



Acute Myelogenous Leukemia (AML) Acute Non-Lymphoblastic Leukemia (ANLL)

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Acute myelogenous leukemia (AML) arises from the [\(A – 1\) myeloid cell lineage](#) and accounts for 15%-20% of all leukemia seen in childhood. This form of leukemia arises from malignantly transformed myeloid progenitor cells that produce leukemic blasts that give rise to the neutrophil, monocyte, erythrocyte, megakaryocyte cell lines. The [incidence of AML](#) is higher during the first year of life and during the adolescent years.

Risk Factors

Genetic risk factors include the presence of AML in a sibling: an identical twin has a 20% chance of developing AML before age 6 years. [\(A – 2\) Syndromes](#) that arise from congenital bone marrow failure, chromosome instability, and DNA abnormalities are also known to increase the risk for AML.

[\(A – 3\) Acquired risk factors](#) such as ionizing radiation, chemicals, and cytotoxic agents also increase the risk for AML. While there are reports implicating retroviruses as predisposing factors, the link is weak and currently unsubstantiated.

AML can also be caused by the use of therapies using agents that increase the risk of developing leukemias, such as radiation, and epipodophyllotoxins. The risk is usually greatest four to five years after therapy and continues for at least 8 years after completion of therapy.

Clinical Signs & Symptoms:

The presenting [\(A – 4\) symptoms](#) of AML generally reflect the altered production of red blood cells, granulocytes and platelets (anemia, infections, and hemorrhage). Hence a life-threatening infection (sepsis) or hemorrhage is a common presenting feature. The anemia is usually normocytic and normochromic; patients may complain of fatigue, pallor, headaches, tinnitus, and respiratory discomfort (dyspnea). Congestive heart failure can be rarely present at diagnosis.

Thrombocytopenia accounts for the bruising, epistaxis, and gingival bleeding; and the absolute neutrophil count of <1000 cells/uL increases the predisposition for infections. The most common sites for infections include the lungs, gingiva, sinuses, skin, and dental and perineal areas.

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About half of the children with AML have hepatosplenomegaly; and lymphadenopathy is commonly seen if there is a monocytic component to the leukemia. [\(A – 5\) Chloromas and myeloblastomas](#) may arise in bones or soft tissue, occurring mostly in the epidural and orbital areas.

CNS involvement may be manifested by cerebral myoblastoma or as a typical meningeal infiltration with or without cranial nerve palsy. Symptoms of CNS involvement include headaches, nausea, vomiting, photophobia, papilledema and cranial nerve palsies. [\(A – 6\) Skin lesions](#) such as leukemia cutis are often colorless or have a purplish color (blueberry muffin); and are often seen in neonates with acute monocytic leukemia (FAB M5).

Classification of AML

There are several classification systems for AML. The most commonly used system is the French-American-British (FAB) system.

[A – 7 FAB classification](#) of acute myeloid leukemia

- M0 Acute myeloid leukemia with minimal evidence of myeloid differentiation
- M1 Acute myeloblastic leukemia without maturation
- M2 Acute myeloblastic leukemia with maturation
- M3 Acute promyelocytic leukemia (APL)
- M4 Acute myelomonocytic leukemia
- M5 Acute monocytic/monoblastic leukemia
- M6 Acute erythroleukemia
- M7 Acute megakaryoblastic leukemia

Because FAB classification does not take into account cytogenetic findings, there has recently been an attempt to combine the morphologic, cytochemistry, and immunologic data previously established by the FAB group with those provided by genetic (conventional cytogenetics and molecular genetics) and clinical findings. This new classification of hematological malignancies, which was sponsored by the World Health Organization (<http://xenia.sote.hu/depts/pathophysiology/hematology/e/who-classification.html>), discriminates disorders that have unique natural history and response to therapy.

