

A Rare Case of Hypocellular Acute Myeloid Leukemia: Clinical and Morphological Features

Nadir Bir Hiposelüler Akut Miyeloid Lösemi Olgusu: Klinik ve Morfolojik Özellikler

Alper Koç¹, Hadice Akyol², Rafet Eren³

¹University of Health Sciences Türkiye, Elazığ Fethi Sekin City Hospital, Clinic of Hematology, Elazığ, Türkiye

²University of Health Sciences Türkiye, Elazığ Fethi Sekin City Hospital, Clinic of Pathology, Elazığ, Türkiye

³Biruni University Faculty of Medicine Hospital, Department of Hematology, İstanbul, Türkiye

To the Editor,

Acute myeloid leukemia (AML) is a myeloid neoplasm characterized by the clonal proliferation of myeloid precursor cells in the bone marrow and peripheral blood. The neoplastic accumulation of these blast cells usually leads to a hypercellular bone marrow morphology. However, in a small subset of AML patients, hypocellular bone marrow may be observed despite significant blast infiltration [1].

A 65-year-old male patient with no prior medical history presented with a 2-week history of fatigue, progressive exhaustion, and headache. On physical examination, he appeared pale, with blood pressure of 98/61 mmHg and pulse rate of 102 bpm. No lymphadenopathy or hepatosplenomegaly was detected. Laboratory investigations revealed hemoglobin of 3.1 g/dL, hematocrit of 8.5%, white blood cell count of $1.3 \times 10^9/L$, neutrophil count of $0.09 \times 10^9/L$, and platelet count of $15 \times 10^9/L$. Lactate dehydrogenase was 270 U/L. Renal and liver function tests, serum electrolytes, and nutritional parameters were within normal limits. A peripheral blood smear showed normocytic and normochromic red blood cells, thrombocytopenia, and leukopenia. No blast cells were observed. The bone marrow aspiration was markedly hypocellular. No megakaryocytes were observed. The majority of nucleated cells identified were consistent with blasts. Immunophenotypic analysis revealed that 76% of these cells were myeloblasts expressing CD45, CD34, CD117, CD13, CD33, and HLADR. A bone marrow core biopsy examination showed hypocellular marrow with less than 20% cellularity (Figure 1). Approximately 80% of that limited number of cells were myeloblasts expressing myeloid and immature markers. The patient was diagnosed with hypocellular AML and started on induction therapy with venetoclax and azacitidine.

Hypocellular leukemia is characterized by bone marrow cellularity of less than 20% [2]. Bone marrow cellularity below 40%-50% has been considered hypocellularity in previous studies and the majority of hypocellular acute leukemia cases are AML, generally observed in older patients, as in our case [3]. Hypocellular acute leukemias reported in young adults and pediatric age groups are typically acute lymphoblastic leukemias. Hypocellular AML accounts for 5%-12% of all AML cases, and more than half of the cases are reported to develop secondary to radiotherapy or chemotherapy [2]. As in our case, in patients presenting with pancytopenia and hypocellular bone marrow, distinguishing from disorders such as hypoplastic myelodysplastic syndrome and aplastic anemia can be challenging. Guidelines have been published to establish diagnostic criteria and achieve a standardized approach for the differential diagnosis of these disorders [4]. However, as cellularity decreases, differential diagnosis becomes more difficult. The presence of 20% or more blasts in the bone marrow helps to differentiate hypocellular AML from other entities. In patients with lower blast percentages, morphological evaluation becomes more challenging, and flow cytometry and cytogenetic analyses are used as complementary methods. The hypocellular AML cases reported in the literature were treated using standard AML protocols and similar response rates were achieved [5]. In conclusion, a small subset of AML patients present with hypocellular AML, which may lead to diagnostic challenges. With this letter, we aim to draw attention to hypocellular AML and to contribute our case, along with its images, to the limited body of literature.

