

# NEWLY DIAGNOSED ACUTE MYELOID LEUKEMIA MIMICKING AN ST-ELEVATION MYOCARDIAL INFARCTION

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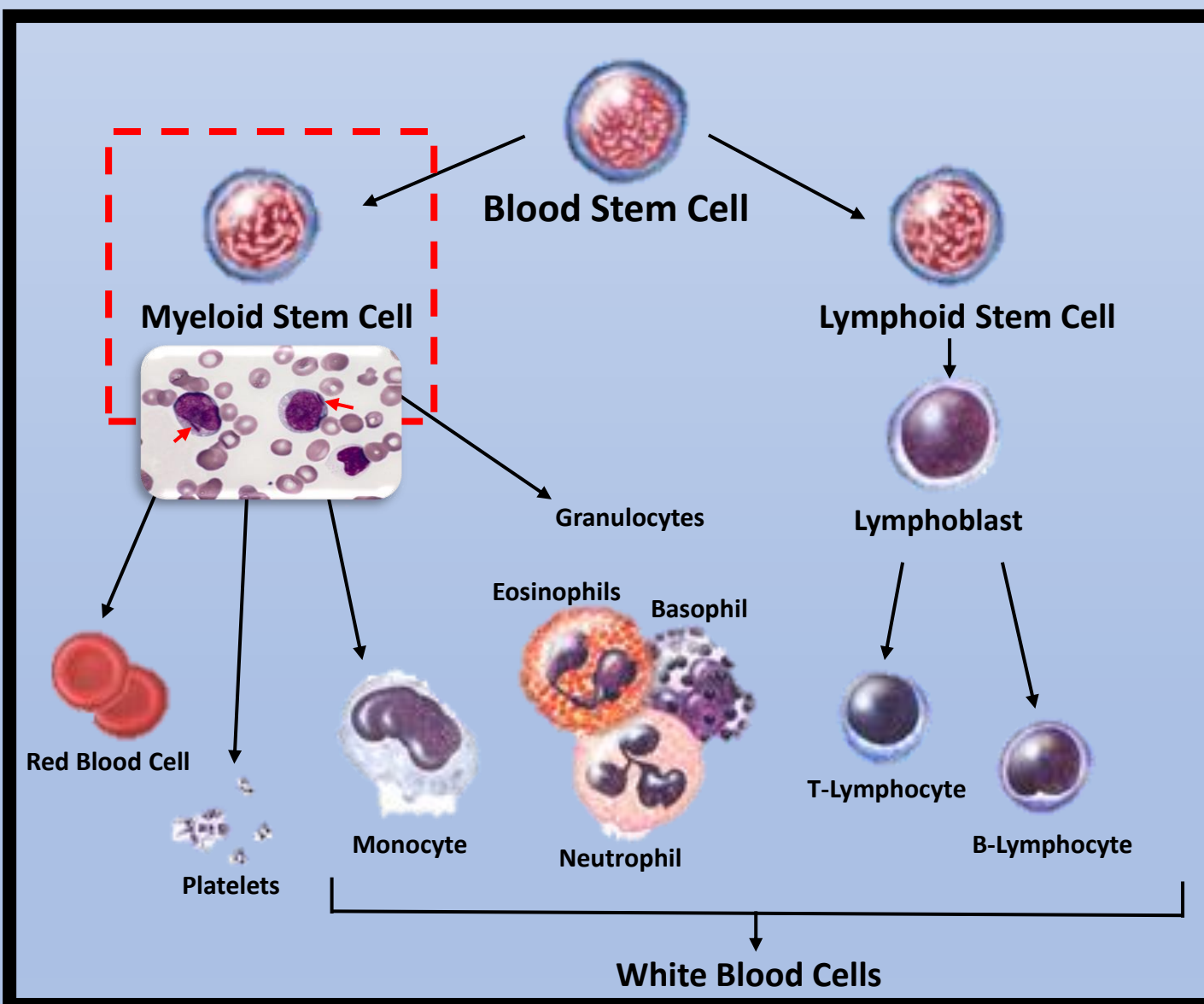
## Introduction

Acute myeloid leukemia (AML) is a rare malignancy of the stem cell precursors of the myeloid lineage that result from genetic variations that lead to neoplastic changes and clonal proliferations. AML is an uncommon malignancy, accounting for only 1.2% of all new cancer diagnoses in the United States, but it accounts for close to one third of all leukemia diagnosed.<sup>1,4</sup>

AML is characterized by clonal proliferation of myeloid precursors that have a decreased ability to differentiate into more mature cellular elements. As a result, leukemic blasts or immature forms accumulate in bone marrow, peripheral blood, and sometimes in other tissues; this is often paired with variable reduction in the production of normal red blood cells, platelets, and mature granulocytes (see figure 1).

