Basic research

Hematology

Expression and clinical significance of SENP1 in patients with acute myeloid leukemia

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Abstract

Introduction: The study aimed to investigate the expression of SUMO1/sentrin-specific peptidase 1 (SENPl) in patients with acute myeloid leukemia (AML) and its significance in the development of AML.
Material and methods: Patients with AML, hematological malignancies, and non-neoplastic hematological diseases were selected as the experimental subjects, and they were divided into the initial diagnosis group (n = 32), the relapse group (n = 8), and the remission group (n = 22) according to their disease status. Meanwhile, patients with non-neoplastic hematological diseases were selected as the controls (n = 30). The expression of SENP1 mRNA and protein in bone marrow samples from the different groups was detected. The effects of SENP1 silencing on cell multiplication and the cell cycle were observed.
Results: The expression of SENP1 from low to high was observed in the controls, initial diagnosis, remission, and relapse groups. The incidence of adverse conditions was the lowest in the initial diagnosis group, followed by the remission group and the relapse group. The proliferation of U937 decreased after SENP1 knockdown, suggesting that SENP1 has a major role in AML cell multiplication. In addition, the apoptosis rate of U937 was increased after SENP1 knockdown. The overall survival (OS) and disease-free survival (DFS) were ranked as follows: relapsed < initial diagnosis < remission < controls.
Conclusions: This article preliminarily demonstrated the high expression of the biomarker SENP1 in AML patients, and found that it was related to the clinical characteristics and prognosis of AML patients.

Key words: acute myeloid leukemia, SENP1, biomarkers, prognosis.

Introduction

Acute myeloid leukemia (AML), a hematological malignancy, is caused by the malignant proliferation of immature myeloid cells in the bone marrow [1, 2]. It often leads to serious symptoms and complications in a short period [3]. Symptoms of AML include fatigue, anemia, easy bleeding and congestion, infections, fever, bone pain, etc. These symptoms are caused by the accumulation of leukemia cells in the bone marrow, inhibiting normal hematopoietic function and resulting in abnormal numbers of red blood cells, platelets, and white blood cells in the blood.

Methods for the treatment of AML include chemotherapy, hematopoietic stem cell transplantation, and targeted therapy [4–6]. The medical community divides AML into eight types, but prognostic evaluation re-

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mains challenging. WHO classifies AML into five categories, including AML of unknown lineage, not specially classified AML, treatment-related AML nuclear myelodysplastic syndrome, AML with multilineage dysplasia, and AML with recurrent genetic abnormalities [7]. Although the WHO classification method is more specific for the prognosis and treatment evaluation of AML with recurrent genetic abnormalities, there are still some patients who cannot be accurately evaluated for prognosis.

Some studies have shown that AML is associated with chromosomal and genetic abnormalities, but the pathogenesis is still unclear [8, 9]. Studies have shown that post-translational protein modification is important in tumor development, among which SUMOylation is an important form of modification. Small ubiquitin-like modifier (SUMO) is a small protein that functions as a post-translational modifier. SUMOylation, the covalent attachment of SUMO to target proteins, is involved in the regulation of various cellular processes, including gene transcription, DNA repair, cell cycle progression, and apoptosis. SENP1 (SUMO1/sentrin-specific peptidase 1) is a specific deSUMOylating enzyme that catalyzes the removal of SUMO modifications from substrate proteins. By modulating the dynamic balance between SUMOylation and deSUMOylation, SENP1 may play a role in tumor initiation and progression. SENP1 can catalyze the removal of SUMO from many proteins, which is related to the development of varieties of tumors [10]. SENP1 is involved in the development of B cells, T cells, and red blood cells, suggesting that the differentiation and development of hematopoietic cells are closely related to SENP1 [11, 12]. Chemotherapy is the main treatment for AMS and uses drugs to kill leukemia cells. Hematopoietic stem cell transplantation is a method of transplanting healthy blood stem cells into a patient's body in order to restore normal hematopoietic function. Targeted therapy is the use of specific drugs or antibodies to treat specific molecules in leukemia cells [13]. Protein abnormalities act in the prognosis and biological characteristics of AML, among which post-translational protein modification is the most delicate regulation. Common post-translational modifications of proteins in eukaryotic cells include SUMOylation, methylation, acetylation, phosphorylation, and ubiquitination.

At present, studies on the expression and significance of SENP1 in AML patients are relatively limited. Therefore, this study aimed to further explore SENP1 expression in people with AML and evaluate its relationship with prognosis.

Material and methods

Subjects

Between October 2020 and April 2023, patients diagnosed with AML in the Department of Hematology of Xianyang Central Hospital were selected for the study. These included newly diagnosed patients, those in complete remission, and those with relapsed AML. Patients with non-malignant hematologic diseases were included as the control group. The inclusion criteria for patients with AML were as follows: diagnosis confirmed by morphology, immunology, cytogenetics, and molecular biology (MICM), and no concurrent systemic malignancies. They were grouped as follows: initial diagnosis group (n = 32), relapse group (n = 8), and remission group (n = 22). The control group consisted of patients with non-malignant hematologic diseases (n = 30).

