



Management of Acute Myeloid Leukemia in Young Adult in the Peripheral Area: A Case Report

Clarissa Nadia Gultom¹, Said Ansori²

¹Dokter umum, RSUD Bangka Tengah, Kota Koba, Bangka Belitung, Indonesia,

²Dokter Spesialis Penyakit Dalam, RSUD Bangka Tengah, Kota Koba, Bangka Belitung, Indonesia

ABSTRACT

Introduction: Acute myeloid leukemia (AML) is an aggressive and rapidly progressing hematopoietic malignancy that originates from myeloid stem cells in the bone marrow. **Case:** An 18-year-old male presented with nausea and vomiting for 5 days, preceded by frequent fatigue and weight loss over the past month. Vital signs were within normal limits. Physical examination revealed anemic conjunctivae and palpable enlargement of the liver and spleen. Laboratory examination revealed extreme leukocytosis, severe anemia, and thrombocytopenia. The patient was diagnosed with acute myeloid leukemia. Management was symptomatic with blood transfusion, and referred for further treatment. **Discussion:** AML is generally characterized by nonspecific symptoms such as anemia, fever, bleeding, hepatosplenomegaly, as well as hematological abnormalities including leukocytosis, anemia, and thrombocytopenia. The diagnosis is made by examining peripheral blood and bone marrow, while the prognosis is influenced by the patient's age, subtype, and cytogenetic abnormalities. **Conclusion:** AML in young adults may present with nonspecific symptoms such as fatigue, nausea, vomiting, and weight loss, which can delay diagnosis. Marked hematologic abnormalities, including extreme leukocytosis, severe anemia, and thrombocytopenia, should raise suspicion for AML. Early recognition, appropriate supportive management, and timely referral from peripheral healthcare facilities are essential to improve patient outcomes and survival.

Keywords: Acute myeloid leukemia, bone marrow, case report, hematopoietic malignancy, leukocytosis, myeloid.

ABSTRAK

Pendahuluan: *Acute myeloid leukemia* adalah keganasan hematopoietik agresif berasal dari sel induk mieloid di sumsum tulang. **Kasus:** Pria berusia 18 tahun datang dengan keluhan tidak spesifik yaitu mual disertai muntah selama 5 hari. Pasien mengeluh sering merasa lelah dan berat badan turun dalam 1 bulan terakhir. Tanda-tanda vital dalam batas normal. Pada pemeriksaan fisik dijumpai kedua mata tampak anemis dan pembesaran hepar dan limpa. Pada pemeriksaan laboratorium dijumpai leukositosis ekstrim, anemia berat, dan trombositopenia. Pasien didiagnosis *acute myeloid leukemia*. Tata laksana sesuai keluhan dan transfusi darah. Selanjutnya, pasien dirujuk untuk penanganan lebih lanjut. **Pembahasan:** AML umumnya ditandai gejala tidak spesifik seperti anemia, demam, perdarahan, hepatosplenomegali, serta kelainan hematologi berupa leukositosis, anemia, dan trombositopenia. Diagnosis ditegakkan melalui pemeriksaan darah tepi dan sumsum tulang, sedangkan prognosis dipengaruhi usia, sub tipe, dan kelainan sitogenetik pasien. **Simpulan:** AML pada dewasa muda dapat muncul dengan gejala yang tidak spesifik seperti lemas, mual, muntah, dan penurunan berat badan, sehingga diagnosis sering terlambat ditegakkan. Adanya kelainan hematologi berupa leukositosis ekstrim, anemia berat, dan trombositopenia harus meningkatkan kecurigaan terhadap AML. Pengenalan dini, tata laksana suportif yang adekuat, dan rujukan tepat waktu dari fasilitas kesehatan perifer sangat penting untuk meningkatkan luaran klinis dan harapan hidup pasien. **Clarissa Nadia Gultom, Said Ansori. Tata Laksana Acute Myeloid Leukemia pada Dewasa Muda di Daerah Perifer: Laporan Kasus.**

Kata Kunci: *Acute myeloid leukemia*, sumsum tulang, laporan kasus, keganasan hematopoietic, leukositosis, mieloid.