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Diagnostic Workup

- Complete history of the illness includes a review of the incidence and duration of signs and symptoms, such as pain, fatigue, infection, fever, bleeding, neurological changes, and a review of potential predisposing factors.
- Physical exam should assess for pallor, petechiae, rash, lymphadenopathy, limping, hepatosplenomegaly, and neurological changes.
- CBC with differential should determine whether values of WBCs, RBCs and platelets are abnormal. Peripheral blasts may or may not be present.
- Bone marrow aspiration to determine if marrow is hypercellular: a minimum 20% blast is required for the diagnosis AML.
- Cytochemical stain: AML is positive for the myeloperoxidase, Sudan black B and esterases stains.
- (A -8) [Cytogenetic abnormalities](#) are found in the leukemic cells of 50% to 60% of children with AML. The cytogenetic abnormalities have also prognostic significance and therapeutic implications (risk based therapy).
- Immunophenotyping: AML-associated antibodies include CD11b, CD13, CD14, CD15, CD33, and CD36
- Lumbar puncture determines CNS involvement.

Medical Management

The intensity of the treatment protocols for AML requires that these patients be cared for in cancer centers or hospitals that can provide the supportive care needed to manage complications that arise during and after therapy.

Optimal treatment for AML requires eradication of the leukemia cells in the bone marrow and other sites.; and supportive care plays a very important part in the management of the disease.. The foundation of the therapeutic approach is a systematic administration of combination chemotherapy. Treatment for AML generally consists of 2 phases:

1. Induction phase
2. Postremission consolidation/intensification.

Similar to the ALL treatment, the induction phase is used to attain remission. The most active chemotherapy agents used during induction are cytarabine (AraC) and an anthracycline (daunorubicin). Current regimens can also include other cytotoxic agents such as etoposide (VP -16), and thioguanine. In addition, daunorubicin can be replaced by either mitoxantrone or idarubicin. A review of several pediatric clinical trials suggests that intensive chemotherapy is required to induce remission. Evaluation of response is usually performed two to three weeks after a course of intensive chemotherapy. If the bone marrow continues to have a leukemia infiltrate, another course of chemotherapy should be started immediately.

Post-remission therapy for children with AML involves varying numbers of intensive chemotherapy courses (short-term treatment for approximately 6 months) and/or allogeneic bone marrow transplantation (ABMT/HSCT). Patients who do not have a sibling donor generally receive a intensive post-remission chemotherapy regimen (high-dose cytarabine with asparaginase). Other agents used include etoposide, thioguanine, anthracyclines, and amsacrine.

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For children at very high risk of relapse who have a matched sibling donor, an unrelated allogeneic transplantation is recommended and offers the only possibility of cure.

Maintenance therapy is generally not a part of most AML protocols because most studies demonstrate no improvement in remission duration in patients who received aggressive post-remission therapy.

CNS prophylaxis includes intrathecal methotrexate, cytarabine, or both, often combined with hydrocortisone. Intrathecal chemotherapy--with or without cranial irradiation--is added to treatment protocols of children who have CNS leukemia at diagnosis (clinical neurologic features and/or presence of leukemic cells on CSF; M4 and M5 AML with inv 16 or 11q23 chromosomal abnormalities).

Relapse/ Recurrence

Patients with AML usually experience a relapse within 4 years of diagnosis, with about 50% of relapses occurring in the first year. Relapses generally occur in the bone marrow and less frequently in the CNS. An important prognostic factor in achieving a second remission is the length of first remission. Lower rates of second remission and survival correlate with short first remissions (<1 year).

Salvage therapy for children with AML who relapse usually include high-dose cytarabine in combination with mitoxantrone, fludarabine, idarubicin and asparaginase. Second transplantations are rarely beneficial for children who relapse after prior bone marrow transplantation.

Future Directions:

New treatment approaches include risk stratification and the use of biologically-targeted therapies to improve anti-leukemic treatment while sparing normal tissues. And although CNS-directed therapy has not yet been shown to contribute significantly to survival, studies in this area are ongoing.

The use of hematopoietic growth factors (G-CSF, GM-CSF) to reduce myelosuppressive toxicities has demonstrated significant reduction in neutrophil recovery time with varying degrees of reduction in morbidity and very little effect on mortality.

The use of intensive therapy and increasing rates of survival in children with AML require that patients receive close surveillance. These patients require periodic cardiac, renal, and auditory monitoring. Also, patients who have total body irradiation should be monitored for growth failure, gonadal and thyroid function, development of cataracts, and secondary malignancies.

