2026 California Thoracic Society Annual Educational Conference

& Chronic Obstructive Pulmonary Disease Symposium

Thursday March 12, 2026-Sunday March 15, 2026

Earn up to 19 CME/CEU/MOC Credits

Jointly Provided by AKH Inc., Advancing Knowledge in Healthcare and the California Thoracic Society

PORTOLA HOTEL & SPA
AT MONTEREY BAY

Thursday March 12, 2026 (6 CME/CEU/MOC Credits)
COPD Symposium
Friday March 13, 2026 (6.5 CME/CEU/MOC Credits):
Advances in Interventional Pulmonary, Remote Monitoring in Pulmonary and Sleep Medicine, Approach to Symptom Management in Chronic Lung Disease and Critical Care
Saturday March 14, 2026 (6.5 CME/CEU/MOC Credits)
Sepsis and Shock, Extracorporeal Membrane Oxygenation, Inpatient Pulmonary Complications of Cancer Care
Sunday March 15, 2026
Fellow and Resident Track Symposium

California Thoracic Society
ATS Chapter Serving California and Arizona

AKH INC.
ADVANCING KNOWLEDGE IN HEALTHCARE

California Thoracic Society
Annual Conference
March 12-15, 2026
Portola Hotel and Spa, Monterey, CA

Saturday March 14, 2026

Advances in Management of the Patient with Sepsis

8:00 am – 8:10 am: Welcome and Introduction

8:10 am – 8:55 am: Keynote Address – Phenotyping and Personalized Medicine in Sepsis

- **Angela Rogers, MD (Stanford)** - This speaker will discuss phenotyping in the patient with sepsis and septic shock and how close we are to precision medicine in managing sepsis.

8:55 am – 9:20 am: Incorporating Artificial Intelligence Decision Making in Identifying Sepsis

- **Gabriel Wardi, MD (UC San Diego)** - This speaker will describe how artificial intelligence can be used to identify the septic patient before they present with end stage symptoms to impact care earlier in the course of illness.

9:20 pm – 9:35 pm: Pro: The Severe Sepsis and Septic Shock Early Management Bundle (SEP-1) Bundle Saves Lives

- **Sean Townsend, MD (CPMC-Sutter)**- This speaker will argue the benefits of the SEP-1 Bundle/how it saves lives.

9:35 pm – 9:50 pm: Con: : The Severe Sepsis and Septic Shock Early Management Bundle (SEP-1) Bundle Does Not Save Lives

- **Natalie Achamallah, MD, MS (Cottage Health)** - This speaker will argue the against the SEP-1 Bundle/highlight its limitations.

9:50-10:00 am Question and Answer

10:00 am – 10:30 am: Break

Extracorporeal Membrane Oxygenation

10:30 am – 10:55 am: When to refer to an ECMO center and when to deploy ECMO

- **Nida Qadir, MD (UC Los Angeles)** - This speaker will discuss the evidence behind the use of ECMO in patients with respiratory failure and when providers should consider referral to an ECMO center and when centers should use ECMO.

10:55 am – 11:20 am: What about ECMO to go?

- **Mazen Odish, MD (UC San Diego)** - This speaker will discuss the advent of mobile ECMO services, how they can help improve patient care, and the use of extracorporeal cardiopulmonary resuscitation.

11:20 am – 11:45 pm: Ventilator Strategies for the patient on ECMO

- **Abirami Kumaresan, MD (Cedars-Sinai)** - This speaker will discuss the how ventilator strategies may differ in the patient on ECMO and how different ECMO configurations impact which ventilator strategy to use.

11:45 pm – 12:10 pm: What you need to know about pediatric ECMO

- **Kathleen Ryan, MD (Stanford)** - This speaker will discuss the utility of ECMO in neonates and children, and the complexities of management in children who needs mechanical support.

12:10 pm – 12:20 pm: Question and Answer

12:20 pm – 1:20 pm: Lunch

Hands-On Session:

1:20 pm – 2:20 pm: Non-Invasive Cardiac Output Monitors **Speaker Abirami Kumaresan, MD (Cedars-Sinai)** ECMO Machines **Mazen Odish, MD (UC San Diego)** ECMO Placement **David Gordon, DNP (UC San Francisco) & Brianna Zuckerman, NP (UC San Francisco)** Ventilator Settings and Portable ventilators **Joe Van Vleet, RT (UC Los Angeles) & Theresa Cantu, RT (Valley Children's)**

2:20 pm – 2:45 pm: Break

Inpatient and Pulmonary Complications of Cancer Care

2:45 pm – 3:10 pm: Pulmonary Complications of Hematopoietic Stem Cell Transplantation

- **Husham Sharifi, MD (Stanford)** - This speaker will discuss the pulmonary complications that arise after HCT, in particular the development of bronchiolitis obliterans syndrome and approaches to management.

3:10 pm – 3:35 pm: Pulmonary Vascular Complications of Malignancy

- **Naomi Habib, MD (Norton Thoracic Institute)**- This speaker will discuss the Pulmonary Vascular Disease complications of malignancy including PA sarcoma, pulmonary tumor thrombotic microangiopathy, and medications that can cause PAH.

3:35 pm – 4:00 pm: Drug induced Interstitial Lung Disease and Pneumonitis During Cancer Therapy

- **Weijia Chua, MD (Stanford)** - This speaker will discuss the pulmonary complications of interstitial lung disease and pneumonitis that develop after chemotherapy and targeted immunotherapy

4:00 pm – 4:25 pm: Respiratory Complications of Acute Leukemia

- **Hugh Davis, MD (City of Hope)** - The speaker will discuss various oncologic emergencies, how they are recognized, and how they are managed in the acute setting.

4:25 pm – 4:35 pm: Question and Answer

5:30 pm – 7:30 pm: Trainee Poster Competition (NON-CME) – Food and beverages will be served

Dr. Hugh Davis received his medical degree from Tufts University School of Medicine in Boston, MA and completed his post-graduate training in both Internal Medicine and Pulmonary and Critical Care Medicine at Cedars Sinai Medical Center in Los Angeles. Dr. Davis is the medical director of intensive care at City of Hope National Medical Center on both the Duarte as well as the Irvine campuses. Dr. Davis serves as Associate Clinical Professor in the Department of Medicine.

Respiratory Complications of Acute Leukemia

Hugh Davis, M.D.

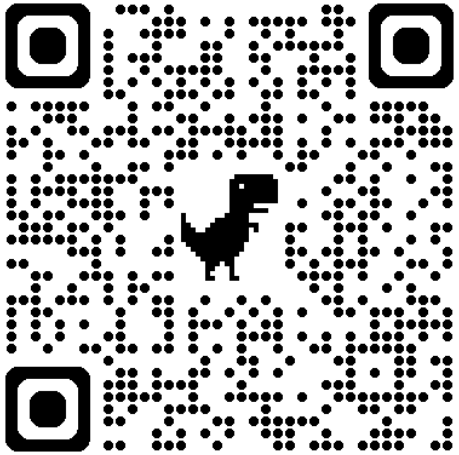
City of Hope National Medical Center

Disclosures

- I have the following relationships with ACCME defined ineligible companies:
  - None
- I **WILL NOT** discuss off-label use and/or investigational use of any drugs or devices.