<https://doi.org/10.55175/cdk.v53i06.1853>



Mermin Dunia Kedokteran is licensed under a Creative Commons Attribution-NonCommercial 4.0 International License.

INTRODUCTION

Acute myeloid leukemia (AML) is a bone marrow disease, a hematopoietic stem cell disorder caused by genetic changes in

blood cell precursors resulting in excessive production of clonal neoplastic myeloid stem cells.¹ AML is relatively rare, accounting for only 1.1% of cancer diagnoses; however,

AML is the second most common type of leukemia and accounts for 1.9% of cancer deaths.² AML can be fatal, and life-threatening complications can quickly

Alamat Korespondensi clarissanadiaa@gmail.com



develop in asymptomatic patients.²

AML should be suspected in individuals with easy bruising or bleeding, recurrent infections, acute (within days or weeks) unexplained cytopenia, and the presence of blast cells in the peripheral blood.³

The incidence of AML has increased in recent years, from 63,840 cases in 1990 to 119,570 cases in 2017 (an increase of 87.3%).⁴ Geographically, Western Europe and South Asia had the highest incidence in 2017, namely 20,020 cases and 21,460 cases, respectively. Southeast Asia also has a fairly high incidence rate, namely 8,970 cases in 2017. Western Europe, South Asia, and North America are the 3 regions with the most AML-related deaths.⁴ The incidence of AML increases with age.^{5,6} Due to the high recurrence rate, survival of this disease is poor.⁶

The etiology of AML is still unknown; Several risk factors identified as potentially leukemogenic are exposure to cigarette smoke, exposure to certain chemicals such as benzene and formaldehyde, certain chemotherapy drugs, exposure to high doses of radiation, blood disorders such as

MDS (myelodysplastic syndrome), genetic mutations and chromosomal abnormalities such as Fanconi anemia, Bloom's syndrome, ataxia-telangiectasia, Down's syndrome, and trisomy 8.⁷

CASE

An 18-year-old man, with complaints of nausea accompanied by vomiting for 5 days. Nausea is felt at any time, not influenced by activity or position. Vomits about 3 times a day, containing food. The patient has felt weak and tired easily over the last 2 weeks, with no improvement with rest. Patients experience gum swelling and decreased appetite. Weight loss of around 5 kg in the last 1 month. The patient had no history of previous disease.

On physical examination, blood pressure was 90/60 mmHg, heart rate 85 times/minute, respiratory rate 19 times/minute, temperature 36.6°C, and saturation 98% without oxygen. The conjunctiva of both eyes appeared anemic, and the liver and spleen were palpably enlarged. Laboratory examination revealed extreme leukocytosis, severe anemia, and thrombocytopenia (**Table 1**). Peripheral blood analysis showed normocytic normochromic erythrocytes and a greatly increased leukocyte count, suggestive of myeloid leukemia (**Table 2**). Ultrasound

showed hepatosplenomegaly. The provisional diagnosis was leukocytosis, ec malignant, severe anemia, and thrombocytopenia.

The patient was admitted for further evaluation. Transfusion therapy of 1,500 mL PRC (packed red cells), accompanied by blood-boosting tablets (ferrous fumarate 60 mg, folic acid 400 mcg) 1 x 1, vitamin C 2 x 250 mg, vitamin D 1 x 400 IU, and 0.9% NaCl infusion 20 drops per minute. The patient was also given an IV injection of ondansetron 3 x 4 mg until complaints of nausea and vomiting subsided.

The patient felt better after the transfusion. The patient was diagnosed with acute myeloid leukemia and referred for further management. However, the patient decided not to continue treatment and examination. The patient chose an alternative treatment.