Acute Promyelocytic Leukemia (APL; M3 – FAB Classification)

(A – 9) [Acute promyelocytic leukemia](#) (APL) is a distinct subtype of AML that is caused by a specific t(15;17) chromosomal translocation. This translocation is associated with disruption of the PML gene and the retinoic acid receptor and results in the production of the *PML-RAR-alpha*

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fusion protein. This protein represses the signaling mechanism that triggers differentiation in APL cells, therefore decreasing terminal cell differentiation and increasing proliferation of promyelocytes.

APL represents approximately 1% of all childhood leukemia and approximately 8-30% of all pediatric AML. It occurs more often in children between 2 to 3 years of age and in adults over 40 years. Incidence is higher in people of Hispanic and Mediterranean descent.

Clinical Characteristics:

APL has non-specific signs and symptoms that include bleeding tendencies resembling disseminated intravascular coagulation (DIC). Bleeding commonly precedes leukemia diagnosis by 2-8 weeks, and is manifested by petechiae, ecchymosis, epistaxis, bleeding in the mouth and venipuncture sites, and hematuria. The bleeding disorder is caused by the release from the leukemia cells of enzymes that activate blood clotting proteins, which causes clots to form in the microvasculature. This clot formation consumes clotting factors and platelets, causing increased bleeding. Thus, children with APL are at high risk for life-threatening hemorrhage (often in the brain and lungs) that has a 15 – 30 % mortality rate. Because of the bleeding complications, APL is considered a medical emergency.

Typically, patients also show pancytopenia (associated with anemia and neutropenia) and thrombocytopenia without any organomegaly. APL is also correlated with low incidence of CNS disease

It is therefore essential that the nurse recognize the presenting symptoms and provide supportive therapy as early as possible. After the physician is notified, the nurse must be ready to provide care needed to prevent further complications.

Medical Management:

The *treatment of APL represents a medical emergency to control the bleeding*, and includes a form of differentiation therapy (activating the retinoid receptor to cause the promyelocytes to differentiate (mature), therefore preventing them from proliferating). The drug used is [\(A – 10\) all-trans-retinoic acid \(ATRA\)](#). Supportive care in the form of close monitoring, usually in the ICU setting, and correcting coagulopathy with platelets transfusion and/or fresh frozen plasma, is of paramount importance in efforts to decrease the relatively high rates of early death from bleeding (5-15 %). Because ATRA is not able to eliminate the leukemic clone, it is often used in combination with chemotherapy (anthracyclines, cytarabine). Clinical investigations have shown that ATRA combined with an anthracycline agent increases the rates of complete remissions with fewer relapses. Relapse is further prevented by using a maintenance therapy with ATRA and low dose chemotherapy.

Patients who become resistant to ATRA and anthracycline-cytarabine chemotherapy might still attain complete remissions with [\(A – 11\) arsenic trioxide \(Trisenox\)](#). Though the action of arsenic trioxide is not completely understood, it appears to induce apoptosis by down-regulating *Bcl-2*, degrading *PML-RAR α* fusion products, and activating the Jun kinases in a *p53*

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independent pathway. At higher concentrations, arsenic trioxide also induces apoptosis of the leukemic blast.

Relapse is managed with bone marrow transplantation (both allogeneic and autologous).

Minimal residual disease is detected using RT-PCR for *RARa/PML*

Prognosis

Ninety percent of patients with newly diagnosed with APL achieve complete remission, and approximately 75% can be cured by the combination of ATRA and chemotherapy. Patients who relapse and are treated with arsenic trioxide have a favorable outcome. Another drug that has produced favorable results in relapsed APL is the monoclonal antibody gemtuzumab ozogamicin (Mylotarg or GO)

Future Directions:

Because of its specific pathogenesis (chromosomal disruption causing alterations in cell proliferation and differentiation), acute promyelocytic leukemia will be the basis for future investigations using targeted therapy to restore differentiation of other types of leukemic blasts.

Current ATRA studies include the use of Lipo-ATRA (liposomal form of all-trans retinoic acid), the role of ATRA in normal hematopoiesis, and the mechanisms of retinoid resistance by the leukemic cells. In addition, studies are currently underway to develop ways to monitor response to therapy, including the role of minimal residual disease and how this information can be best used to improve treatment. Other studies include the role of arsenic trioxide in the treatment of relapse. Arsenic trioxide is favored because of its low toxic effects.