Case Example

- 52 year-old male
  - Presented to community hospital 98 miles away 4 hours ago
  - Chief complaint of fatigue and DOE
- HR = 102, RR = 26, O2 sat = 96%, BP = 116/84
- Local hematologist is requesting transfer for higher level of care
  - Aute leukemia suspected
- Air transport initiated
- **Code Acute Leukemia** activated

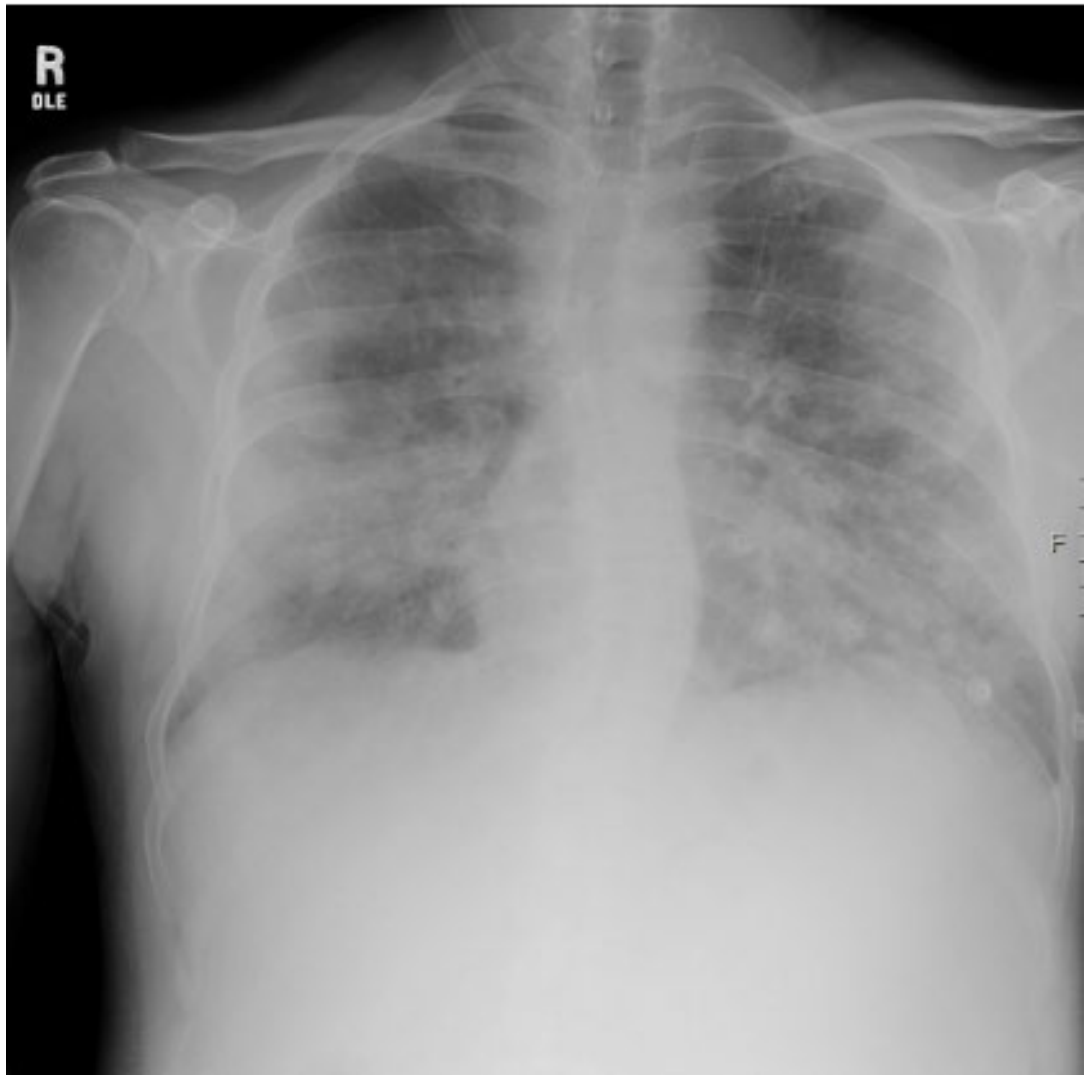


WBC Count	305.62 !!
RBC Count	2.63 ▼
Hemoglobin	6.9 !!
Hematocrit	23.4 ▼
Platelet Count	45 ▼
MCV	89.0
MCH	26.2
MCHC	29.5 ▼
RDW	18.7 ▲
MPV	10.7

Blast Percent	05/14/25	75.6 !!

Sodium Level, Blood	141
Potassium Level, Blood	5.8 ▲
Chloride Level, Blood	97 ▼
Carbon Dioxide Level, Blood	11 !!
Glucose Level (Random), Blood	30 !!
Blood Urea Nitrogen Level, Bl...	51 ▲
Creatinine Level, Blood	2.00 ▲
eGFR, Blood	36 ▼
Anion Gap, Blood	33 ▲
Magnesium Level, Blood	2.2
Phosphorus Level, Blood	9.7 !!

PH, ARTERIAL	7.10 !!
PCO2, ARTERIAL	41.5
PO2, ARTERIAL	83.0
HCO3-, ARTERIAL	12.7 ▼
FIO2 (ARTERIAL)	60.0
BASE EXCESS	-15.8
O2 Saturation, Arterial	93.0
Patient Temperature	36.5



Respiratory Complication of Acute Leukemia

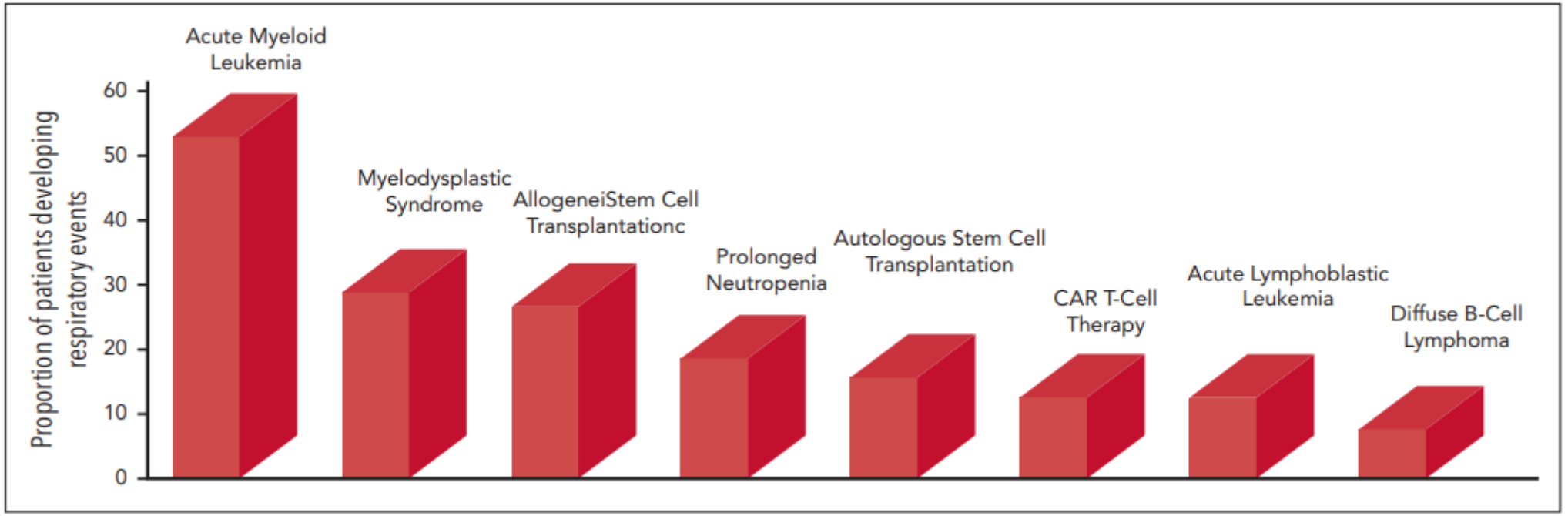
- **Disease-specific complications of acute leukemia**
  - **Leukostasis**
  - **Leukemic pulmonary infiltration (LPI)**
  - **Acute lysis pneumopathy (ALP)**
- Infectious complications
- Therapy related complications
- Transfusion related complications
- Hemorrhagic complications
- Etc…