DISCUSSION

AML is an aggressive and rapidly growing hematopoietic malignancy that originates from myeloid stem cells in the bone marrow. Normally, these progenitor cells differentiate into myeloid-derived blood cells, primarily granulocytes and monocytes. In AML, genetic abnormalities disrupt normal hematopoietic differentiation, leading to uncontrolled cell proliferation. These immature, abnormal cells, blast cells, rapidly accumulate in the bone marrow and bloodstream, ultimately resulting in disruption of normal hematopoiesis, bone marrow failure, fatal infections, bleeding, and organ infiltration.⁶ Abnormal differentiation of myeloid cells results in more immature malignant cells and fewer differentiated red blood cells, platelets, and white blood cells.⁸

The median age at diagnosis of AML is 68 years in the United States, and the incidence continues to increase with age.⁵ In adults aged 60 years and over, the incidence is 3–4 cases per 100,000 people per year. The incidence of AML in young adults aged between 15 and 39 years is lower than in older people, estimated at around 0.7 to 2.0 cases per 100,000 people per year.⁹ Most AML cases found in the 11–20-year age range were diagnosed using bone marrow aspiration. A prospective cross-sectional study of acute myeloid leukemia cases over a 2-year period found that there were more

Table 1. Laboratory examination during hospital treatment.

| Laboratory | H1 | H3 | H5 | Unit | Reference |
|-------------|------|-------|------|---------------------|-----------|
| Leukocyte | 161 | 188.9 | 184 | 10 ³ /uL | 4–10 |
| Hemoglobin | 3,7 | 5.6 | 9.4 | g/dL | 13–18 |
| Hematocrit | 11 | 16 | 26 | % | 40–54 |
| Erythrocyte | 0.98 | 1.65 | 2.83 | million/uL | 4.5–6.2 |
| MCV | 113 | 98 | 93 | fL | 81–96 |
| MCH | 38 | 34 | 33 | Pg | 27–36 |
| MCHC | 33 | 35 | 36 | g/L | 31–37 |
| Platelet | 130 | 119 | 147 | 10 ³ /uL | 150–400 |

Abbreviations: MCV: Mean corpuscular volume; MCH: Mean corpuscular hemoglobin; MCHC: Mean corpuscular hemoglobin concentration.

Table 2. Peripheral blood analysis results.

| | |
|-------------|---|
| Erythrocyte | Normocytic normochromic was found in normoblasts in 5/100 leukocytes. |
| Leukocyte | The number increased greatly; all granulocyte series were found from blast to segment, with segment predominance. |
| Platelet | Sufficient quantity, spread out. |
| Impression | Suspect acute myeloid leukemia. |



male cases (57.15%) than female cases (42.85%).¹⁰ In another study, data showed that AML was slightly more common in men, but the average lifetime risk for both sexes was about 0.5% (1 in 200 Americans).⁶

The clinical signs and symptoms of AML are varied and nonspecific, often due to cytopenia caused by leukemic cell infiltration of the bone marrow. Patients present with fatigue, bleeding, infection, and fever due to decreased red blood cells, platelets, or white blood cells. Paleness, fatigue, and shortness of breath during activity are often found.¹¹ Some cases are discovered through routine blood tests, and others may present with symptoms of complications.¹

Symptoms include recurrent infections, anemia, easy bruising, excessive bleeding, headaches, and bone pain. General weakness, fatigue, shortness of breath, and chest tightness, depending on the degree of anemia. The development of these symptoms is relatively rapid, often within a few days to weeks.³ Leukemic cells also tend to infiltrate various body tissues, such as the gums, spleen, skin, lymph nodes, and central nervous system. Recurrent and severe bleeding episodes and infections are life-threatening complications of leukemia.¹²