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Helpful Weblinks

NCI Cancer.gov

<http://www.meb.uni-bonn.de/cancer.gov/CDR0000062896.html>

e-Medicine.com

Mark E. Weinblatt, MD - Acute Myelocytic Leukemia

This website contains a manuscript on AML that describes pathophysiology, clinical features, and treatment modalities of AML.

<http://www.emedicine.com/ped/topic1301.htm>

Merck & Co., Inc., Whitehouse Station, NJ

Acute Myelocytic Leukemia – The Merck Manual

This website contains a brief description of AML including symptomatology, treatment, and prognosis. Good resource both for the nurse and patient.

<http://www.merck.com/mmhe/sec14/ch176/ch176c.html>

Related www.Cure4kids.org Seminars:

Seminar #326 [Atypical Presenting Features of Acute Myeloid Leukemia](#)

Brandon Triplett, MD, Fredric Hoffer, MD, Surender Rajasekaran, MD and Deborah Jones, MD

<http://www.cure4kids.org/seminar/326>

Seminar #432 [Relapsed AML](#)

Bassem Razzouk, MD

<http://www.cure4kids.org/seminar/432>

Seminar #185 [AML](#)

Jeffrey Rubnitz, MD, PhD. and Cesar Nuñez, MD

<http://www.cure4kids.org/seminar/185>

Seminar #45 [Arsenic Trioxide in Childhood Leukemia](#)

Vikramjit S. Kanwar, MD MRCP(UK) FAAP and Sheila Shurtleff, PhD

<http://www.cure4kids.org/seminar/45>

Seminar #266 [Arsenic Trioxide in Childhood Leukemia – in Portuguese](#)

<http://www.cure4kids.org/seminar/266>

Seminar #88 [Early Complications of AML](#)

Monika Metzger, MD, Nobuko Hijiya, MD and Jeffrey Schmidt, MD

<http://www.cure4kids.org/seminar/88>

Seminar #116 [Childhood Myeloid Leukemia](#)

Raul C. Ribeiro, MD

<http://www.cure4kids.org/seminar/116>

Seminar #58 [Minimal Residual Disease in Acute Myeloid Leukemia](#)

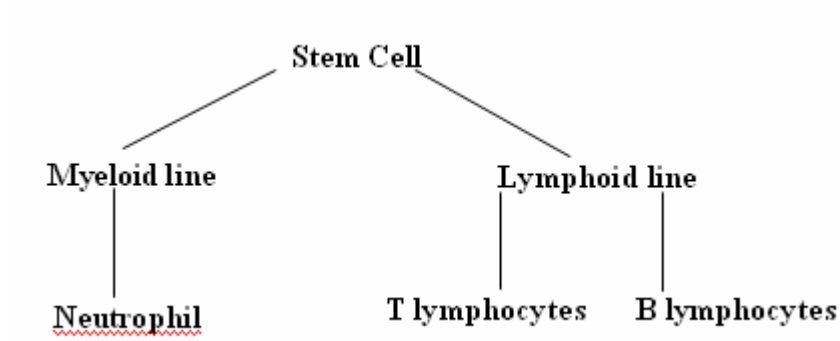
Raul Ribeiro, MD, Dario Campana, MD PhD, Jeffrey Rubnitz, MD, PhD. and Michele Pritchard, RN, PNP