ICU admission and respiratory failure

	Incidence of respiratory events	Need for ICU admission	Hospital respiratory mortality
Haematological malignancies			
Acute myeloid leukaemia[5,18–23]	22–84%	66%	45%
Acute lymphoblastic leukaemia[18,22,23]	7–18·5%	12–15%	38·5%
Lymphoproliferative diseases[5]	8%	8%	40-50%
Myelodysplastic syndrome[18]	29·4%	20%	17%
Autologous haemopoietic stem cell therapy[24,25]	3–28%	42%	3–55%
Allogeneic haemopoietic stem cell therapy[26,27]	24–30%	50%	51%
Prolonged neutropenia[6,28]	8–29·5%	11–16%	5–12%

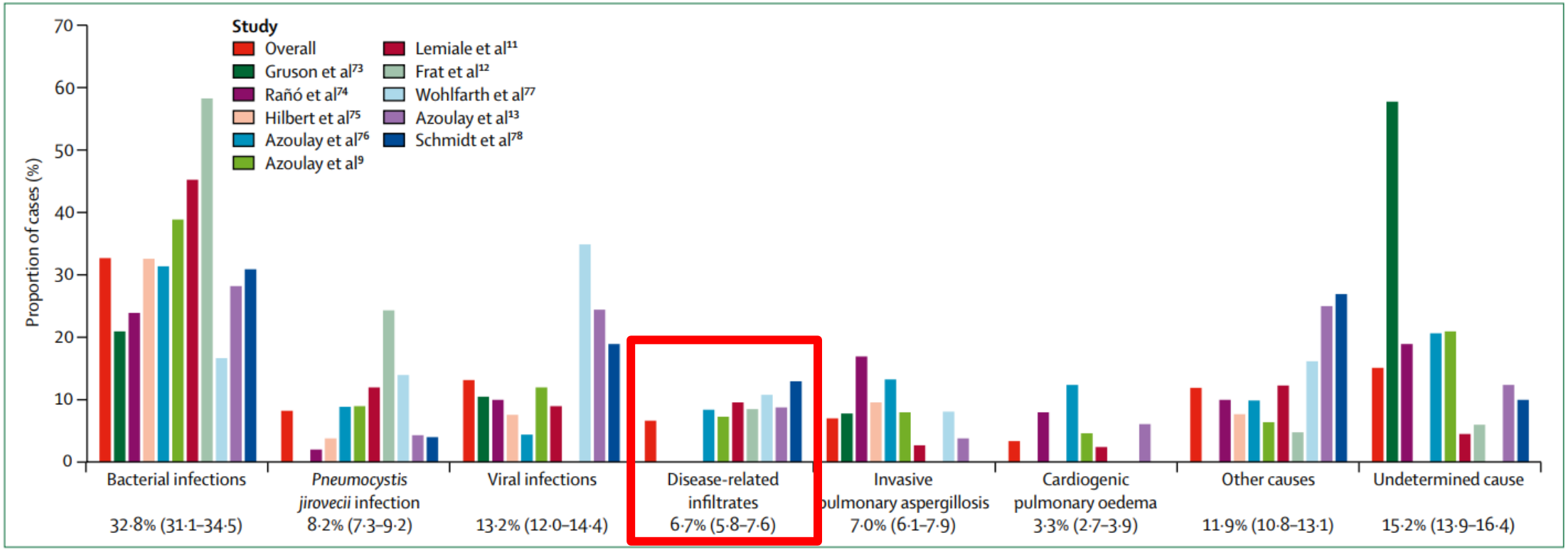
Azoulay, et al. Lancet Repir. Med 2019; 7: 173-86

Respiratory failure with hematologic malignancies



Azoulay, et al. Blood. 2024

Pulmonary infiltrates with immunocompromised patients



Azoulay, et al. Lancet. 2019

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New AML: ICU Utilization

- 25% ICU utilization rate at some point. [1, 2, 3]
- ICU admission most common during induction phase [4, 5]
- Acute respiratory failure (ARF) most common reason for ICU [4, 5]
  - Approximately 50% of cases
- Mechanical ventilation frequently needed[6]
  - Approximately 50% of cases of ARF with new AML

1. Fer
2. De
3. Ha
4. Ra
5. Schllongowski, et al. Harmatologica, 2011
6. Tavares, et al. Leukemia and Lymphoma. 2018

Respiratory Failure in AML

- Highest risk in the earliest phases[1]
  - High risk of respiratory failure in first 10 days after diagnosis
    - Up to 80% of cases
  - Leukemia-specific etiology in 61%
  - 28-day mortality 34%

Moreau, et al. Leukemia & Lymphoma. 2014

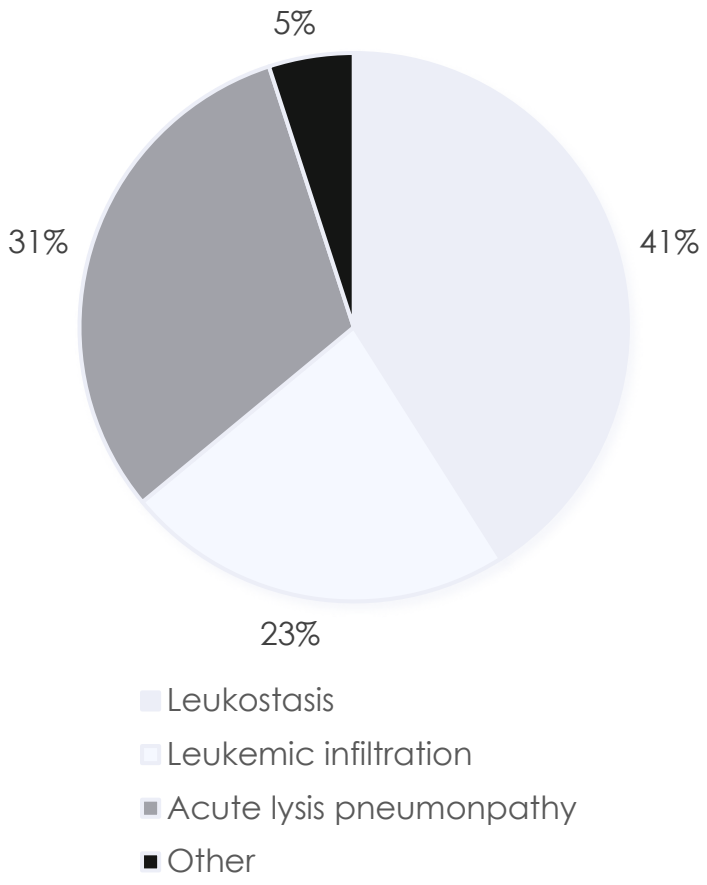
ARDS with newly diagnosed AML

- ARDS among newly diagnosed AML:
  - 9% within first 15 days
  - 14% within first 30 days
- Development of ARDS associated with:
  - Higher WBC count
  - Higher blast %
  - Higher transfusion requirements
- 30-day mortality of 64%

