

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

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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 two-week history of fatigue, progressive exhaustion, and headache. On physical examination, he appeared pale, with a blood pressure of 98/61 mmHg and a pulse rate of 102 bpm. No lymphadenopathy or hepatosplenomegaly was detected. Laboratory investigations revealed a 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. Peripheral blood smear showed normocytic, normochromic red blood cells, thrombocytopenia, and leukopenia. No blast cells were observed. 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. Bone marrow core biopsy examination of the patient showed a hypocellular marrow with less than 20% cellularity (Figure 1). Approximately 80% of 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 a bone marrow cellularity of less than 20% (2). Bone marrow cellularity below 40-50% is considered hypocellular in previous studies. The majority of hypocellular acute leukemia cases are AML, and, as in our case, they are generally observed in older patients (3). Hypocellular acute leukemias reported in young 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 are published to establish diagnostic criteria and achieve a standardized approach for differential diagnosis of these disorders (4). However, as cellularity decreases, differential diagnosis becomes more difficult. 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. 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 presents with hypocellular AML, which may lead to diagnostic challenges. With this letter, we aimed to draw attention to hypocellular AML and to contribute our case, along with its images, to the limited body of literature.

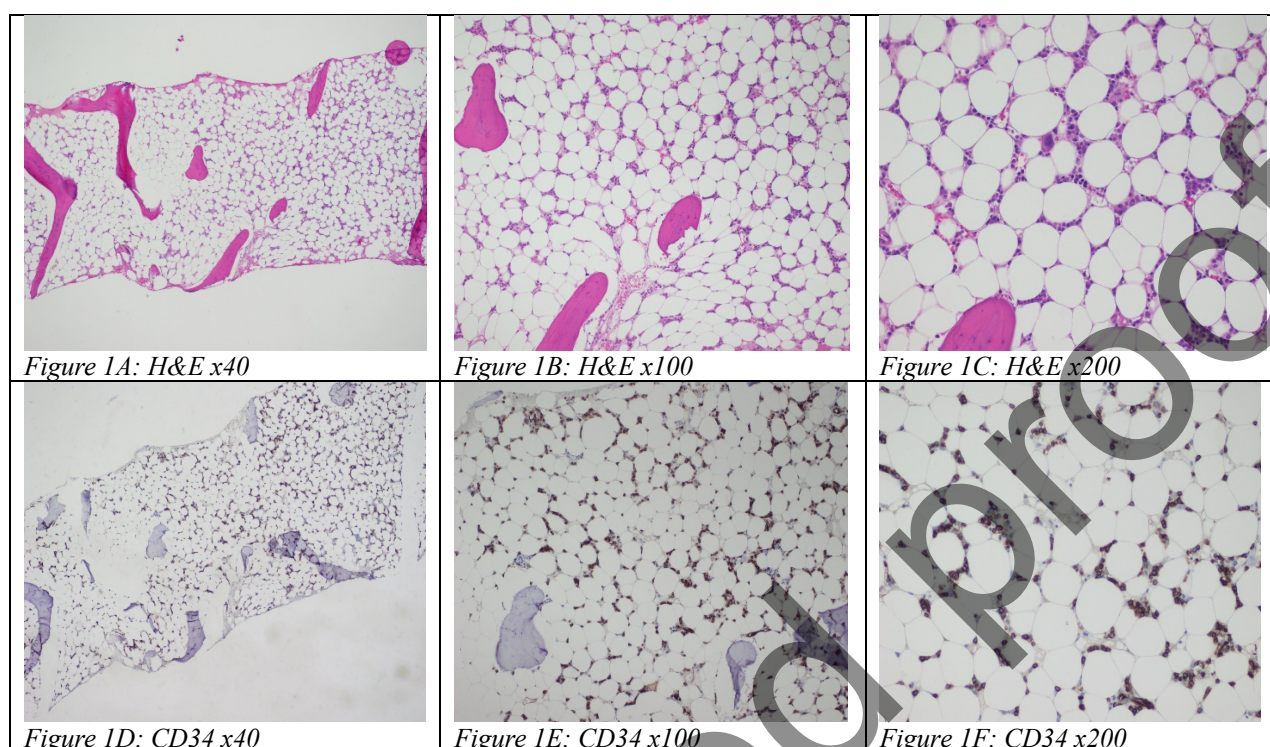


Figure 1: Patient's bone marrow biopsy, 1A-1B-1C; hypocellular bone marrow at 40x, 100x, and 200x magnification in hematoxylin-eosin (H&E) staining, respectively. 1D-1E-1F; myeloblasts at 40x, 100x, and 200x magnification with CD34 staining, respectively.

References

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