↳ AML (Acute myeloid leukemia):

- Acquired genetic mutation, incidence increase with age.
. WBC ⬆️ >10K- not functional cells, thrombocytopenia (leading to bleeding symptoms).
. Blasts ⬆️ > 20%
. Extramedullay filtration [Hypertrophy gum, hepatomegaly, spleenomegaly]
. Expansion of bone marrow, causing sturnum & back pain
. Hypocalcemia, Hyperurecemia, Hyperphosphotemia, hyperKalemia, Metabolic acidosis, DIC

* 2 Types: de novo AML / Secondary AML (Myelodysplastic syndrome [<20% Blasts]/ Myeloproliferative/ radiation - chemotherapy).

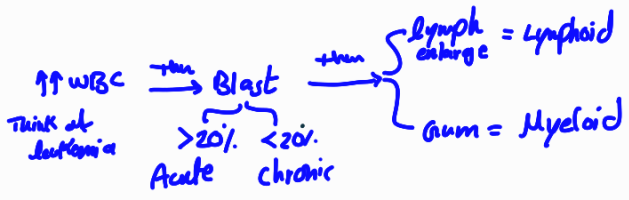
* 2 Mutations: uncontrolled proliferative then impaired differentiation.

* Risk factors: Secondary AML/ chemicals/ syndromes (down, neurofibromatosis)

* Treatment: 1) Induction (remission) → High dose of chemo, 1_4 weeks of bone marrow suppression.
2) Consolidation (post remmission) → Cyclic Chemotherapy or BM transplant.
#NO Maintenance

* Subtypes: ⇒ Promyleocytic M3: (t 15,17) involving RAR gene/ GOOD prognosis 👍/ associated with DIC / Auer rods found/ Treatment → Tretinoin drug.
⇒ Myelomonocytic M4: inverted (16)/ associated eosinophilia/ GOOD prognosis 👍/ associated with leukemia cutis (skin)/ CNS disease may occur.
#BOTH favorable category.
#Tretinoin does NOT produce DIC, but produce Retinoic Acid Syndrome complication.

↳ ALL (Acute lymphoid leukemia):



- No association with age.
. ⬆️Tdt, ⬆️CD19, CD10
. ⬆️ WBC ⬆️Blasts >20%, pancytopenia.
. CNS, Testicular involvement more that AML/ Gout / DIC / Hyperviscosity
* Classifications → Morphology (depending on blast size) / Immunophenotype (B- lineage 80%= most common: Pro-B , T- linage 20%= Most common Pre-T)

* Subtype : t(9,22) ⇒ BCR/ABL (very poor prognosis)
* High risk ALL :

1. Pre - T
2. Pro - B
3. Age > 35 years,
4. WBC > 30 G/L in B-ALL
   > 100 G/L in T-ALL
5. No remission after 4 weeks of induction therapy
6. Chromosome Philadelphia - positive or BCR/ABL (+)

* Treatment: Induction → consolidation (cycle chemo - BM Transplant) → Maintenance + CNS proplaylaxis

↳ CLL (Chronic lymphoid leukemia):

. Most common luckamia, Mainly elderly, Male predominance.
. Lymphocytesis, enlarged lymph nodes (painless, bilateral), Spleenomegaly
.⬆️ WBC (indicates leukemia), <20% Blasts (indicats Chronic), DAT +3 (indicates for autoimmune hemolysis).
. B lymphocytes look mature but they're malignant→ Aressted between stage pre_B & mature B-cell).
. 🚫 CD10, ✅ CD 19, 20, 5
. Polychromesia
. **Smadge** cells
. Findings found incidentally, then processe & symptoms appear.
* ⇒ Clinical Staging: [Rai/ Binet]→ depend on presence of lymphocytosis & degree of organomegaly & thrombocytopenia.
⇒ Cytogenic Staging

* Mutation of VH genes→ Unmutated [rapid prognosis] / Mutated [Slow prognosis].

* Treatment: NOT all patients need treatment, until B- Symptoms start to appear.
#ALL,CML need treatment regardless of clinical status!
Symptoms: (1) B-symptoms: weight loss, night sweats, fever, fatigue.
(2) ⬇️Hb (3) lymphadenopathy (4) Hepatosplenomegaly if progressive
⇒ Chemotherapy

↳ CML (Chronic myeloid leukemia):

. fatigue, malaise, weight loss → accidentaly discovery
. Spleenomegaly
. ⬆️WBC, <20% Blasts 🚫lymphadenopathy
. **Basophilia- Eosinophilia**/ Granulocyte of different stages of maturation.
* Test by→ BM, Karyotyping, FISH.
* **Phases**: Chronic → accelerated (blasts 10-20%) → blast crisis(blasts > 20%)⇒ AML
* t(9,22) fusion of BCR on chromosome 22 with ABL from chromosome 9
* Treatment: Anti-tyrosin kinase for philadelphia [Target therapy] ⇒ Imatinib (competitive inhibition)
→ Side effects: (1) Myleosuppression (2) Resistance, other treatment: 2nd generation of TKI/ Chemotherapy / Transplant