Van de Louw, et al. Leukemia & Lymphoma, 2018

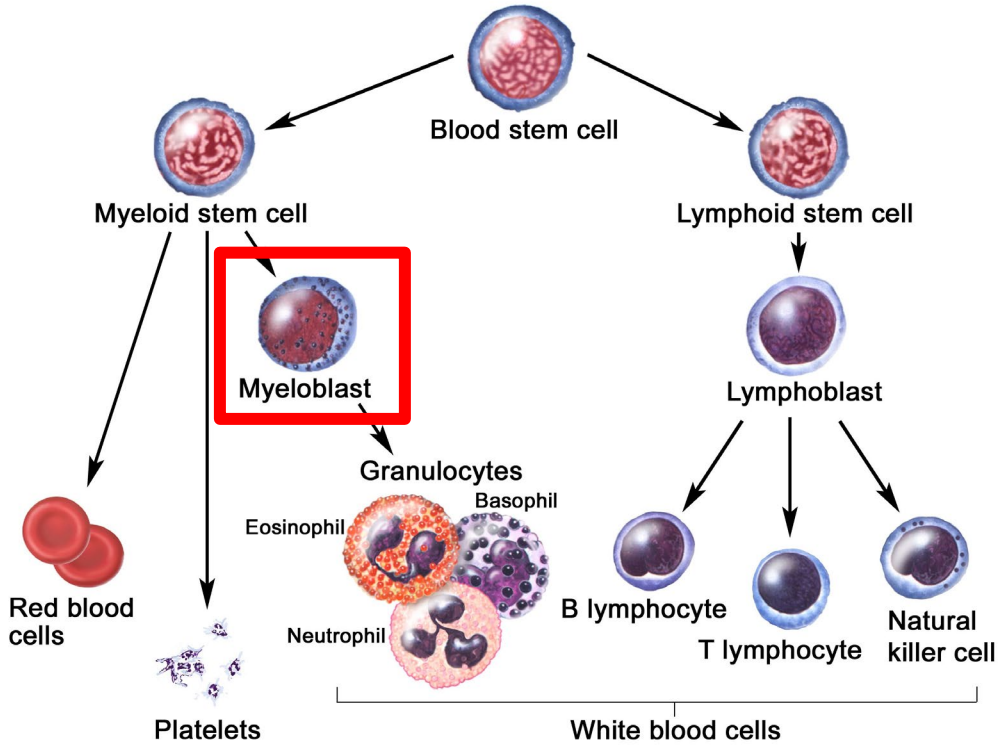
Leukemia specific pulmonary complications

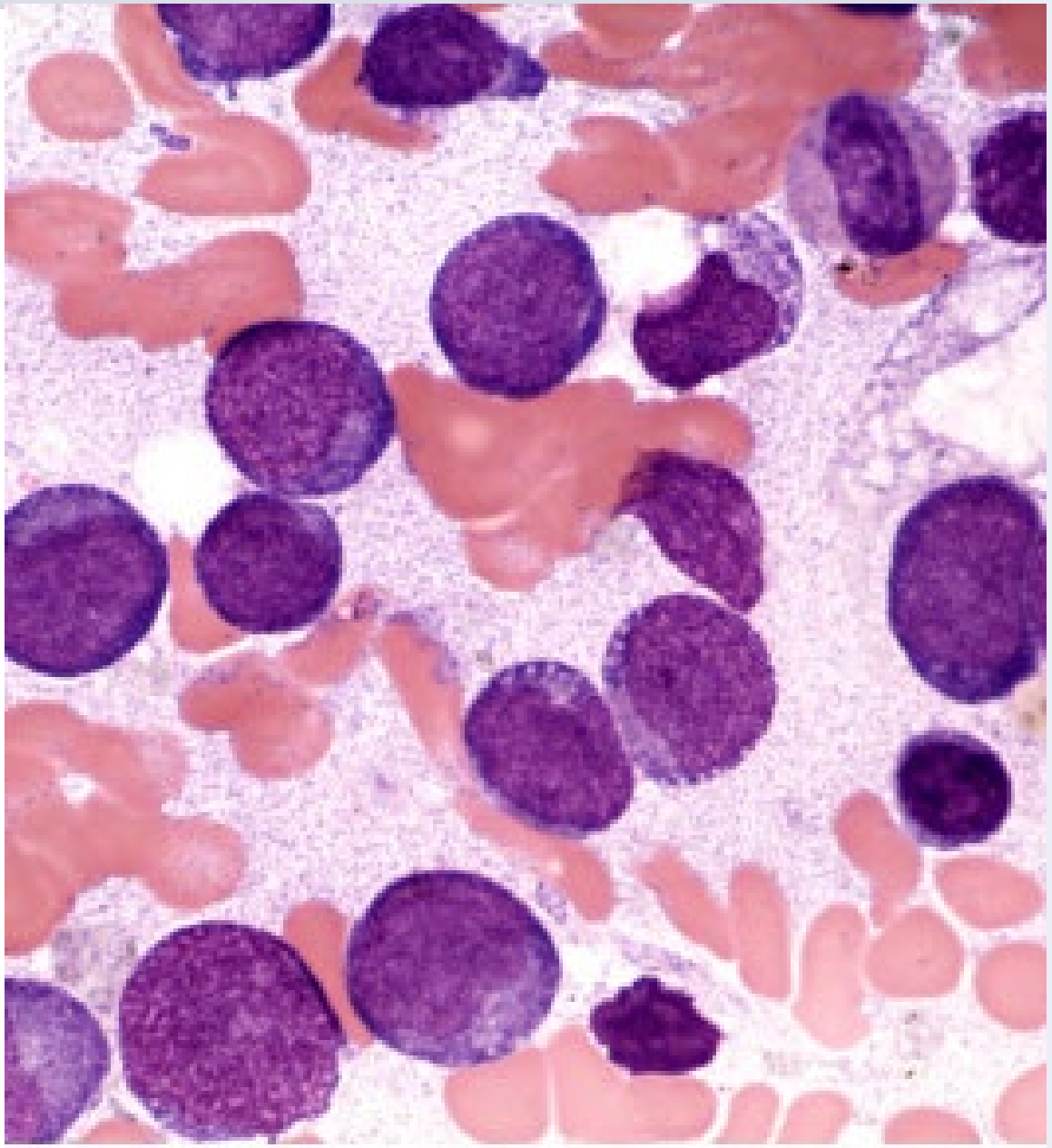
- Leukostasis
- Leukemic pulmonary infiltration (LPI)
- Acute lysis pneumopathy (ALP)



Moreau, et al. Leukemia & Lymphoma. 2014

Blast cells

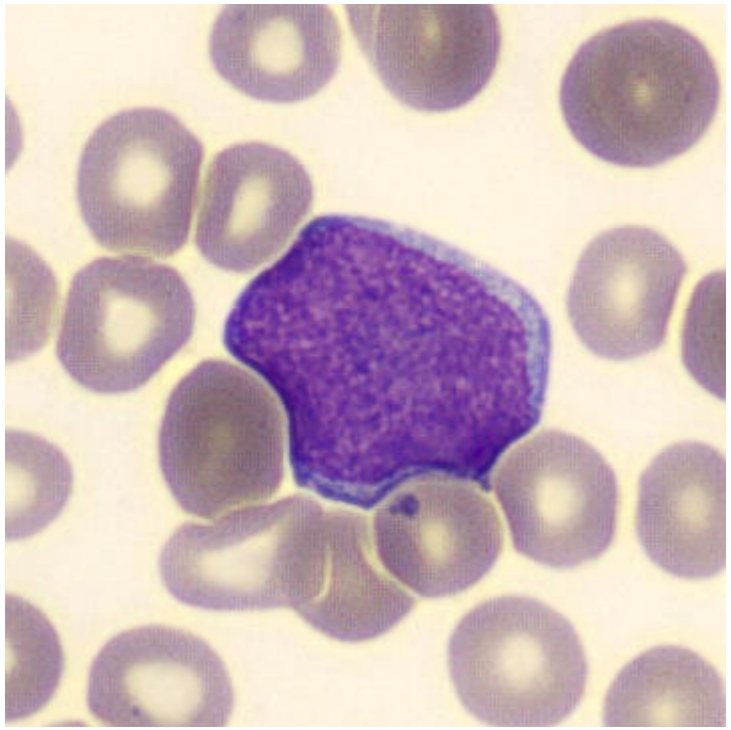




From Atlas of Hematopathology (2nd Edition) 2018

Blast cells

- Immature myeloid precursor
- Enhanced adhesion to vascular endothelium[1]
  - Blast cells secrete cytokines TNFa, IL1β
  - Leading to expression of:
    - Intracellular adhesion molecule – 1
    - Vascular cell adhesion molecule – 1
    - E-selectin
- Adhesion → Migration →Infiltration
  - Release of metalloproteinases[2]
    - Cellular injury



