Annals of Hematology (2025) 104:2545–2549
https://doi.org/10.1007/s00277-025-06312-2

CASE REPORT

Promising activity of Selinexor in the treatment of a patient with refractory *NUP98-NSD1+/FLT3-ITD+* acute myeloid leukemia

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Received: 1 December 2024 / Accepted: 9 March 2025 / Published online: 18 March 2025

Abstract

Nucleoporin 98 (NUP98) fusion oncoproteins are associated with various hematologic malignancies. Acute myeloid leukemia (AML) with *NUP98-NSD1* typically co-occurs with *FLT3-ITD* mutations, exhibiting poor initial responses to traditional chemotherapy. This case report describes a relapsed and refractory AML case co-expressing *NUP98/NSD1* and *FLT3/ITD* after matched sibling haplo-identical allogeneic hematopoietic stem cell transplantation, achieving molecular remission with a salvage therapy combining selinexor, venetoclax, and azacitidine. To our knowledge, this is the first report demonstrating the effectiveness of this combination therapy for relapsed/refractory *NUP98-NSD1+/FLT3-ITD+*AML. This report highlights the potential synergy between selinexor and established AML therapies, suggesting a promising approach to improve outcomes for refractory AML patients.

Keywords NUP98-NSD1 · FIT3-ITD · Acute myeloid leukemia · Selinexor · Hematopoietic stem cell transplantation

Background

Chromosomal translocations represent critical genetic lesions in acute myeloid leukemia (AML). The t(5;11)(q35;p15.4) translocation, which fuses the *NUP98* and *NSD1* genes, is a recurrent aberration in AML [1]. Translocations involving *NUP98* occur at relatively low frequency in AML but can be missed with routine karyotyping. The *NUP98-N*SD1 fusion gene is found in about 16.1% of pediatric and 2.3% of adult AML patients with a normal karyotype and poor response to conventional chemotherapy [2]. Additionally, the *NUP98-NSD1* fusion gene frequently co-occurs with *FLT3-ITD* mutations, leading to higher rates of induction failure [3–6]. Patients with *NUP98-NSD1+/FLT3-ITD+*AML are considered high-risk, with reduced event-free survival and a tendency to develop chemotherapy-resistant disease.

Despite our understanding of the molecular mechanisms and co-occurring genetic events in NUP98-rearranged malignancies, effective treatment options for these patients remain limited. Developing targeted therapies for NUP98 fusion-positive malignancies is therefore a crucial and promising pursuit. One potential strategy is to develop inhibitors targeting the plant homeodomain domain, as several fusion partners depend on this region for sustained cell growth [7]. Another successful approach explored in experimental models involves inhibiting upregulated transcriptional targets, such as CDK6 [8]. Additionally, NUP98 fusion proteins are recruited to their target genes through the mixed lineage leukemia (MLL) complex, which requires a direct interaction between MLL and Menin. Targeting this Menin-MLL interaction has shown promise in inhibiting the propagation of NUP98-rearranged AML both ex vivo and in vivo [9]. Another potential approach is to inhibit cofactors necessary for the formation of nuclear puncta and transcriptional regulation. For example, inhibiting a cofactor like XPO1 could prevent access to critical gene loci, such as the HOXA locus, thereby blocking the expression of NUP98 fusion target genes [1]. Here, we reported a refractory case of molecular relapse of AML co-expressing *NUP98/NSD1*

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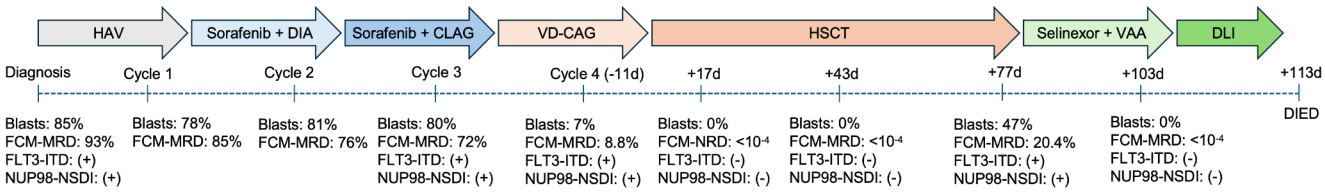

Fig. 1 Clinical course of the patient. HAV: homoharringtonine, cytarabine, and venetoclax; DIA: decitabine, idarubicin, and cytarabine; CLAG: cladribine, cytarabine, and granulocyte colony-stimulating factor; VD-CAG: venetoclax, decitabine, aclacinomycin, cytarabine, and granulocyte-colony stimulating factor; HSCT: hematopoietic stem cell transplantation; VAA: venetoclax, azacitidine, and cytarabine; DLI: donor lymphocyte infusion; d: day.

and *FLT3/ITD* post-matched sibling allogeneic hematopoietic stem cell transplantation (allo-HSCT). After adding selinexor combined with venetoclax and azacitidine to the treatment regimen, the patient achieved a molecular remission.

