benign hyperplastic colloid nodule,follicular adenoma,Carcinoma thyroid, thyroid cyst / nodule.

160.Enlarged cervical lymph node : Reactive lymph node enlargement ,Tuberculous lymph node,Lymphoma , Metastatic malignancy.

161.Lump Breast in 40 yrs old female : Carcinoma breast,fibrocystic disease,fibroadenoma,non specific abscess.

162.Posterior fossa brain tumors : Medulloblastoma,pilocytic astrocytoma,meningioma,schwanomma.

163.Ulcer Intestine : Tuberculosis,Typhoid,Inflammtory bowel disease,Carcinoma,Perforation.

164.Brown black pigment in liver : Haemochromatosis,Bile pigment in cholestasis,lipofuscin pigment in aging,fomalin pigment,metastatic melanoma.

165.follicle like arrangement in thyroid : Follicular adenoma,follicular carcinoma,follicular variant of papillary carcinoma thyroid.

Some other Last Points in Special Pathology not to be missed:

167.Active gastritis or colitis means presence of polymorphs .

168.crypt abscess means presence of collections of neutrophils in glands or crypts lumen.

169.Clinical outcome of a malignant tumor rests mainly on staging/lymph node invasion / mets.

170.LFT's mean Serum billirubin,SGOT,SGPT,serum albumin,LDH.

171.RFT's mean BUN and serum creatinine.

172.Thyroid function tests include TSH,T4,T3.

173.Cardiac Profile include : Troponin,Creatine Kinase ,LDH.

174.Lipid Profile include : Cholesterol,HDL,LDL,VLDL,Triglycerides.

175.Special Liver stains include : PAS,PAS-D,Reticulin.

176.Methenamine silver and PAS stains are done for kidney biopsies to see thickening of basement membrane of capillaries.

177.ZN stain is done to look for AFB (Acid fast bacillus) in Tuberculosis.

178.Van gieson and masson trichrome stains are done to look for collagen fibers / fibrosis.

179.Alcian blue is the stain used to look for mucin.

180.GMS stain is done for fungus. (Gomori methenamine silver)

181.Sudan black and Oil Red 0 are special stains for fat.

182.Perls Prussian blue stain is used to look for iron.

183.The most frequent causes of mild anemia with hypochromia and microcytosis are iron deficiency anemia,the anemia of chronic disease and b thallesemia minor.

184.The granulomas of sarcoidosis are characteristically non caseating.

185.The hallmark of the megaloblastic anemia is the finding of megaloblastic erythroid hyperplasia in the bone marrow.

186.Nodular melanoma has two pattern of growths,horizontal and vertical.It tends to spread vertically more than horizontally.

187.The majority of salivary gland tumors occur in parotid and most common tumor is Pleomorphic adenoma.

188.Adenoid cystic carcinoma is a common malignant salivary gland tumor.

189.Biopsy is always preferable over FNAC.

190.Biopsies are of many types : Excision biopsy,Trucut biopsy,Incisional biopsy.

191.Routine histopathological stain is Haematoxylin and Eosin.

192.Routine cytological stain is Giemsa and Papanicoalau(for nucleus).

193.Common Histo fixative is 10 % Formalin.

194.Common Cytology fixative is 95% ethyl alcohol.

195.Special stains / Immunostains are needed when diagnosis cannt be made with a Routine stain.

196.Kaiserling solution is used to keep specimens fixed in museums for long term.

197.When give Differential diagnosis of any lump always go in this order : infections,benign tumors and than malignant tumors.

198.FNAC common sites for superficial lumps – lymph nodes,salivary glands,thyroid and breast.

199.FNAC deep lumps common sites include – Liver,lung and abdominal lymph nodes.

200.FNAC of deep seated lumps need help of Ultrasound /CT.