Name	Cause	Notes	Histology	Treatment
Hodgkin Lymphoma [HL]: arises from germinal B-cells centres, mainly involves the axillary, lower cervical and mediastinal LNs and spreads to the adjacent ones, B-symptoms [fever, night sweats, weight loss..],cells express programmed death receptor [to evade immunity] and that receptor is the main target in therapy				
Nodular sclerosis HL [classic]		-most common mainly in *children and young adults* -presents as enlarged cervical or mediastinal lymph node mainly in young females*	-Nodules separated by fibrous bands [sclerosis] in the lymph node -RS cell forming lacunar cells [*clear cytoplasm - white like a moon*]	
Mixed cellularity HL [classic]		-common in *old people* -associated with EBV infection -eosinophilia: IL-5 produced by RS cells attract them	-Few neoplastic [RS or Hodgkin mononuclear] cells with a background filled with lymphocytes and eosinophils -No sclerosis *[remember mixed cellularity as multiple cell types; neoplastic, lymphocytes and eosinophils]*	
Non-classic lymphocyte-predominant HL		-cells do not express CD15,30 but express CD20,45 -excellent prognosis	-Lymphohistiocytic variant RS [LP cells] -Popcorn cells [vesicular with whte lobulated nucleus and small blue nucleoli -Large follicles arranged in nodules with no fibrous septa	
Non-Hodgkin lymphoma [NHL]				
Follicular Lymphoma	Bcl2 -> IgH translocation t(14;18) leading to overexpression of Bcl2* ⅓ of the patients have mutations in genes encoding histone-modifying proteins	-mainly common in *old [>50] males, and westerns more than asians* -second most common NHL -low grade -B-cells germinal centre express CD20,10, Bcl6 and ***Bcl2*** -Indolent	-Nodular proliferation of B-cells forming follicles that disturb that architecture of the LNs [unlike follicular hyperplasia*] -Centrocytes -> large, irregular and *low grade* -Centroblasts -> vesicular nucleus and small nucleoli and *high grade* [still maturing]	-Chemotherapy is ineffective in early stages -Chemotherapy only in high-grade and symptomatic patients -Drugs -> anti-CD20 [targets normal and neoplastic cells, bye bye immunity?], anti-Bcl2
Diffuse large B-cell lymphoma	-Mutant Bcl6 -> activating the proliferation of B-cells Bcl2 -> IgH translocation t(14;18) -Mutant MYC gene -> activating the cell cycle	-most common [non-cutaneous extranodal] NHL, *mainly in adults* -high-grade -positive for CD20 **subtypes** 1. De novo 2. secondary DLBCL both arise from a low-grade tumour 3. primary mediastinal DLBCL, in the thymus most commonly in women 4.EBV-associated DLBCL, in immunosuppressed patients 5. HHV-8 DLBCL, primary effusion lymphoma, in immunosuppressed, cyclin D1 mimicker protein*	-Diffuse cells disturbing the morphology of the lymphoid -Abnormally large lymphocytes with irregular nucleus and small, prominent nucleoli	
Burkitt lymphoma	MYC -> IgH translocation t(8;14) [~~burkitt~~, burkeight - eight;14] *thanks for the trick pathoma :p* , warburg metabolism, more anabolic activity	-originate from B-cells germinal centres -most common *NHL** in *children* -endemic in Africa (in the jaw), sporadic in the rest of the world (in the abdomen or CNS), [20% EBV infection] -Immunodeficiency BL -express CD20,10 & Bcl6 -the fastest growing human cancer	-intermediate, monomorphic cells with round or oval nucleus and multiple nucleoli [high mitotic activity] -tingible body macrophages* -*starry sky* appearance -lipid vacuoles	
Extranodal marginal zone lymphoma		-more mature B-cells -second most common extranodal [DLBCL first] -indolent -associated with chronic inflammation [autoimmune diseases or H.pylori gastritis] -MALToma in mucosal sites	-infiltration and destruction of the epithelium	
Mantle cell lymphoma	Cyclin D1 -> IgH	-arises from naive B-cells	-centrocytes in a diffuse	