This case presented with non-specific symptoms, namely nausea accompanied by vomiting, fatigue, and weight loss in the last 1 month. The patient experienced gum swelling and decreased appetite. On physical examination of the abdomen, hepatosplenomegaly was found. Common findings in AML include bruising and hepatosplenomegaly, whereas lymphadenopathy is rare. Myeloid sarcoma, the equivalent of myeloid, can appear as thickened, hyperpigmented, and rough skin lesions. Disseminated intravascular

coagulation (DIC), clinically characterized by oral mucosal bleeding, purpura, extremity petechiae, and bleeding from intravenous line sites, is common in AML.³

Peripheral blood smear examination is very important. Characteristic features, in addition to generalized thrombocytopenia, include blasts, which are large, immature leukocytes with a high nuclear-to-cytoplasmic ratio, irregular nuclear contour, and smooth chromatin with prominent or numerous nucleoli. Blasts usually have a pale or dark blue cytoplasm with varying eosinophilic coloration. In addition, schistocytes can be observed in DIC when it occurs simultaneously.³ The results of routine blood tests in this case showed that the hemoglobin level was very low, namely 3.7 g/dL, leukocytosis was found at $161 \times 10^3/\mu\text{L}$, and a decrease in the number of platelets, namely $130 \times 10^3/\mu\text{L}$.

In one study, hemoglobin concentrations ranged from 3.1 to 16 g%. Nine (64.28%) patients showed hemoglobin values between 7.1 and 10 g%. Leukocytosis was found in 12/14 acute leukemia patients. Two (14.28%) patients were classified as subleukemia. The platelet count is between $50 \times 10^9/\text{L}$ and $1.50 \times 10^9/\text{L}$. Seven (50%) patients experienced severe thrombocytopenia.¹⁰

Anemia interferes with tissue oxygenation. If hemodynamics are stable, PRC transfusion is recommended if Hb is $< 7 \text{ g/dL}$.¹³ Prophylactic platelet transfusion is also given to stable patients without bleeding if the platelet count is less than $10 \times 10^9/\text{L}$. In febrile patients, the threshold increases to $20 \times 10^9/\text{L}$.¹⁴

Patients require adequate hydration. Complaints of nausea and vomiting are treated with 5-HT₃ receptor antagonists, such as ondansetron, which block serotonin

receptors in the central nervous system and the digestive tract. Patients are also given ferrous fumarate and folic acid, which are important for the formation of hemoglobin. In one study of AML therapy, a reduced risk of bacterial and fungal infections, bleeding, and reduced macrophage activation was found in the group given vitamins C and D.¹⁵ Once the condition was stable, patients were advised to be referred for further examination and treatment.

Life expectancy depends on age, with an estimated 5-year survival of 62% in patients diagnosed before age 50 years, 37% for patients aged 50–64 years, and only 9.4% for patients aged 65 years and older at diagnosis.¹⁶ In younger patients, a complete remission rate of $\geq 80\%$ can be achieved.¹⁷

The prognosis of AML is influenced by subtype, chromosomal abnormalities (cytogenetics), age, leukocyte count, hematological abnormalities that cause AML, AML caused by other cancer therapy, infection, and AML status after therapy.¹⁷

CONCLUSION

AML can occur in young adults and may initially present with nonspecific symptoms, making early diagnosis challenging. This case demonstrates that extreme leukocytosis, severe anemia, thrombocytopenia, and hepatosplenomegaly are important clinical clues that should prompt further hematologic evaluation. Early recognition, appropriate supportive management, and timely referral from peripheral healthcare settings are essential to optimize treatment opportunities and improve patient outcomes.

Informed Consent

The patient or a family member has provided written or verbal consent for the publication of the manuscript and all identifiable data.

