DOI: 10.4274/tjh.galenos.2025.2024.0391
Turk J Hematol 2025;42:142-143

# Acute Myeloid Leukemia with *NUP98::LNP1* Fusion Mimicking Chronic Myeloid Leukemia

## Kronik Miyeloid Lösemiyi Taklit Eden *NUP98::LNP1* Füzyonu ile Seyreden Akut Miyeloid Lösemi

Haiyang Wang[1]*, Yu Peng[2]*, Zailin Yang[2]

[1]*The Affiliated Hospital of Xuzhou Medical University, Department of Hematology, Xuzhou, P.R. China*
[2]*Chongqing University Cancer Hospital, Chongqing Key Laboratory for the Mechanism and Intervention of Cancer Metastasis, Department of Hematology-Oncology, Chongqing, P.R. China*
**These authors contributed equally to this work.*

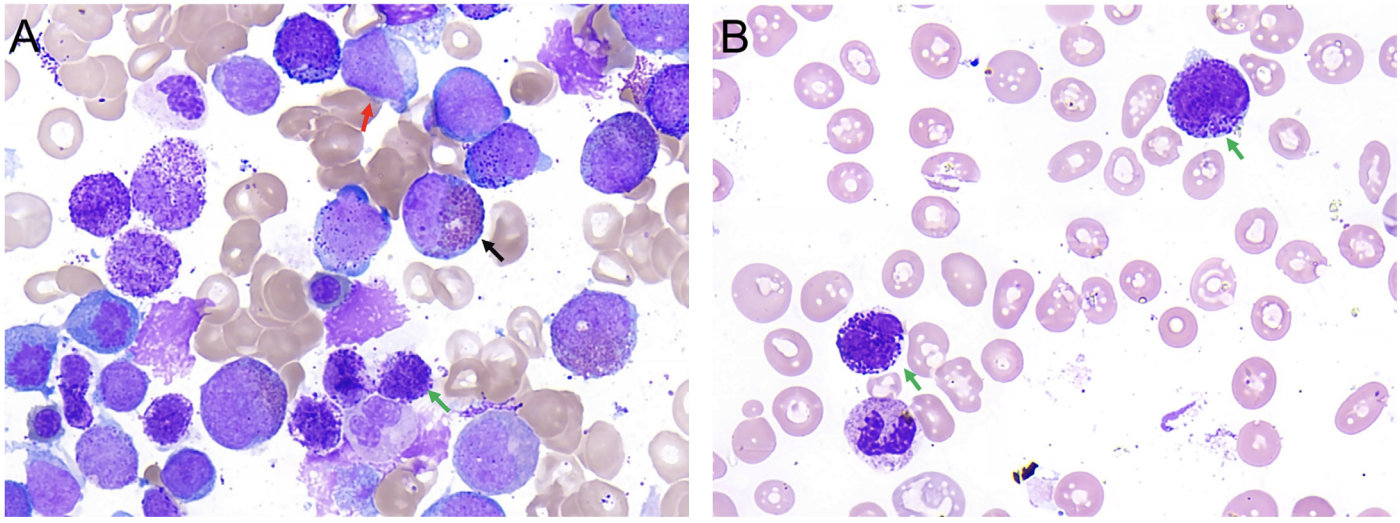


**Figure 1.** A, B) The red arrow points to a myeloblast, the black arrow points to an eosinophil, and the green arrows point to basophils. Original magnification of 1000$^{x}$, Wright-Giemsa stain.

A 60-year-old female patient presented with a history of hepatitis, tuberculosis, and gastric cancer. She had developed recurrent fever during the treatment for gastric cancer. Peripheral blood (PB) examination showed white blood cell count of 47.7x10$^{9}$/L, with 1% blasts, 1% promyelocytes, 2% neutrophilic myelocytes, 5% neutrophilic metamyelocytes, 13% neutrophils, 8% lymphocytes, 3% monocytes, 3% eosinophils, and 64% basophils. Hemoglobin was 87 g/L and platelet count was 75x10$^{9}$/L. A bone marrow (BM) smear revealed active proliferation of granulocytes with 10% myeloblasts (Figure 1A). The proportions of eosinophils and basophils in the BM were also notably increased, accounting for 9% and 24% of nucleated cells, respectively (Figure 1A). Additionally, the percentage of basophils in the PB was significantly elevated, reaching 52%

**Address for Correspondence/Yazışma Adresi:** Zailin Yang, M.D., Chongqing University Cancer Hospital, Chongqing Key Laboratory of Translational Research for Cancer Metastasis and Individualized Treatment, Department of Hematology-Oncology, Chongqing, P.R. China
**E-mail:** zailinyang@cqu.edu.cn **ORCID:** orcid.org/0000-0001-8773-1022

