Acute Myelogenous Leukemia (AML)

Acute myelogenous leukemia (AML) is characterized by an accumulation of immature myeloid forms, also called blasts, caused by acquired oncogenic mutations that impede the normal differentiation process. The accumulation of these immature blast forms suppress normal hematopoiesis and result in bone marrow failure and complications related to anemia, thrombocytopenia, and neutropenia. These neoplasms can occur at all ages but incidence rises throughout life and the median onset is at 60 years of age. There are several different types of AML and can be classified according to the French-American-British (FAB) classifications which divide AMLs into subtypes based on the degree of differentiation and the lineage of the leukemic blasts. The cytoplasm of myeloblasts often contains Auer rods, which are distinctive needle like granules. Auer rods are particularly numerous in the M3 FAB subtype, which is also associated with a (15;17) translocation called acute promyelocytic leukemia. The t(15;17) creates a fusion gene that codes a part of the retinoic acid receptor alpha fused to a protein called PML. This fusion protein can interact with transcriptional repressors causing the inhibition of granulocytic maturation. In addition, this form of AML is commonly associated with bleeding caused by disseminated intravascular coagulation caused by severe thrombocytopenia. A different type of AML, the M5 subtype, is characteristic for gum infiltration which can help differentiate this subtype from others. Treatment of AML depends on the type but can involve chemotherapy or bone marrow transplant. The M3 subtype is unique and responds to pharmacologic doses of all-trans retinoic acid, a form of vitamin A, by binding with the aforementioned fusion protein and antagonizing its inhibitor effect on the transcription of target genes.



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Myeloblasts Increase on Peripheral Smear
Mile post blasting
Acute myelogenous leukemia (AML) is characterized by an accumulation of immature myeloid forms, also called blasts, caused by acquired oncogenic mutations that impede the normal differentiation process. Therefore the number of circulating myeloblasts are typically increased on peripheral smear.

Median Onset 60 Years
(60) six old monkeys
These neoplasms can occur at all ages but incidence rises throughout life and the median onset is at 60 years of age.

T 15;17 M3 Subtype
Mile post 15;17 with (3) Tree in between
The M3 FAB subtype is associated with a (15;17) translocation called acute promyelocytic leukemia (APML or APL). The t(15;17) creates a fusion gene that encodes a part of the retinoic acid receptor alpha fused to a protein called PML. This fusion protein can interact with transcriptional repressors which result in the inhibition of granulocytic maturation.

Auer Rods
Hourglass on a rod
The cytoplasm of myeloblasts often contains Auer rods, which are distinctive needle-like granules. Auer rods are particularly numerous in the M3 FAB subtype and identification of these rods on histology can help with disease diagnosis.

Disseminated Intravascular Coagulation (DIC) is a Common Presentation
Dice
In addition, this form of AML is commonly associated with bleeding caused by disseminated intravascular coagulation (DIC) caused by severe thrombocytopenia.

M5 Gum Infiltration
(M) Monkey (5) Hand Bubble-gum
Of note, the M5 subtype (Acute Monocytic Leukemia) is characteristic for gum infiltration, which can help differentiate this subtype from others.

Responds to Vitamin A
Viking (A) Apple cutting down tree
Translocation guides therapy because these tumors respond to pharmacologic doses of all trans retinoic acid (a form of vitamin A) by binding with the fusion protein and antagonizing its inhibitor effect on the transcription of target genes. The M3 subtype of AML, otherwise known as APL, is the one that is responsive mainly to this treatment.

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