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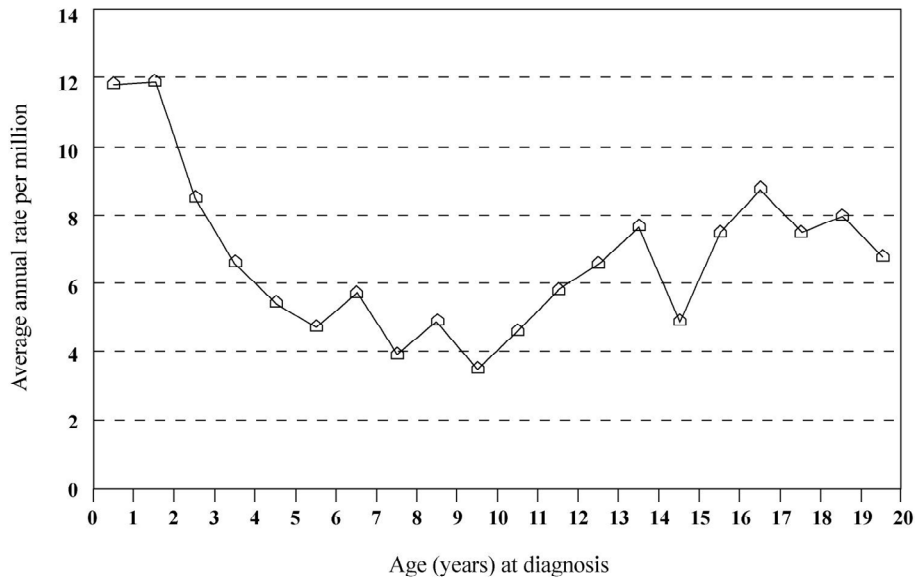
Appendix:

A – 1 Cell Lineage



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A – 2 Incidence of AML according to age at diagnosis



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A – 2 Associated Syndromes

Down Syndrome (Trisomy 21): 14 times more at risk for leukemia; neonates may show a transient proliferation of blasts that regress spontaneously within 1 – 2 months.

Fanconi Anemia: An inherited disease that affects primarily the bone marrow, causing decreased production of all types of blood cells. Eighty percent of Fanconi's anemia patients demonstrate skin pigment changes, such as darkened areas, vitiligo, and café-au-lait spots. They have short stature with skeletal anomalies such as upper limb abnormalities (missing or extra digits, underdeveloped or absent bones), scoliosis, hip, leg and toe abnormalities, facial anomalies such as eye/eyelid and ear abnormalities, deafness, and anatomical anomalies such as kidney, GI and cardiopulmonary malformations. Infants may show failure-to-thrive syndromes and retardation

Bloom Syndrome: An autosomal recessive genetic disease caused by a mutation of the BLM gene. Patients with Bloom's syndrome have small body size, photosensitivity, and infertility.

Kostmann Syndrome: An autosomal-recessive disorder of neutrophil production that causes severe congenital neutropenia (SCN), and is often associated with life-threatening bacterial infections.

Diamond Blackfan Anemia (DBA): A congenital bone marrow failure syndrome characterized by a failure of the bone marrow to produce red blood cells. "Anemia" means low red cell counts. Severe anemia is frequently found at birth and the majority of individuals are diagnosed before their first birthday.

Paroxysmal Nocturnal Hematuria (PNH): A rare, acquired (non-genetically transmitted), chronic disorder that affects the production of stem cells (precursors of all blood cells, including red blood cells, white blood cells, and platelets). The disorder leads to anemia, thromboses and increased susceptibility to infections.

Li-Fraumeni Syndrome: A rare inherited disorder (autosomal dominant--one copy of the altered gene is sufficient to cause the disorder) caused by a mutation in the *p53* or *CHEK2* (tumor-suppressor genes. Though the mutation greatly increases a person's risk of developing several types of cancer, particularly as children or young adults, fewer than 400 families worldwide have been diagnosed with the condition.

Neurofibromatosis (Von Recklinghausen's disease): An autosomal dominant disorder characterized by increased skin pigmentation, peripheral nerve tumors and a variety of other dysplastic abnormalities of the skin, nervous system, endocrine organs, and blood vessels. The responsible gene is located on the long arm of chromosome 17.

Myelodysplastic Syndromes – see Document 3

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A – 3 Acquired/Treatment-related

Benzene: household cleaning products

Alkylating Agents: chlorambucil, cyclophosphamide, melphalan

Nitrosoureas: nitrogen mustard (Mustargen)

Epipodophyllotoxins: etoposide, teniposide

Ionizing Radiation: x-rays, sun exposure

Topoisomerase Inhibitors: anthracyclines, camptothecins



A – 4 Presenting Symptoms/Common Findings in Childhood AML/ANLL

Finding	% of Patient Presenting
Hepatosplenomegaly	50
Fever	30 - 34
Bleeding	33
Pallor	25
Anorexia/weight loss	22
Weakness/fatigue	19
Sore throat	18
Bone and joint pain	18
Lymphadenopathy	13 – 20
Gastro-intestinal symptoms	13
Swollen gingiva/gingival hypertrophy	9 – 15
Chest pain	5
Leukemia cutis	4 - 9
Recurrent infections	3
Chloroma	2 - 16