1. Stucki, et al. Blood. 2001
2. Stafanidakis, et all. Blood. 2009

Leukostasis and Hyperleukocytosis (HL)

- Hyperleukocytosis (HL)[1]
  - WBC > 100 x10^9/L or 100,000/µL
  - Present in up to 10% of de novo AML
  - Associated with TLS and DIC
  - Associated with:
    - AML subtype M4 and M5
    - MLL rearrangement 11q23
    - FLT3-ITD mutation
- Leukostasis seen in approx. 30% of HL[2]
  - Clinical manifestations of vascular occlusion

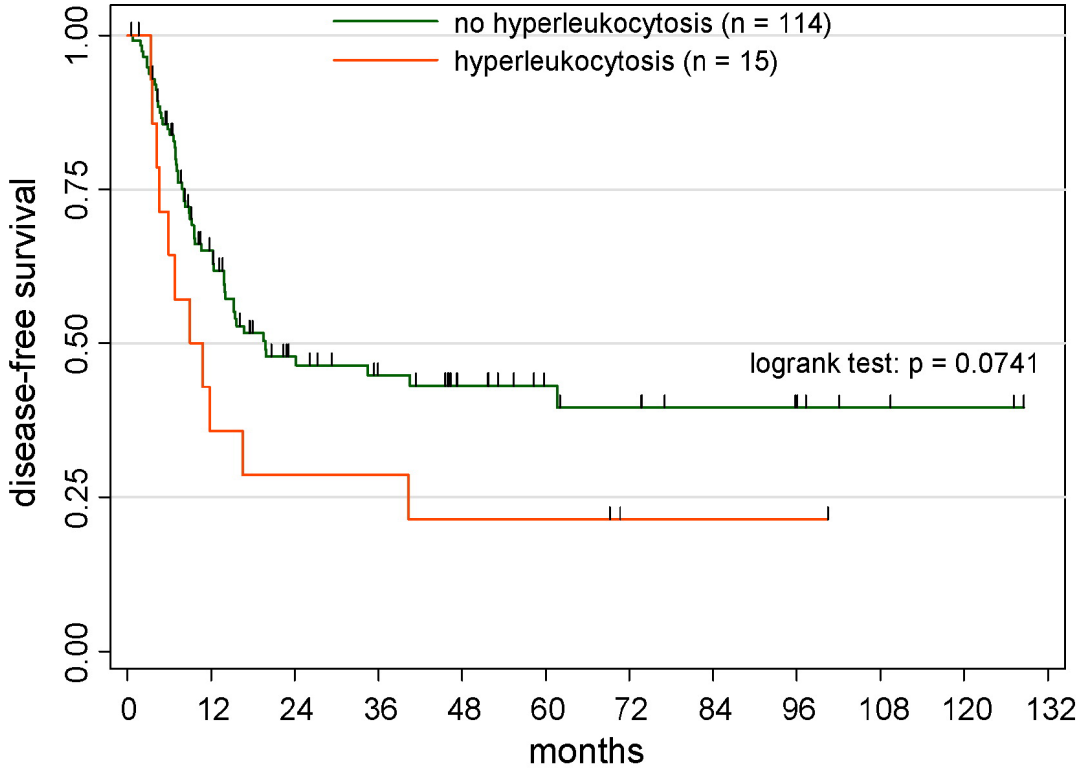
FAB Class	Subtype	Proportion of AML
M0	Undifferentiated	5%
M1	Myeloblastic without maturation	10-15%
M2	Myeloblastic with maturation	20-30%
M3	Promyelocytic	10%
M4	Myelomonocytic	25 %
M5	Monocytic	10%
M6	Erythroleukemic	<5%
M7	Megakaryoblastic	<5%

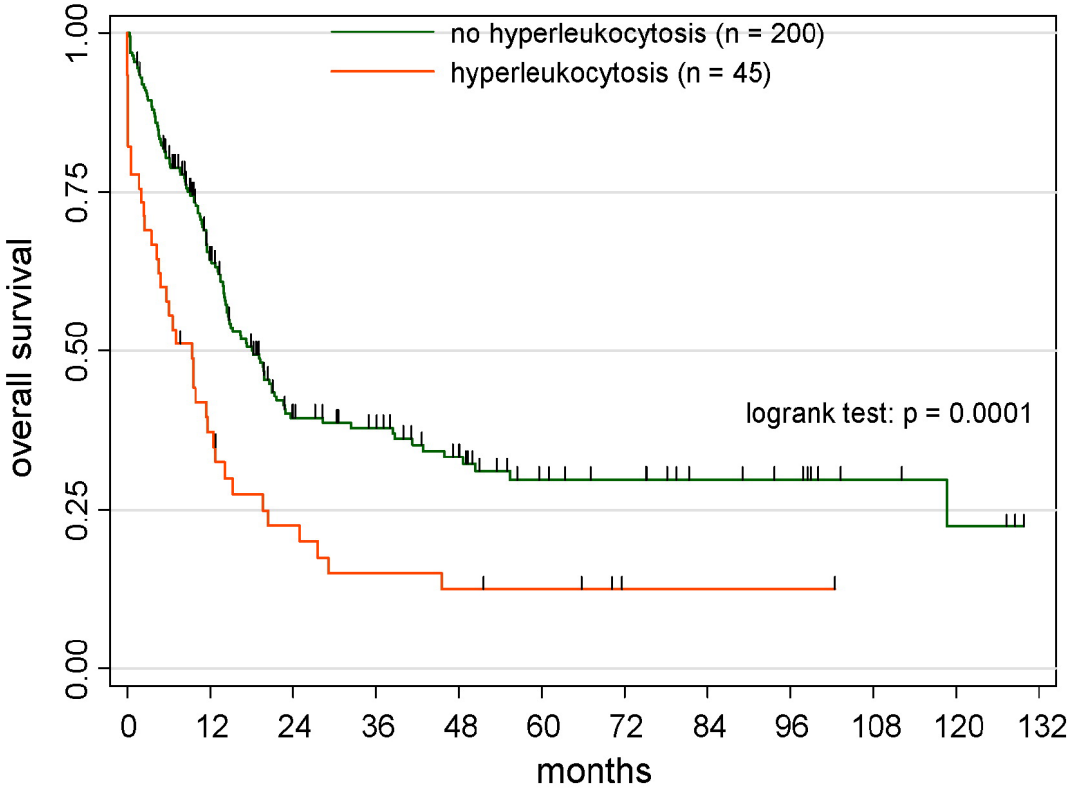
1. Rollig,

2. Stahl,

3. Table adapted from Schiffer, et al. Cancer Medicine, 6th ed. 2003

Hyperleukocytosis: survival impact with new AML





Marbello, et al. Leukemia Research. 2008

Leukostasis

- Changes in blood rheology: hyperviscosity
  - Mechanical obstruction of pulmonary vasculature
- 40% of new AML patients show pulmonary vascular occlusion[1]
- Non-specific radiographic findings present in approx. 60% [2]
  - Basilar airspace disease
  - Diffuse interstitial changes
  - Unilateral findings less common
- Concomitant hypoxemia and gas exchange abnormalities