Bone marrow samples were collected from all subjects, including 2 ml of bone marrow aspirate or 2 ml of peripheral blood mononuclear cell (PBMC) samples, which could be used instead of bone marrow samples when the peripheral blood leukemia cell count was > 80% in patients with newly diagnosed AML before initial chemotherapy. Two milliliters of bone marrow aspirate were collected from the controls. Inclusion criteria: all enrolled patients met the diagnostic and treatment criteria for hematologic diseases. Informed written consent was obtained from all participants. Exclusion criteria: patients with other malignant tumors or those unwilling to participate in the study.

Acute monocytic leukemia cell lines

The cell line U937 is a commonly adopted laboratory model to study the pathogenesis and treatment of acute monocytic leukemia. The U937 strain was originally derived from a peripheral blood sample from a patient with acute monocytic leukemia. These cells exhibit characteristics of monocytes and can proliferate indefinitely under culture conditions. The U937 cell line has been widely adopted in basic and clinical research on monocyte differentiation, apoptosis, immune regulation, and other processes. U937 cells were obtained from the Department of Hematology Laboratory, the Second Affiliated Hospital of Xi'an Jiaotong University.

T-PCR

Monocytes in bone marrow blood of the experimental group and the controls were isolated and the total RNA was extracted, using a reverse transcription kit (TaKaRa) for synthesis of cDNA and Bio-Rad PCR amplification. After the end of the reaction, the expression of SENP1 was calculated by $2^{-\Delta\Delta Ct}$. The specific operation steps are displayed in Table I.

Western blot

Protein immunoblotting (Western blot) is a commonly used experimental technique to de-

tect the presence and expression of specific proteins in samples [14, 15]. Mononuclear cells were isolated from the bone marrow samples of the experimental group and the controls. The cells were lysed using RIPA Lysis Buffer (RIPA) to extract protein, and the loading samples were prepared. The protein was analyzed by Quantity One gel analysis software, the absorbance of each protein band was detected, and the protein expression was calculated. Each protein sample was tested in triplicate and the average value was taken.

Cell culture

Cell culture refers to the process of removing cells from the *in vivo* or *in vitro* environment and providing suitable conditions in an appropriate medium so that they can continue to grow and reproduce *in vitro* [16, 17]. U937 cells were cultured in a sterile mixture of fetal bovine serum and RPMI-1640 medium in a ratio of 1 : 9. The culture conditions were 37°C and 5% CO_2 under saturated humidity. The growth of U937 cells was observed regularly. When the cells were in log phase, they were used for the experiments.

Cell transfection

Cell transfection refers to the process of introducing foreign genes or other biological molecules into cells, which is widely used in biological research [18, 19]. It can be used in gene function research, gene therapy, drug screening, and other fields. U937 cells in the logarithmic growth phase were digested with trypsin. Cells were seeded in 6-well plates at 1×10^5 cells per well and cultured for 24 h. When cell confluence reached approximately 75%, transfection was performed according to the Lipofectamine 2000 manufacturer's instructions. The cells were divided into two groups: sh-NC and sh-SENP1. The silencing efficiency of sh-SENP1 was determined by RT-qPCR 48 h after transfection.

Cell cycle detected by flow cytometry

U937 cells were divided into sh-NC and sh-SENP1 groups. The cells in the logarithmic growth phase were seeded in 6-well plates at 5×10^4 cells per well. After the cells adhered to the wells, the medium was discarded, and trypsin was added. The cells were gently tapped to facilitate detachment, and the resulting single-cell suspension was collected. After that, they were washed twice with 1 ml of precooled PBS, supplemented with fluorescein stain PI and ribonuclease, and the cells were incubated at 37°C in the dark for 0.5 h. The distribution in G0/G1, S, and G2/M phases was determined by flow cytometry. All experiments were repeated three times to ensure the accuracy and reliability of the results.

Cell Counting Kit-8 (CCK-8) assay to detect cell multiplication

CCK-8 is a commonly used assay for assessing cell proliferation and cytotoxicity. The method uses WST-8, a water-soluble tetrazolium salt, as an indicator to determine the metabolic activity of cells through a reduction reaction [20, 21]. Forty-eight h after transfection, the cells were digested, resuspended, and seeded in 96-well plates at 5,000 cells per well. A total of 100 µl of medium was added to each well, and the cells were cultured overnight.

Table I. Operation steps for PCR amplification of DNA

Serial number	Steps
1	DNA template preparation: Extracting DNA from the sample.
2	PCR reaction system preparation: Determining the required composition of the PCR reaction system according to the experimental design.
3	Primer design: upstream 5′-AAGATTCCCAGACTCCAACTCCCA-3′; downstream 5′-TGAATGTTCCCGCTCCTGCAAT-3′.
4	Setting the PCR reaction conditions: Determining the appropriate PCR reaction conditions according to the length and sequence characteristics of the target DNA.
5	PCR amplification: The PCR reaction system was added to a PCR tube or microplate and put into a PCR instrument for amplification.
6	PCR cycle: a PCR reaction typically consists of three main steps: denaturation, annealing, and extension.
7	PCR cycles: according to the required number of target DNA amplification, appropriate PCR cycles were set.
8	At end of PCR reaction: after completing the PCR reaction, stopping heating and maintaining the reaction system at low temperature to prevent the production of non-specific amplification products.
9	Analysis of PCR products: the PCR products were analyzed and detected by gel electrophoresis, real-time PCR, or other methods.