	translocation t(11;14)	-most common in *older men* -affects the lymph nodes and Waldeyer's ring -lymphomatoid polyposis [submucosal nodules] -negative for CD10 and Bcl6	pattern [no follicles-	
Small lymphocytic lymphoma (in LNs) [chronic lymphocytic leukemia (in blood and BM)]	-deletion mutation causing increased Bcl2 -active B-cell receptor which activates bruton tyrosine kinase and promotes cell survival -patients with p53 mutations have worse prognosis	-low-grade, mature B-cells neoplasm -affects *elderly, mainly in western countries* -CLL is the most common leukemia in adults -express CD,1920, Bcl2 and CD5 [weird -> T cell marker] -leukocytosis [>200000] -hepatosplenomegaly and LAD in 50% -immunosuppression in 50% -> hypogammaglobulinemia -anemia [ABs against B-cells -> cold IHA] and thrombocytopenia -Ritcher transformation -> predominance of large cells	**SLL** -small, round neoplastic cells with dark chromatin infiltrate the lymph nodes along with large *prolymphocytes* -proliferation centres, high mitotic activity **CLL** -leukemic cells similar to lymphocytes, occasional prolymphocytes and *smudge cells*	
Precursor B&T cells neoplasm	-mutations in RAS and tyrosine kinase proteins promoting cells survival -T-LL -> NOTCH mutation in 70% -B-LL -> PAX5 mutation -T-ALL mutations in PTEN, CDKN2A -*childhood* B-ALL hyperdiploidy, ETV6 and RUNX1 mutations t(12;21) -adult B-ALL ABL, BCR t(9;22) -> encodes new tyrosine kinase protein	-immature blasts, aggressive -express CD34, TDT Lymphoblastic lymphoma -> in LNs [T is more common] Acute lymphoblastic leukemia -> in blood and BM [B is more common *B-ALL more common than T and most common *childhood* malignancy in childhood, disseminates to solid organs ***T*-ALL in ***T**eenagers*, **T**hymus, boys -anemia and thrombocytopenia and damage to solid organs secondary to leukemic infiltration	-large blasts with little, agranular cytoplasm -pale chromatine	B-ALL -> favourable [age 2-10, low WBC count] responds to chemo, good prognosis unfavourable [age <2, adolescents or adults, high WBC count] poor prognosis
Plasma cell myeloma [multiple myeloma]	-cyclin D1 or D3 -> IgH translocation t(11;14) -MYC gene mutation	-plasma cells neoplasm -most common in *elderly* -plasma cells secrete monoclonal proteins, sometimes only light chains can be found in urine ***Bence Jones proteins*** [diagnostic] -malignant plasma cells activate expression of NF-κB ligand RANKL activating osteoclasts and causing *bone lesions* -pathologic fractures, hypercalcemia -anemia -renal failure [due to obstruction or hypercalcemia -> kidney stones] -immunosuppression -high ESR -amyloidosis* -pancytopenia	-RBCs rouleaux formation [characteristic] -in the BM increased plasma cells and abnormal, multinucleated figures with cytoplasm full of Igs-containing vacuoles	-slowly growing -> not treated by conventional chemotherapy -Lenalidomide -> inhibits oncogenic proteins -proteasome inhibitors
Hairy cell leukemia	-mutations in serine/threonine kinase BRAF gene	-uncommon,low-grade B-cell leukemia -old patients mainly *smoking men* -pancytopenia and splenomegaly [infiltration of these organs], BM fibrosis	-few leukemic cells with prominent cytoplasmic hairy projections	very sensitive to chemotherapy
Peripheral T-cell lymphoma		-most common *mature* T-cell lymphoma -aggressive -severe inflammation [neoplastic cells secrete cytokines] -cells express CD2,3,5,7 and negative for TDT		
Cutaneous lymphoma	Mycosis fungoides and sezary syndrome	-most common cutaneous lymphoma -*CD4+* T-cells -a history of erythema which progresses to plaque and then tumour -most commonly at the junction between dermis and epidermis	-lymphocytes with irregular [cerebriform] nucleus	
Adult T-cell lymphoma/leukemia	human T-cell leukemia virus 1 [HTLV-1],TAX protein causes: 1.proliferation of the host cell PI3 kinase and cyclin D1->suppresses expression of CDK inhibitors->activates NF-κB -> cell survival 2. genomic instability	-*CD4+* T-cells -sporadic, endemic in japan, west africa and the caribbeans -skin lesions, LAD and lymphocytosis, hypercalcemia -hepatosplenomegaly -express CD25 [characteristic]		

*pathoma extra information in ***Red***
* Bcl2 is an anti-apoptotic
* follicular hyperplasia is benign and associated with HIV or rheumatological diseases and does not disturb the architecture of the LNs and negative for Bcl2
*Cyclin D1 keeps the cell cycle going and promotes the transition from G1 to S
*most common lymphoma in children *generally* is Hodgkin lymphoma
*tingible body macrophages have the ability to engulf apoptotic lymphoid cells
* build-up of abnormal proteins [amyloid] in tissues and organs

- Cell markers -> RS cells : *CD15,30* [**markers of classic HL**] but not CD3,20 or 45,
All B-cells: *CD19,20,22,* germinal centres : *CD10,Bcl6*, plasma cells: only *CD138*, All mature T-cells: *CD2,3,5,7*, All blasts: *CD34*, lymphoblasts: *CD10, TDT*

- Anemia and thrombocytopenia: SLL, CLL, Precursor T&B cells neoplasm, multiple myeloma [pancytopenia], hairy cell leukemia [pancytopenia]
- Hepatosplenomegaly: CLL, hairy cell leukemia [splenomegaly], adult T-cell leukemia
-Hypercalcemia: plasma cell myeloma, adult T-cell leukemia