Patients with a new AML diagnosis may present with non-specific clinical manifestations (see Table 1), but some are asymptomatic and present with only laboratory abnormalities. Although uncommon, patients with newly recognized AML may present with symptoms that mimic ST-elevation myocardial infarction (STEMI).

## Pathophysiology of AML



**Figure 1:** In normal hematopoiesis, a myeloblast (red dotted box) is an immature precursor to of myeloid white blood cells; a normal myeloblast will mature into a white blood cells such as granulocytes – eosinophils, basophils, and neutrophils – or monocytes. In AML, a single myeloblast accumulates genetic changes that inhibit maturation, increase its proliferation, and protect it from apoptosis. Top left corner, shows peripheral smears from a patient with AML exhibiting two myoblasts with rod-like structure (Auer rods) in the cytoplasm (red arrows).

## Clinical Manifestations of AML

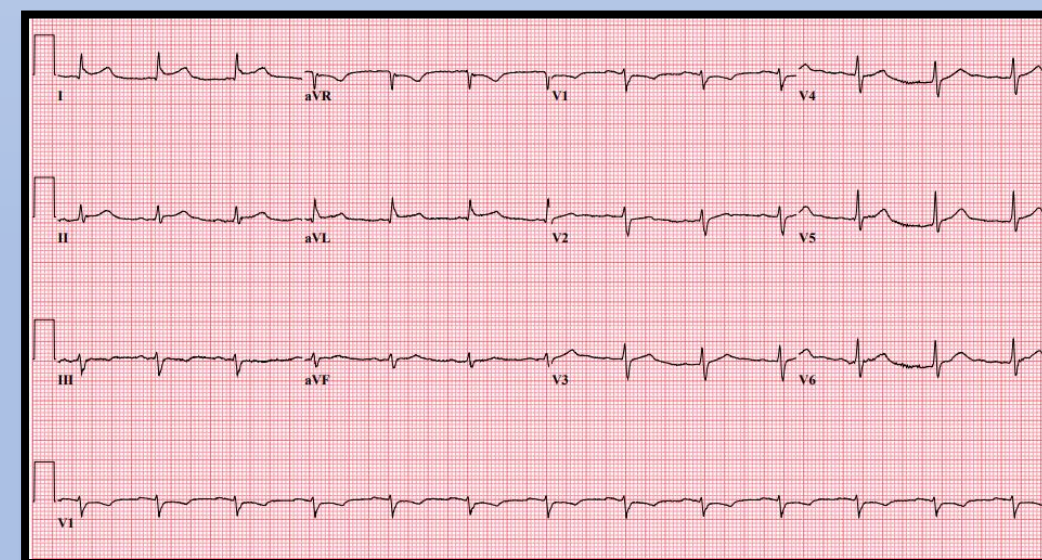
Physical Exam Findings	Cause
Fever, pallor, ill appearing	Risk for infection and sepsis
Conjunctival hemorrhage, retinal hemorrhage, rashes	Thrombocytopenia
Cranial nerve palsies, headache	CNS involvement
Gingival hypertrophy	Leukemia infiltration
Flow murmur, shortness of breath, dyspnea	Anemia

**Table 1:** Patients with a new AML diagnosis may present for care across the healthcare system, with patients seeking care for varied manifestations of AML. Clinical manifestations of AML includes but not limited to the physical exam findings above.

## Case Presentation

Here, we report a case of an 84-year-old female with past medical history significant for atrial fibrillation and inflammatory arthritis who presented to the emergency room due to “crushing” chest pain that radiated to jaw and left shoulder for 2 days. She was found to have leukocytosis at 18.1 K/uL, thrombocytopenia with platelets of 89 K/uL, and anemic with hemoglobin of 9.8 g/dL. EKG demonstrated ST segment elevations in leads 1 and aVL (see figure 2) with a high sensitivity troponin at 2,181 ng/L.

She was immediately taken to the cardiac catheterization lab for diagnostic coronary angiogram. However, cardiac catheterization revealed no significant disease explaining her symptoms. The overall picture was more consistent with myopericarditis, and she started on colchicine. Blood cultures as well as pleural fluid anaerobic and fungal cultures were all negative for infectious organisms that may be the source of the myopericarditis.