**Received/Geliş tarihi:** October 14, 2024
**Accepted/Kabul tarihi:** March 17, 2025

(Figure 1B). The morphological features of the BM and PB closely mimicked chronic myeloid leukemia in the chronic high-risk phase, characterized by a significant elevation in basophils. Interestingly, both quantitative reverse-transcription polymerase chain reaction and fluorescence in situ hybridization indicated negativity for the *BCR::ABL1* fusion gene. The chromosomal karyotype was 46,XX,t(3;11)(q12.2;p15.4). Meanwhile, RNA sequencing identified only the *NUP98::LNP1* fusion transcript, with no detectable mutations in other genes such as *ASXL1*, *NRAS*, *KRAS*, *SRSF2*, *TET2*, *CBL*, *CSF3R*, *JAK2*, *ETNK1*, or *SETBP1*. Ultimately, a diagnosis of acute myeloid leukemia (AML) with *NUP98* rearrangement was established, as AML with *NUP98* rearrangements is exempt from the historical threshold of 20% blasts according to the 2022 guidelines of the World Health Organization [1,2].

AML with *NUP98::LNP1* fusion transcripts is extremely rare. To date, only one case has been reported in which blasts exceeded 20% [3]. The present case is the first reported instance of this specific morphological presentation in AML with *NUP98::LNP1* fusion transcripts. Numerous studies have demonstrated that *NUP98* rearrangements are associated with unfavorable prognosis, highlighting the critical importance of their identification [4,5,6].

**Keywords:** Acute myeloid leukemia, *NUP98*::*LNP1*, Morphological presentation

**Anahtar Sözcükler:** Akut myeloid lösemi, *NUP98::LNP1*, Morfolojik görünüm

### Ethics

**Informed Consent:** Written informed consent was obtained from the individual for the publication of any potentially identifiable images or data included in this article.

### Footnotes

### Authorship Contributions

Data Collection or Processing: H.W., Y.P.; Analysis or Interpretation: H.W., Z.Y.; Literature Search: Y.P., Z.Y.; Writing: H.W., Y.P., Z.Y.

**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. Khoury JD, Solary E, Abla O, Akkari Y, Alaggio R, Apperley JF, Bejar R, Berti E, Busque L, Chan JKC, Chen W, Chen X, Chng WJ, Choi JK, Colmenero I, Coupland SE, Cross NCP, De Jong D, Elghetany MT, Takahashi E, Emile JF, Ferry J, Fogelstrand L, Fontenay M, Germing U, Gujral S, Haferlach T, Harrison C, Hodge JC, Hu S, Jansen JH, Kanagal-Shamanna R, Kantarjian HM, Kratz CP, Li XQ, Lim MS, Loeb K, Loghavi S, Marcogliese A, Meshinchi S, Michaels P, Naresh KN, Natkunam Y, Nejati R, Ott G, Padron E, Patel KP, Patkar N, Picarsic J, Platzbecker U, Roberts I, Schuh A, Sewell W, Siebert R, Tembhare P, Tyner J, Verstovsek S, Wang W, Wood B, Xiao W, Yeung C, Hochhaus A. The 5th edition of the World Health Organization Classification of Haematolymphoid Tumours: Myeloid and Histiocytic/Dendritic Neoplasms. Leukemia. 2022;36:1703-1719.
2. The WHO Classification of Tumours Editorial Board. WHO Classification of Tumors: Haematolymphoid Tumours, 5th ed. Lyon, IARC Press, 2024.
3. Gorello P, Brandimarte L, La Starza R, Pierini V, Bury L, Rosati R, Martelli MF, Vandenberghe P, Wlodarska I, Mecucci C. t(3;11)(q12;p15)/NUP98-LOC348801 fusion transcript in acute myeloid leukemia. Haematologica. 2008;93:1398-1401.
4. Heikamp EB, Henrich JA, Perner F, Wong EM, Hatton C, Wen Y, Barwe SP, Gopalakrishnapillai A, Xu H, Uckelmann HJ, Takao S, Kazansky Y, Pikman Y, McGeehan GM, Kolb EA, Kentsis A, Armstrong SA. The menin-MLL1 interaction is a molecular dependency in *NUP98*-rearranged AML. Blood. 2022;139:894-906.
5. Tian J, Zhu Y, Li J, Yang G, Weng X, Huang T, Zhao L, Sun H, Yan Z, Zhang S. The landscape of NUP98 rearrangements clinical characteristics and treatment response from 1491 acute leukemia patients. Blood Cancer J. 2024;14:81.
6. Michmerhuizen NL, Klco JM, Mullighan CG. Mechanistic insights and potential therapeutic approaches for NUP98-rearranged hematologic malignancies. Blood. 2020;136:2275-2289.