Case description

A 37-year-old male presented with a history of dizziness and fatigue. Laboratory results showed a white blood cell count of 165×10^9/L, hemoglobin of 3.8 g/dL, platelet count of 63×10^9/L. Myeloid blast cells of bone marrow accounted for 85%, and morphological typing was M5. Chromosomal analysis revealed a normal karyotype, but molecular testing showed the presence of the *NUP98-NSD1* fusion and *FLT3-ITD* mutation, confirming the diagnosis of high-risk AML.

The patient initially received induction chemotherapy with the HAV regimen (homoharringtonine, cytarabine, and venetoclax). Bone marrow examination on day 28 showed no CR. He was then treated with sorafenib in combination with the DIA regimen (decitabine, idarubicin, and cytarabine) and the CLAG regimen (cladribine, cytarabine, and granulocyte colony-stimulating factor). However, bone marrow aspiration after two cycles revealed 80% blast cell hyperplasia, indicating chemotherapy resistance and refractory AML. Next, the treatment regimen was adjusted to VD-CAG regimen (venetoclax, decitabine, aclacinomycin, cytarabine, and granulocyte-colony stimulating factor). Venetoclax dosing was adjusted based on antifungal prophylaxis. After VD-CAG, minimal residual disease decreased to 8.8%, and the *NUP98-NSD1* fusion ratio dropped to 1.06%.

The patient then underwent allo-HSCT using his haploidentical sister as the donor. The pre-transplantation regimen included fludarabine, busulfan, thiotepa, and melphalan, while graft-versus-host disease prevention involved cyclophosphamide, anti-human T lymphocyte porcine immunoglobulin, cyclosporine, and mycophenolate mofetil. The nucleated cell dose infused was 11.7×10^8/kg, and the CD34 + cell dose was 16.9×10^6/kg. Neutrophil engraftment occurred on day 14, and platelet engraftment on day 11 post-transplantation. By days 17 and 43, both peripheral blood and bone marrow were negative for blasts, and flow cytometry confirmed that minimal residual disease was undetectable. Additionally, both the *NUP98-NSD1* fusion gene and *FLT3-ITD* mutation were absent. However, on day 77 post-transplantation, bone marrow blasts had increased to 47%, indicating AML recurrence. The *NUP98-NSD1* fusion ratio rose to 2.24%, and the *FLT3-ITD* mutation was detected at a 1.42% frequency. The patient was then treated with selinexor plus the VAA regimen (venetoclax, azacitidine, and cytarabine). After one cycle, the patient achieved morphologic leukemia-free state, with both the *NUP98-NSD1* fusion gene and *FLT3-ITD* mutation becoming undetectable. The patient then received donor lymphocyte infusion. Despite this, the patient ultimately passed away due to a severe fungal infection. The clinical course of the patient is shown in Fig. 1.

Discussion and conclusions

Early case reports noted unfavorable outcomes in NUP98-rearranged leukemia patients [10–12], and subsequent studies confirmed that NUP98 gene fusions define a high-risk subset of leukemia [2–4, 13–15]. Moreover, these findings extend to co-occurring mutations, especially *FLT3-ITD* [3–6]. Previous studies reported that patients with *NUP98-NSD1*+/*FLT3-ITD* + AML had a CR rate of only 30%, with cumulative recurrence rates as high as 80–90% [4]. Ostronoff et al. [5] found that patients with both *NUP98-NSD1* and *FLT3-ITD* had double the recurrence and mortality rates compared to those with similar diagnoses without these genetic aberrations. Bolouri et al. [3] reported a significantly lower 10-year overall survival rate for AML patients with both *FLT3-ITD* mutations and *NUP98-NSD1* fusions compared to patients without these mutations or with only *FLT3-ITD* mutations. While the molecular mechanisms and co-occurring genetic events of NUP98 rearrangements are well understood, effective treatment options remain limited.

To improve the outcomes for patients with *NUP98*-rearranged malignancies, therapeutic strategies targeting transcriptional and epigenetic machinery, cooperating mutations, and signaling pathways are being explored [1].

Venetoclax, combined with hypomethylating agents, has shown high response rates and prolonged survival in AML. Kivioja et al. [16] found that cells expressing *NUP98-NSD1* were significantly more sensitive to the B-cell lymphoma-2 inhibitor. Given that *NUP98* fusion often co-occurs with other mutations, combination therapies may improve outcomes. Sun et al. [17] reported that venetoclax and azacitidine could improve treatment responses in adult AML patients with *NUP98-NSD1*, and the inclusion of FLT3 inhibitors and HSCT could fully reverse poor outcomes. Similarly, our patients achieved partial remission and a reduced tumor burden following treatment with the VD-CAG regimen. And he achieved molecular CR after HSCT, with the *NUP98-NSD1* fusion becoming negative. Unfortunately, this benefit was temporary, highlighting the need for effective targeted therapies for *NUP98* fusion-positive malignancies.