Golub, TR et.al (1997) Principles and Practice of Pediatric Oncology, Pizzo & Poplack (Eds)

Gingival hypertrophy



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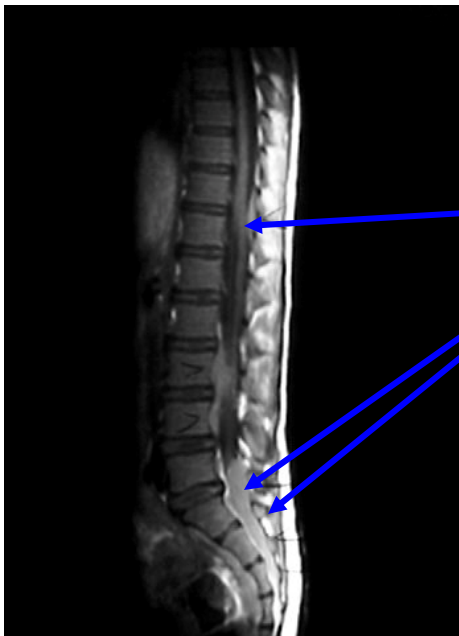
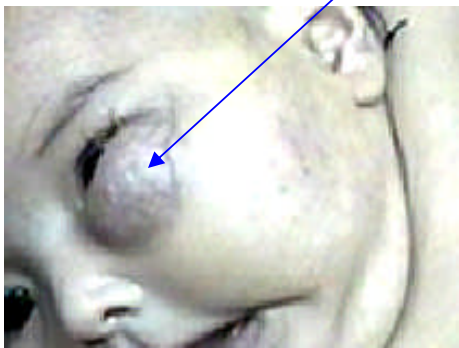
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A – 5 Chloroma



Peri-orbital chloroma

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Epidural chloroma causing extradural cord compression

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A – 6 Leukemia cutis/Blueberry muffin



Courtesy of Carlos Rodriguez-Galindo, MD SJCRH

Subcutaneous nodules in an infant with AML



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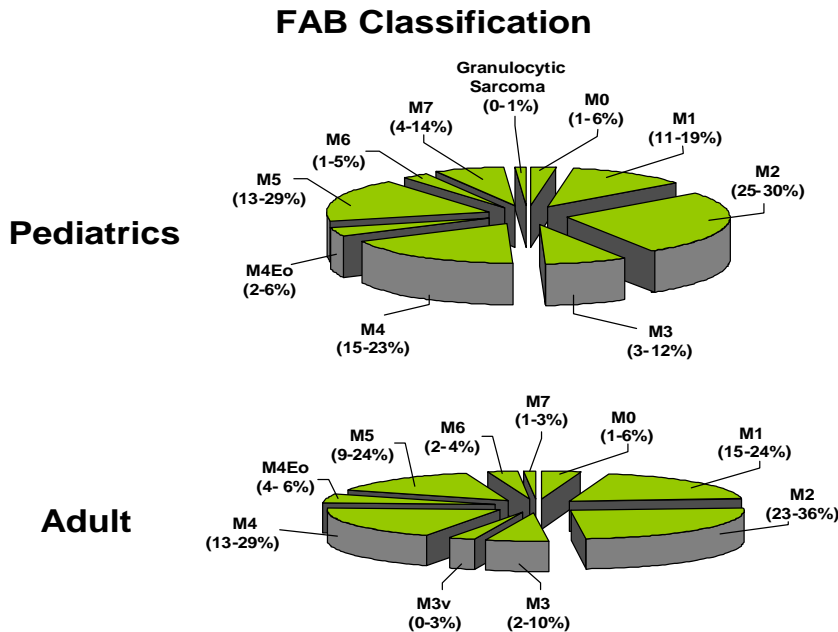
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A – 7 FAB Classification System of Acute Myelogenous Leukemia