**Figure 2:** EKG demonstrated ST-segment elevations in leads 1 and aVL.

In the setting of the patient’s anemia and thrombocytopenia, a peripheral blood smear (PBS) was evaluated and suggestive of possible hematologic neoplastic process. Given worsening thrombocytopenia, colchicine was discontinued. Further evaluation with bone marrow aspirate and biopsy as well as flow cytometry of the PBS demonstrated a new diagnosis of AML with 26.5% CD34 positive myeloblasts in circulation. As the patient continued to decline, she opted for inpatient hospice and was pronounced deceased shortly thereafter.

## Discussion

Early manifestations of AML typically presents with non-specific symptoms relating to anemia and thrombocytopenia (see table 1). Additionally, skin manifestations, including erythema nodosum, stasis dermatitis, and vitiligo nodules, and/or erythematous or as a violaceous papules or plaques, is an atypical presentation of AML seen in 10% of AML cases.<sup>5</sup> However, we did not see any skin manifestations in our patient. The involvement of the heart in AML is not uncommon. Robert et al studies a large case series involving 420 autopsies of leukemic patients and found that 69% of patients suffered from some degree of involvement of the heart.<sup>7</sup> However, an initial manifestation of AML as pericarditis is very infrequent. The exact incidence is unknown, but it is estimated to occur in 1-2% of the AML cases.<sup>2,5</sup>

Interestingly, in addition to anemia symptoms, our patient presented with retrosternal chest pain accompanied by elevated HS troponin at 2,181 ng/L and ST-segment elevations on EKG in leads 1 and aVL. This prompted a cardiac catheterization which showed no evidence of coronary artery disease. Workup to determine the etiology of myocarditis, including serologies for bacterial and viral illnesses as well as cardiac catheterizations was unremarkable. A confirmatory endomyocardial biopsy would be required to definitively diagnose myopericarditis and associated etiology. However, an endomyocardial biopsy is not recommended in patients with no or mild left ventricular dysfunction (ejection fraction greater than or equal to 45%).

Unexplained initial leukocytosis at 18.1 K/uL, worsening thrombocytopenia, decreasing platelet levels, and anemia prompted a PBS which demonstrated suspicion of blast cells; this required further evaluation via flow cytometry. Immunohistochemistry staining of bone marrow biopsy with CD34 can be used to diagnose AML if ≥20% blasts present.<sup>3</sup> In our patient, flow cytometry revealed acute myeloid leukemia with 26.5% CD34 positive myeloblasts circulating in blood. The presence of blasts on peripheral smear and bone marrow biopsy confirmed the patient’s AML diagnosis. After exclusion of other etiologies, we determined the cause of her STEMI-like presentation and myopericarditis to be AML due to the close associate of the two.

## Conclusion

- This case study illustrates an unusual case of an 84-year-old female presenting with myopericarditis as an initial manifestation of AML.
- The awareness of such atypical presentation presentations can lead to a timely diagnosis and intervention in patients with AML.
- Moreover, early peripheral blood smear evaluation, particularly in the setting of leukocytosis, anemia, and STEMI-like symptoms and/or myopericarditis may be effective in the early diagnosis of patients with AML.

## References

1. Newell LF, Cook RJ. Advances in acute myeloid leukemia. *BMJ*. 2021 Oct 6;375:n2026. doi: 10.1136/bmj.n2026. PMID: 34615640.
2. Ferrel MN, Ryan JJ, Han FT. Acute myeloid leukemia causing acute thrombosis of the coronary arteries: a case report. *J Med Case Rep*. 2022 Apr 12;16(1):149. doi: 10.1186/s13256-022-03280-3. PMID: 35413942; PMCID: PMC9003162.
3. Narayanan D, Weinberg OK. How I investigate acute myeloid leukemia. *Int J Lab Hematol*. 2020 Feb;42(1):3-15. doi: 10.1111/ijlh.13135. Epub 2019 Dec 10. PMID: 31820579.
4. Ferrel, M.N., Ryan, J.J. & Han, F.T. Acute myeloid leukemia causing acute thrombosis of the coronary arteries: a case report. *J Med Case Reports* 16, 149 (2022).
5. Agrawal K, Miles L, Agrawal N, Khan A. Atypical Presentation of Acute Myeloid Leukemia. *World J Oncol*. 2018 Feb;9(1):29-34. doi: 10.14740/wjon1083w. Epub 2018 Mar 8. PMID: 29581813; PMCID: PMC5862080.