Previous studies have shown that NUP98 fusions interact with the *MLL1* protein complex and colocalize with *MLL1* on the HoxA/B cluster genes [18]. Recent research has also emphasized the crucial role of Menin, a protein that directly interacts with *MLL1*, in NUP98-rearranged leukemia. Genetic inactivation or small molecule inhibition of menin has demonstrated anti-leukemic effects in NUP98-rearranged leukemia models [19]. However, while menin inhibitors have shown anti-leukemic activity, durable responses have not been achieved, especially in the presence of concomitant synergistic mutations [9, 19, 20]. Additionally, 50% of AML patients with *MLL1* rearrangements or *NPM1* mutations did not respond to menin inhibitors, and approximately 38% of patients treated with Revumenib developed mutations in menin, leading to clinical resistance [21]. Furthermore, a significant challenge is that menin inhibitors are currently difficult to obtain in China. Thus, other targeted therapies or combinatorial treatments may be necessary to achieve improved clinical efficacy and overcome resistance.

The N-terminus of *NUP98* contains intrinsically disordered phenylalanine-glycine (FG)/glycine-leucine-phenylalanine-glycine (GLFG) repeats that interact with cofactors [22, 23]. The intrinsically disordered FG/GLFG repeat domains in wild-type *NUP98* mediate phase separation [24], and *NUP98* fusion oncoproteins have been shown to localize in nuclear puncta [1, 2]. XPO1, a nuclear export protein, has been identified in these puncta and interacts with the FG/GLFG repeat domains in a Ran guanosine triphosphate–dependent manner [25]. *NUP98* fusions inhibit XPO1-dependent nuclear export, resulting in the retention of transcription factors such as NF-κB and nuclear factor of activated T cells, which enhances transcription of their downstream targets [25]. Inhibition of XPO1 cargo-binding reduces DNA binding near Hox gene promoters and alters the localization of nuclear puncta [26]. Selinexor, an XPO1 inhibitor, has been shown to synergize with hypomethylating agents (HMAs) and B-cell lymphoma-2 (BCL-2) inhibitors to induce apoptosis in AML cells [27, 28]. AML cell lines and primary blasts demonstrated that the effectiveness of decitabine was significantly enhanced when combined with selinexor [28]. Additionally, selinexor has been shown to synergize with venetoclax to induce apoptosis in AML cells by downregulating MCL-1 [27]. Furthermore, both selinexor and eltanexor have demonstrated synergy with the BCL-2 inhibitor venetoclax in double-hit lymphoma cell lines and patient-derived xenografts harboring MYC and BCL-2 alterations [29]. Based on these findings, we chose a regimen including selinexor for our patient, and interestingly, the combination with venetoclax and azacitidine induced the negative of *NUP98-NSD1* fusion gene and *FLT3-ITD* mutation. We observed that, based on venetoclax, HMAs, and cytarabine therapy, replacing aclarubicin with selinexor led to a dramatic improvement in efficacy, shifting from partial remission to molecular remission. This not only demonstrates the synergistic effect of selinexor with venetoclax and HMAs but also underscores the promising potential of selinexor for patients with NUP98-rearranged AML. However, it is important to note that the patient only achieved morphologic leukemia-free state, prompting us to perform a donor lymphocyte infusion, which may help achieve a more sustained response [30]. Despite this, the duration of remission remains uncertain, as the patient ultimately succumbed to a severe infection.

To our knowledge, this is the first report demonstrating that the combination of selinexor, venetoclax, and azacitidine is an effective therapy for relapsed/refractory *NUP98-NSD1*+/*FLT3-ITD*+AML. Further studies are needed to explore the potential synergistic mechanisms of this combination therapy.

Author contributions X.L.Y. designed the research. K.Y. and B.B.Y. performed the research, analyzed the data and wrote the paper. Y.L.Z. and Q.Y.H. contributed essential data collection. All authors read and approved the final manuscript.

Funding No funding was received to assist with the preparation of this manuscript.

Data availability No datasets were generated or analysed during the current study.

Declarations

Ethics approval The study was approved by the Ethics Committee of 923rd Hospital of the Joint Logistics Support Force of the People's Liberation Army.

Competing interests The authors declare no competing interests.

Consent to participate Full written consent was obtained from the le-

gal guardians of patient for the writing and publication of this manuscript.

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