1. Porcu, et al. Br. J Haematology. 1997
2. Stafanski, et al. Medicine. 2016

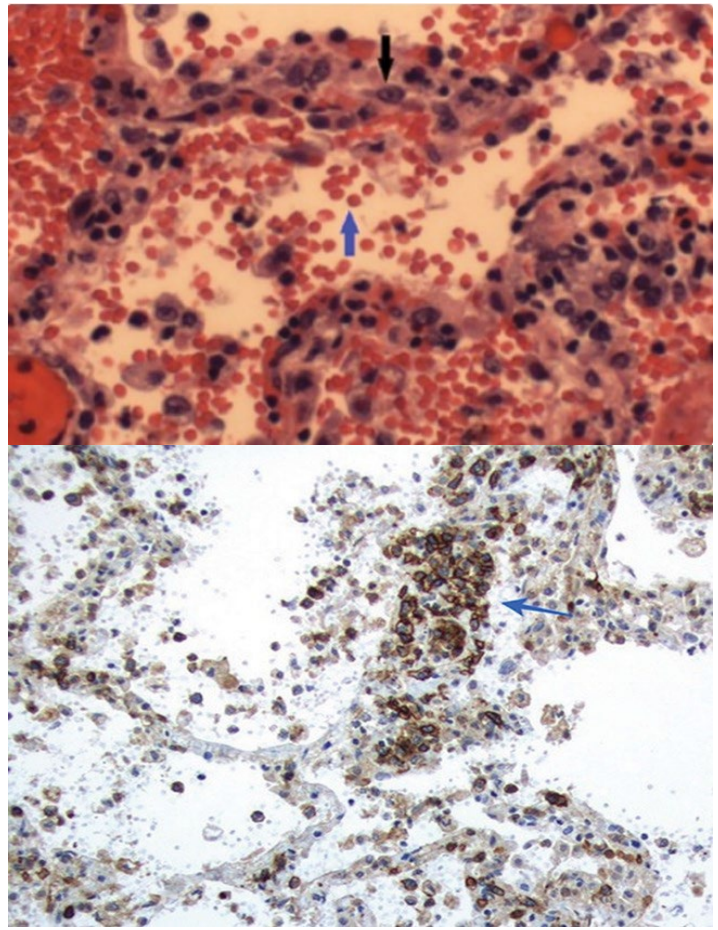
Evaluation of hypoxemia

- "Pseudohypoxemia"[1]
  - Myeloblasts → abnormal cellular O2 consumption
    - "Normal" PaO2 may not reflect actual tissue delivery
  - "Leukocyte Larceny" [2]
    - PaO2 may drop in ABG sample during processing[3]
    - Myeloblastic consumption
    - Rate of decline is proportional to WBC count
    - Consumption can be slowed by cooling of the sample[2]
  - Pulse oximetry may be more reliable at the bedside

1. Hess
2. Fox, et al. Am. J. of Med. 1979
3. Chillar, et al. Blood 1980

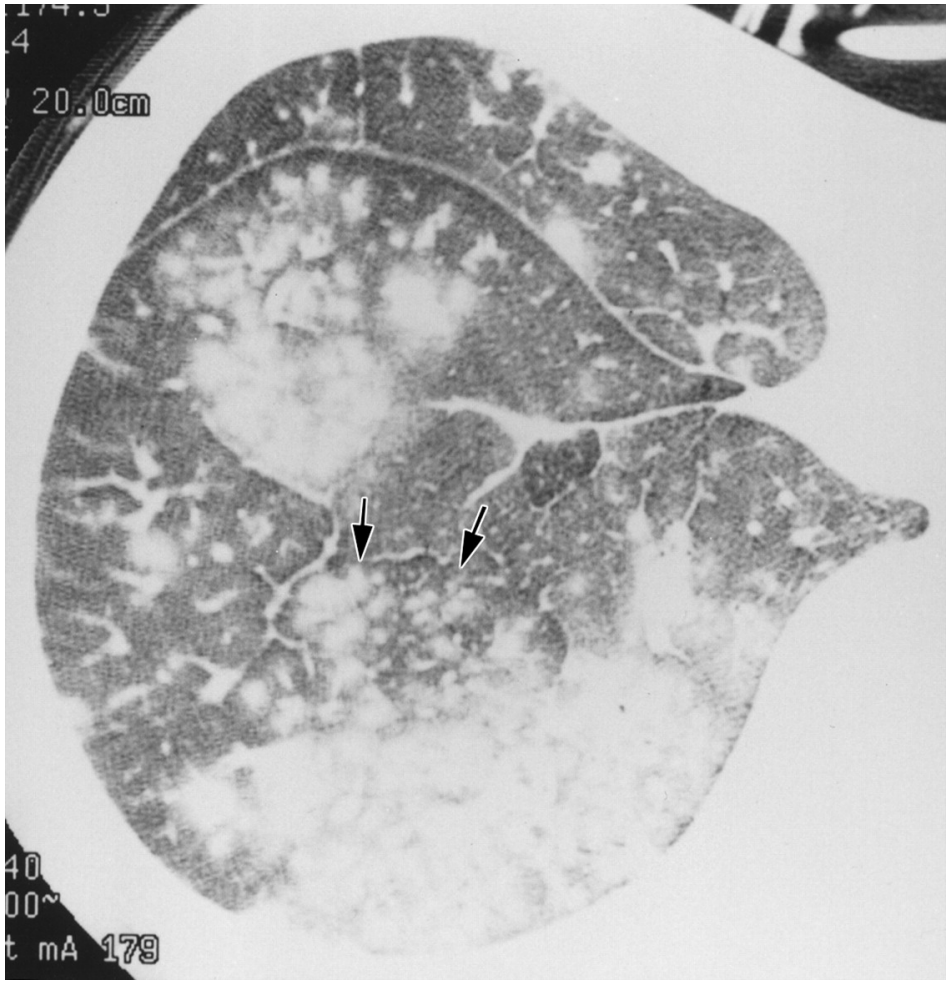
Pulmonary leukemic infiltration (PLI)

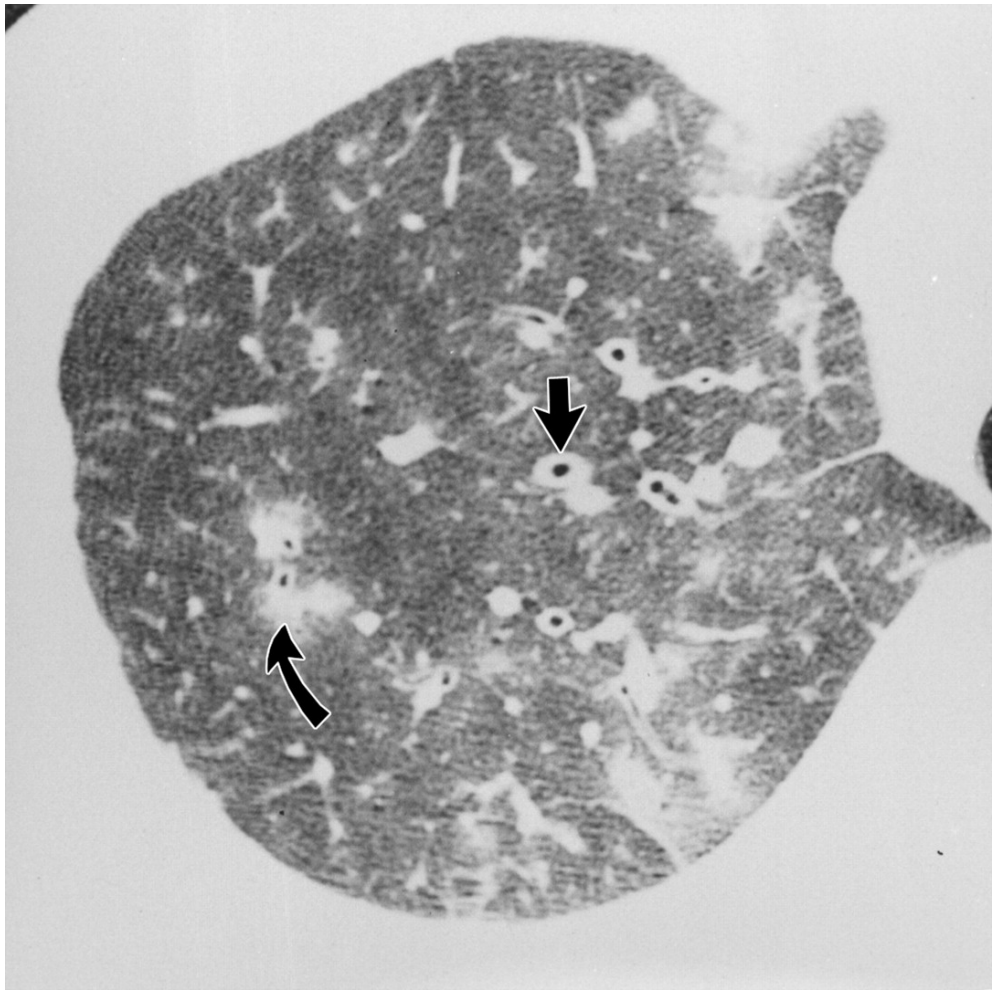
- Extravascular migration into lung parenchyma
- Not necessarily associated with hyperleukocytosis
- Incidence established historically post-mortem:
  - 25-30% of new AML [1, 2]
- Interstitial infiltrative changes on CT [3]
  - Perilymphatic
  - Peribronchovascular
  - Septal/interlobular