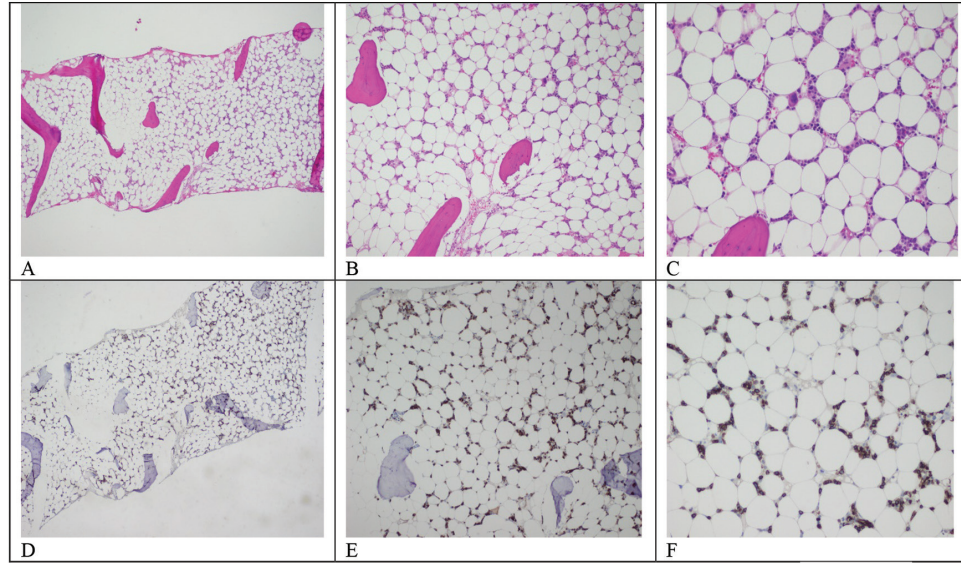


Figure 1. Findings from the patient's bone marrow biopsy. A, B, C; Hypocellular bone marrow at 40 $\times$, 100 $\times$, and 200 $\times$ magnification, respectively, with hematoxylin and eosin staining. D, E, F; Myeloblasts at 40 $\times$, 100 $\times$, and 200 $\times$ magnification, respectively, with CD34 staining.

Keywords: Hypocellular leukemia, Acute myeloid leukemia, Hypocellularity

Anahtar Sözcükler: Hiposelüler lösemi, Akut miyeloid lösemi, Hiposellülarite

Ethics

Informed Consent: Informed consent was obtained from the patient for this case report.

Footnotes

Authorship Contributions

Surgical and Medical Practices: A.K., H.A.; Concept: A.K., H.A.; Design: H.A., R.E.; Data Collection or Processing: H.A.; Analysis or Interpretation: R.E.; Literature Search: A.K.; Writing: A.K.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

References

1. Tuzuner N, Cox C, Rowe JM, Bennett JM. Hypocellular acute myeloid leukemia: the Rochester (New York) experience. *Hematol Pathol.* 1995;9:195-203.
2. Al-Kali A, Konoplev S, Lin E, Kadia T, Faderl S, Ravandi F, Ayoubi M, Brandt M, Cortes JE, Kantarjian H, Borthakur G. Hypocellular acute myeloid leukemia in adults: analysis of the clinical outcome of 123 patients. *Haematologica.* 2012;97:235-240.
3. Čolović N, Denčić-Fekete M, Peruničić M, Jurišić V. Clinical characteristics and treatment outcome of hypocellular acute myeloid leukemia based on WHO classification. *Indian J Hematol Blood Transfus.* 2020;36:59-63.
4. Bennett JM, Orazi A. Diagnostic criteria to distinguish hypocellular acute myeloid leukemia from hypocellular myelodysplastic syndromes and aplastic anemia: recommendations for a standardized approach. *Haematologica.* 2009;94:264-268.
5. Song IC, Jo DY, Kim HJ, Min YH, Hong DS, Lee WS, Shin HJ, Lee JH, Park J, Kim HJ. Clinical features and outcomes of hypocellular acute myeloid leukemia in adults: a Korean AML registry data. *Medicine (Baltimore).* 2021;100:e24185.



Address for Correspondence/Yazışma Adresi: Alper Koç, M.D., University of Health Sciences Türkiye, Elazığ Fethi Sekin City Hospital, Clinic of Hematology, Elazığ, Türkiye
E-mail: alperkoc44@hotmail.com ORCID: orcid.org/0000-0003-0844-2658

Received/Geliş tarihi: May 4, 2025
Accepted/Kabul tarihi: May 27, 2025
Epub: May 27, 2025
DOI: 10.4274/tjh.galenos.2025.2025.0176



©Copyright 2025 by Turkish Society of Hematology Turkish Journal of Hematology, Published by Galenos Publishing House.
Licensed under a Creative Commons Attribution-NonCommercial (CC BY-NC-ND) 4.0 International License.