REFERENCES

1. Pelcovits A, Niroula R. Acute myeloid leukemia: a review. *R I Med J* (2013) [Internet]. 2020;103(3):38–40. PMID: 32236160.
2. Stubbins RJ, Francis A, Kuchenbauer F, Sanford D. Management of acute myeloid leukemia: a review for general practitioners in oncology. *Curr Oncol*. 2022;29(9):6245–59. <https://doi.org/10.3390/curroncol29090491>.
3. Vakiti A, Reynolds SB, Mewawalla P. Acute myeloid leukemia [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 [cited 2026 May 28]. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK507875/>.
4. Irawan C, Steven R, Gunarsa RG, Tenggara JB. Luaran Hasil Leukemia Mieloid Akut yang Menjalani Terapi pada Ruang Kemoterapi Semi-Isolasi. *J Penyakit Dalam Indones*. 2022;9(3):155. <https://doi.org/10.7454/jpdi.v9i3.857>



5. Bhansali RS, Pratz KW, Lai C. Recent advances in targeted therapies in acute myeloid leukemia. *J Hematol Oncol.* 2023;16(1):29. <https://doi.org/10.1186/s13045-023-01424-6>.
6. Karjalainen E, Repasky GA. Molecular changes during acute myeloid leukemia (AML) evolution and identification of novel treatment strategies through molecular stratification. *Prog Mol Biol Transl Sci.* 2016 Jan 1;144:383–436. <https://doi.org/10.1016/bs.pmbts.2016.09.005>.
7. Yuliana. Perkembangan terapi leukemia mieloid akut di RS Siloam Balikpapan. *Cermin Dunia Kedokt.* 2017;44(3):216–20. <https://doi.org/10.55175/cdk.v44i3.835>.
8. Khwaja A, Bjorkholm M, Gale RE, Levine RL, Jordan CT, Ehninger G, et al. Acute myeloid leukaemia. *Nat Rev Dis Prim.* 2024;147(2):229–46. <https://doi.org/10.1038/nrdp.2016.10>
9. Wachter F, Pikman Y. Pathophysiology of acute myeloid leukemia. *Acta Haematologica.* 2024;147:232–49. <https://doi.org/10.1159/000536152>.
10. Nagar V, Patil N, Gudur A, Gudur R. Clinicopathological study of acute myeloid leukemia in a tertiary care hospital. *Int J Health Sci (Qassim).* 2022 Apr 8;11:3070–7. <https://doi.org/10.53730/ijhs.v6nS2.5735>.
11. Kabel AM, Zamzami F, Al-Talhi M, Al-Dwila K, Hamza R. Acute myeloid leukemia: a focus on risk factors, clinical presentation, diagnosis and possible lines of management. *J Cancer Res Treat.* 2017;5(2):62–7. <https://doi.org/10.12691/jcrt-5-2-4>.
12. Misirlioglu M, Adisen MZ, Yilmaz S. Diagnosis of acute myeloid leukemia in a dental hospital; report of a case with severe gingival hypertrophy. *Niger J Clin Pract.* 2015 Jul 1;18(4):573–6. <https://doi.org/10.4103/1119-3077.151803>.
13. Trentino KM, Farmer SL, Leahy MF, Sanfilippo FM, Isbister JP, Mayberry R, et al. Systematic reviews and meta-analyses comparing mortality in restrictive and liberal haemoglobin thresholds for red cell transfusion: an overview of systematic reviews. *BMC Med.* 2024;18(1):154. <https://doi.org/10.1186/s12916-020-01614-w>.
14. Chan KY, Chan TSY, Gill H, Chan TCW, Li CW, Au HY, et al. Supportive care and symptom management in patients with advanced hematological malignancies: a literature review. *Ann Palliat Med.* 2022;11(10):3273–91. doi: 10.21037/apm-22-691.
15. Mouchel PL, Berard E, Tavitian S, Gadaud N, Vergez F, Rieu JB, et al. Vitamin C and D supplementation in acute myeloid leukemia. *Blood Adv.* 2023;7(22):6886–97. doi: 10.1182/bloodadvances.2023010559.
16. Hastuti TS, Sumantri R, Wijaya I. Complete remission of acute myeloid leukemia in induction and consolidation chemotherapy without bone marrow transplantation: lessons learned from good presentation case. *Maj Kedokt Bandung.* 2019;51(1):31–8. <https://doi.org/10.15395/mkb.v51n1.1634>.