1. Na
2. Gr
3. Heyneman, et al. Am J of Roentgenol. 2000
4. Image from: Fayed, et al. Oxf. Med Case Reports. 2019

Pulmonary leukemic infiltration (PLI)

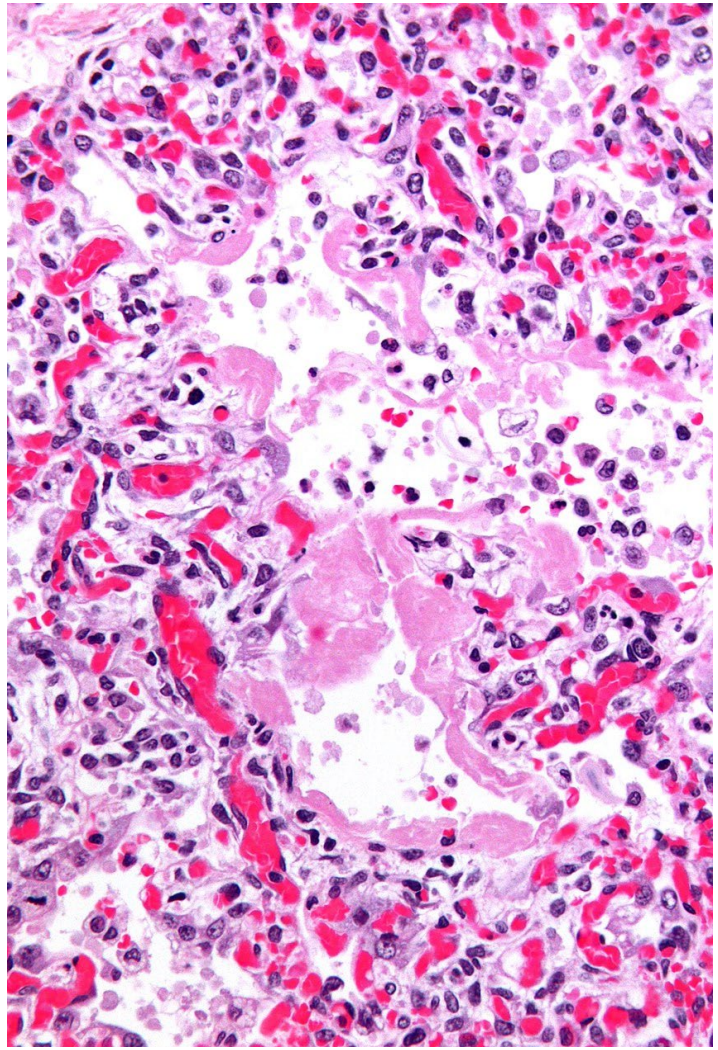




Heyneman, et al. AJR. 2012.

Acute Lysis Pneumopathy (ALP)

- Temporal association is key!
  - Onset following induction chemotherapy
- Release of proteolytic enzymes
  - Vascular endothelial damage
- More common in the setting of HL
  - Up to 75% of ALP occurs in the setting of HL [1]
- Difficult to distinguish from edema
- Diffuse alveolar damage on histopathology
  - Proliferative phase
  - Degenerating blasts seen in the interstitium [2]



1. Ma
2. Tryka, et al. Cancer 1982

	Leukostasis	LPI	ALP
Temporal onset	Before induction chemotherapy	Before induction chemotherapy	Immediately following induction chemotherapy
Hyperleukocytosis	Yes	Inconsistent	Higher risk when present
Radiologic abnormalities	Non-specific	1. Peribronchovascular 2. Interlobular septal thickening 3. Ground glass	Non-specific
Anatomic and pathologic findings	Occlusion of vascular lumen by blast aggregates	Blast cells found: 1. Around arteries and bronchi 2. Within interlobular spaces 3. Within alveolar septa	Diffuse alveolar damage (DAD) with degenerating blast cells in the interstitial space
Extrapulmonary manifestations	Neurologic symptoms	Infiltrative syndrome	Acute tumor lysis syndrome
Specific management	Cytoreductive therapy +/- Leukapheresis Dexamethasone 10 mg q6-8 hrs	Cytoreductive therapy Dexamethasone 10 mg q6-8 hrs	Dexamethasone 10 mg q6-8 hrs

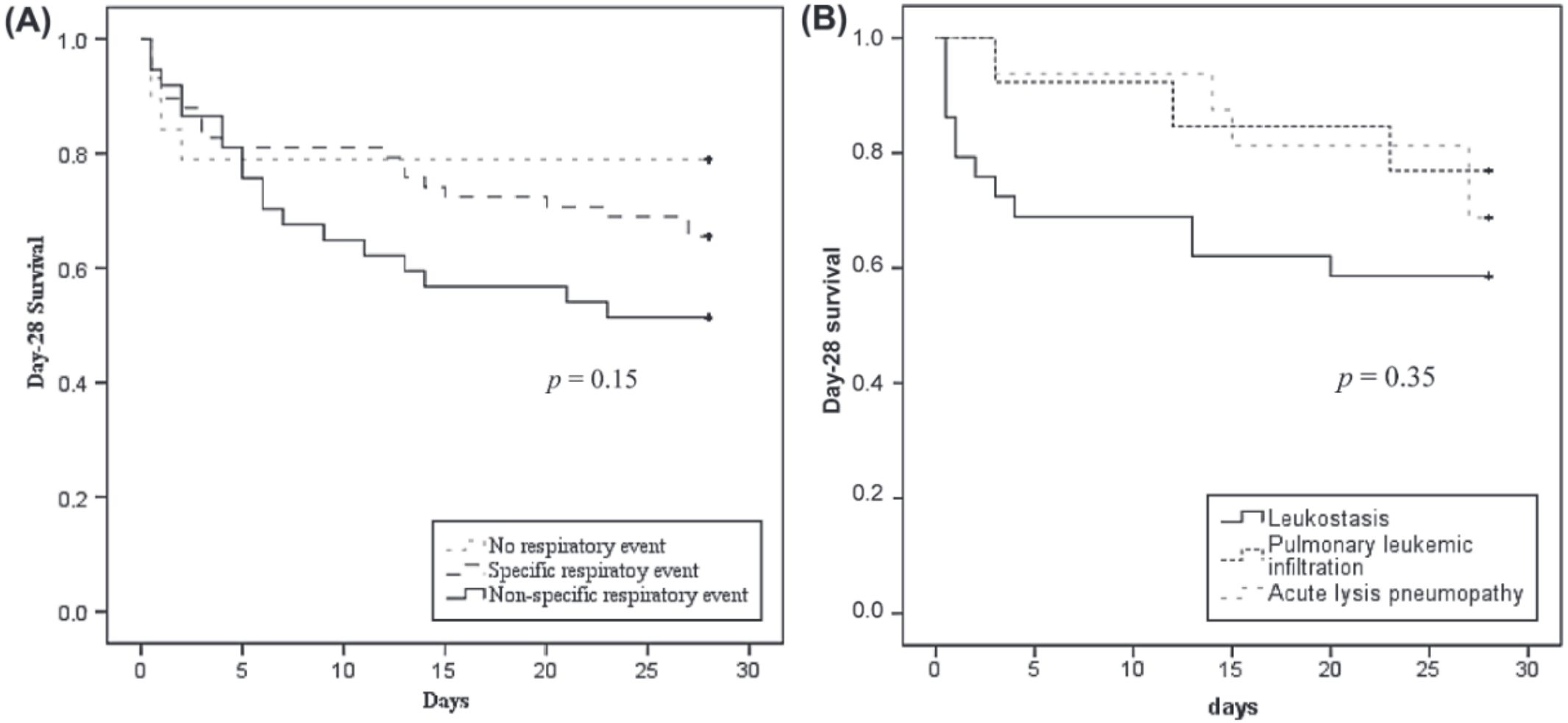
Adapted from: Moreau, et al. Leukemia and Lymphoma. 2014, Chean, et al. Expert Rev. Resp. Med. 2024

