

Leukemias, Student classification

Leukemias

Abnormal increase of immature white blood cells – blasts. -> acute Either lymphoid or myeloid origin. Leukocytosis
Neoplastic cell infiltrate in BM -> disrupts normal cell maturation -> anemia, immunocompromisation,
thrombocytopenia Infiltration of liver LNs other organs is common

Myeloid: 1) acute myeloid leukemia immature neoplastic myeloblasts stopped early in maturation, predominance in
BM and peripheral blood often occurs in adults less responsive to therapy than ALL Auer rods – azurophilic, MPO All
trans retinoic acid – acute promyelocytic leukemia

2) chronic myelogenous leukemia adults 40-50y neoplastic cells capability of further differentiation – granulocytic
elements small number of blasts 9:22 = philly, merging of bcr abl gene responsive to targeted therapy of tyrosine-
kinase bcr-abl prominent SPLENOMEGALY LAP(leukocyte alkaline phosphatase) neg. increased basophils,
distinguished from leukemoid reaction

Acute leukemias have a more stormy onset: ca 3 months with suppression of normal BM function -> cytopenias
Without treatment 6-12month survival

Lymphoid: Common in childhood(acute) 1) ALL (fdT nuclear staining) Mostly pre B cells in children (CD20), pre T
cells more in adolescents (CD3). Predominance of lymphoblasts in circulation and BM Responsive to therapy
Clumped chromatin, PAS positivity, more than 25% neoplastic cells in BM

2) CLL/SLL Almost always B cells Widespread infiltration of nonfunctional B cells in BM, liver, LNs, spleen Neoplastic
cells CANNOT differentiate into plasma cells -> Hypogammaglobulinemia Smudge cells, lymphocytosis in CLL,
lymphadenopathy (SLL), hepatosplenomegaly

3) Hairy cell leukemia B cells with hair like filamentous projections acc. in red pulp Middle aged males,
splenomegaly, pancytopenia, TRAP staining