FAB Type	Common Name	Criteria for Diagnosis	Histochemistry
M1	Acute Myeloblastic Leukemia without maturation	Blast >90% non-erythroid cells; 10% of cells are maturing granulocytes or monocytes	MP +
M2	Acute Myeloblastic Leukemia with maturation	Blasts from 30 – 89% non-erythroid cells, >10% maturing granulocytic cells, <20% monocytic cells	MP+
M3	Acute Promyelocytic Leukemia (hypergranular variant)	<20% abnormal hypergranular promyelocytes, Auer rods common	MP+
M3V	Acute Promyelocytic Leukemia (microgranular variant)	Fine granular cytoplasm in promyelocytes, reniform nuclei, dark primary granules	MP+
M4	Acute Myelomonocytic Leukemia	Blasts > 30% non-erythroid cells, > 20 but < 80% of cells are of monocytic lineage; blood monocyte count >5x10 ⁹ /L or elevated serum lysozyme or NSE +	MP+ NSE+
M4Eo	Acute Myelomonocytic Leukemia with eosinophilia	Abnormal eosinophils with specific eosinophilic granules and large basophilic granules	MP+ NSE+ Eos-PAS+
M5	Acute Monocytic Leukemia	>80% non-erythroid cells are monoblasts, promonocytes or monocytes M5a - >80% of monocytic cells are monoblasts M5b - <80% of monocytic cells are monoblasts	NSE+
M6	Acute Erythroleukemia	>30% non-erythroid cells are blasts >50% of marrow cells are erythroblasts	Erythroblasts PAS+
M7	Acute Megakaryocytic Leukemia	>30% non-erythroid cells are megakaryocytes; cytoplasmic blebs, myelofibrosis	Platelet Perox + (EM)

Golub et.al Acute Myelogenous Leukemia in Pizzo & Poplack. Principles and Practice of Pediatric Oncology