Bronchoscopy and BAL

- Controversial
  - Diagnostic yield is generally low (50%) [1, 2]
  - Clinically significant results are unlikely (15-20%) [1, 2]
  - Exposure to broad-spectrum antimicrobials negatively impacts results [2]
- Limited clinical utility
  - HSV & Influenza account for majority of results with changes in mgmt.[2,3]
    - ?Benefit of invasive testing vs non-invasive
  - Only 11-13% BAL results → new antimicrobial coverage [3, 4]

1. Ro
2. G
3. Ko
4. Buckley, et al. Leuk Lymphoma. 2019

Outcomes



Moreau, et al. Leukemia and Lymphoma. 2014

Outcomes

- Better outcomes at specialized high-volume centers[1, 2, 3]
- Post-hoc analysis of EFRAIM: ARF with AML phenotypic clusters[4]
  - 1. **Leukemic:** higher WBC, lower resp. SOFA, earlier ICU adm
  - 2. **Pulmonary:** more resp sx, diffuse pulm involvement, later ICU adm
  - 3. **Inflammatory:** more organ failure, more shock, higher non-resp SOFA
- Best prognosis among new AML with ARF and higher WBC[4]
  - Highlights reversibility of ARF with new leukemia

1. Halper
2. Kaplan
3. Totesen, et al. Clin. Epidemiology. 2019
4. Secreto, et al. Annals of Intensive Care. 2023

Post-hoc analysis of EFRAIM

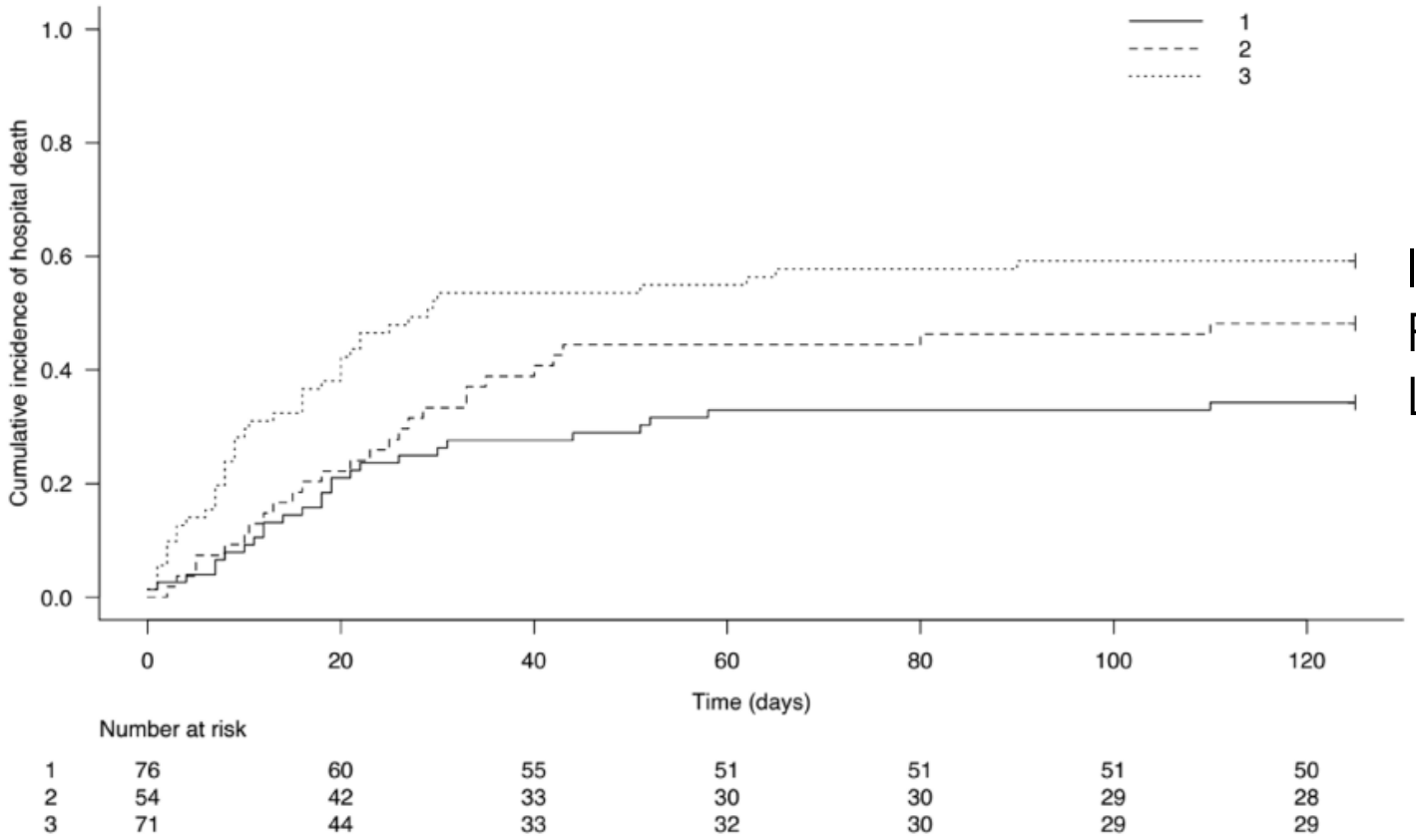


Fig. 3 Cumulative incidence of hospital mortality according to the 3 clusters

Inflammatory
Pulmonary
Leukemic

1. Totes

2. Secreto, et al. Annals of Intensive Care. 2023

Leukapheresis

- Effective in decreasing circulating WBC [1,2]
- Meta-analyses fail to show benefit in early mortality [3,4,5]
- Practice patterns:[6, 7]
  - Aprox. 80% support leukapheresis + induction for leukostasis symptoms
  - Not beneficial when performed without intensive chemotherapy
- Not without risk
  - Vascular access, anaphylactic reactions, electrolyte abnormalities, etc.
- American Society for Apheresis: 2B [8]

1. Bruse 2013
2. Bug,
3. Obe
4. Bew
5. Rina
6. Stah
7. Shall
8. Connelly-Smith, et al. J of Clinical Apheresis. 2023

Thank you!

hudavis@coh.org