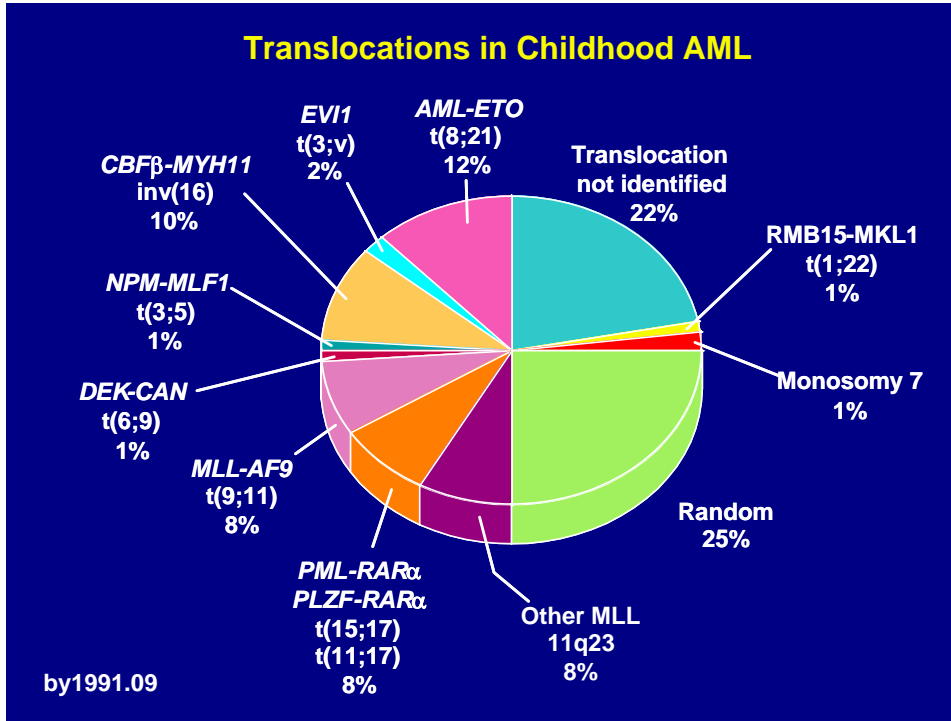


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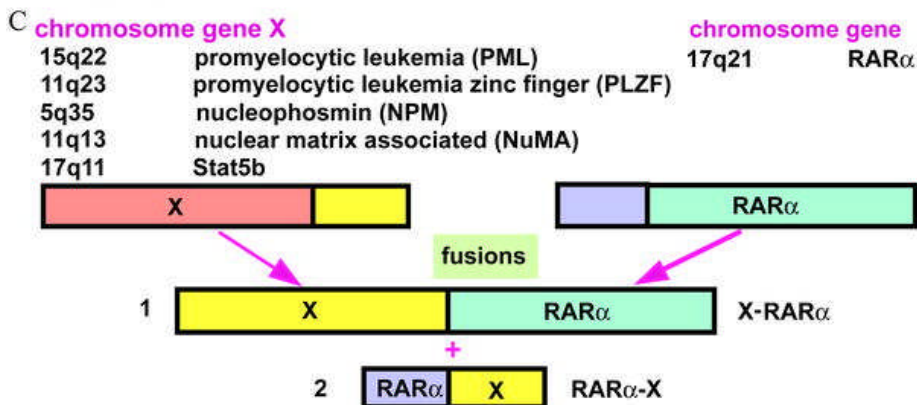
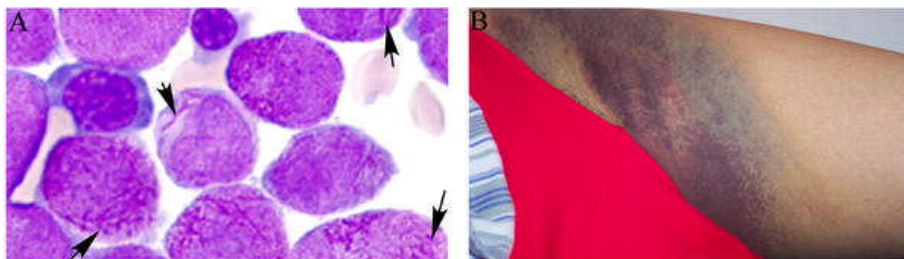
A – 8 Cytogenetics of AML –due mostly to translocations



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A – 9 Acute Promyelocytic Leukemia



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The Three Features of APL

The three features of APL are (A) accumulation of abnormal promyelocytes; (B) fibrinogenopenia and disseminated intravascular coagulation; and (C) the chromosomal translocation t(15;17)(q22;q21) and the resultant fusion transcripts, and variants.

Guang-Biao Zhou et. al Retinoic Acid and Arsenic for Treating Acute Promyelocytic Leukemia; Public Library of Science, Jan. 2005

PLoS Medicine, San Francisco, CA

<http://medicine.plosjournals.org/perlserv/?request=get-document&doi=10.1371/journal.pmed.0020012>



A – 10 All-trans-retinoic acid (ATRA)

All-trans retinoic acid (ATRA) is an effective differentiating agent for the leukemia cells of patients with APL. ATRA has the ability to restore the signaling mechanism that was repressed by PML-RARalpha protein fusion, thus avoiding premature cell breakdown and the ensuing bleeding problems. Most patients with APL achieve a complete remission induction therapy with a combination of ATRA and chemotherapy (cytarabine and daunorubicin).. The advent of ATRA therapy revolutionized the treatment of APL and markedly improved the prognosis.

ATRA syndrome is a serious side effect of ATRA treatment and includes fever, respiratory distress, and hypotension. The ATRA syndrome can be prevented by the addition of chemotherapy (hydroxyurea) and/or dexamethasone if the WBC is increasing



A – 11 Arsenic Trioxide (Trisenox; Cell Therapeutics Inc./Cephalon, Inc).

<http://www.fda.gov/CDER/foi/label/2000/21248lbl.pdf>

Common toxicity symptoms include gastrointestinal side effects (nausea, vomiting, diarrhea, and abdominal pain), fatigue, edema, hyperglycemia, dyspnea, cough, rash or itching, headaches, and dizziness. These adverse effects have not been observed to be permanent or irreversible, nor do they usually require interruption of therapy.

Another important adverse event is QT prolongation – a change in the time it takes for the heart to relax after each beat. One serious reported case of QT prolongation evolved into an abnormally rapid heartbeat. This episode resolved spontaneously and the patient was re-treated with Trisenox without recurrence of the event.





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