Table II. Comparison of baseline characteristics of the subjects

Groups	Number of participants	Sex (male/female)	Age [years]	BMI [kg/m^2]
Initial diagnosis	32	14/18	66.4 ±5.7	23.5 ±4.7
Relapse	8	4/4	65.3 ±4.8	17.8 ±5.8
Remission	22	12/10	67.7 ±6.3	18.5 ±6.3
Controls	30	16/14	65.8 ±7.54	24.3 ±5.5

Next, the cells were treated according to the CCK-8 kit instructions. At 24, 48, 72, and 96 h after inoculation, 10 µl of CCK-8 assay solution was applied. Incubation was carried out for 4 h. The absorbance at 450 nm was measured, and the growth curve was plotted. Each experiment was performed three times to ensure reproducibility. By comparing cell viability between the sh-NC and sh-SENP1 groups, the association of SENP1 knockdown with U937 cell proliferation could be evaluated.

Statistical analysis

Statistical analyses were performed using SPSS version 21.0. Continuous variables were expressed as mean ± standard deviation ($\bar{x} \pm s$). Comparisons between two groups were conducted using the independent samples *t*-test, while comparisons among multiple groups were performed using one-way analysis of variance (ANOVA), followed by Tukey's post hoc test. The Kaplan–Meier method was employed to construct overall survival (OS) and disease-free survival (DFS) curves, and differences between groups were assessed using the log-rank test. A two-tailed *p*-value < 0.05 was considered statistically significant. Sample size and power analysis: Due to the clinical difficulty in recruiting patients with relapsed AML, a prospective power analysis was not performed. A retrospective power calculation using G*Power indicated that, assuming an effect size (Cohen's *d*) of 0.8 and a significance level of $\alpha = 0.05$ (two-tailed), the statistical power for detecting differences in SENP1 expression between the newly diagnosed group ($n = 32$) and the relapse group ($n = 8$) was approximately 65%. Given the limited sample size, particularly in the relapse group, the study may have had limited power to detect smaller differences.

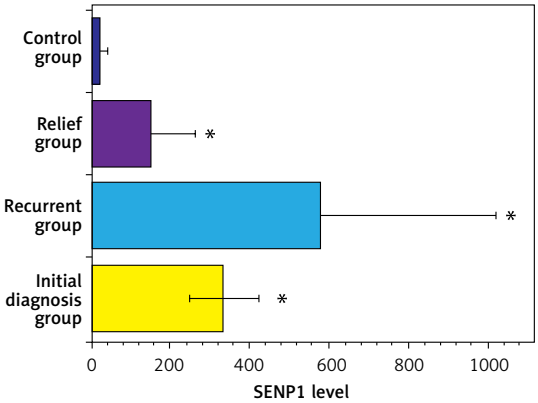


Figure 1. Comparison of RT-PCR results of SENP1 expression

**Represents versus controls, p < 0.05.*

Results

General characteristics

The general characteristics of the subjects are displayed in Table II. There were no significant differences in gender, age, or body mass index (BMI) among the initial diagnosis group, the relapse group, the remission group, and the controls.

SENP1 expression

In Figure 1, SENP1 expression was lowest in the control group and highest in the relapse group. Compared to the control group, the 95% confidence interval for SENP1 expression in the newly diagnosed group was 1.98–2.70, which was significantly higher ($p < 0.05$). The 95% confidence interval for SENP1 expression in the relapse group was 4.35–5.89, with a highly significant difference compared to the control group ($p < 0.001$).

Figure 2 presents the Western blot bands of SENP1 for each patient group. The band intensity of SENP1 in the control group was the lowest, while the intensity in the relapse group was the highest. The band intensities in the initial diagnosis and remission groups were intermediate.

Comparison of poor prognosis

The 95% confidence interval for the incidence of adverse prognosis in the relapse group was 56.8–93.2%, which was significantly higher than that of the newly diagnosed group (16.3–45.9%) and the remission group (32.5–67.5%). The χ^2

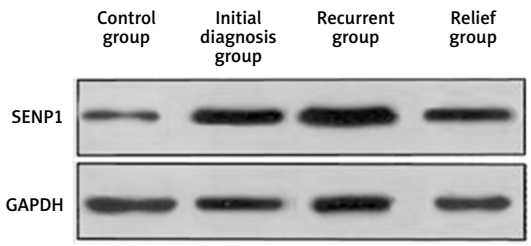


Figure 2. Western blot bands of SENP1 for each patient group

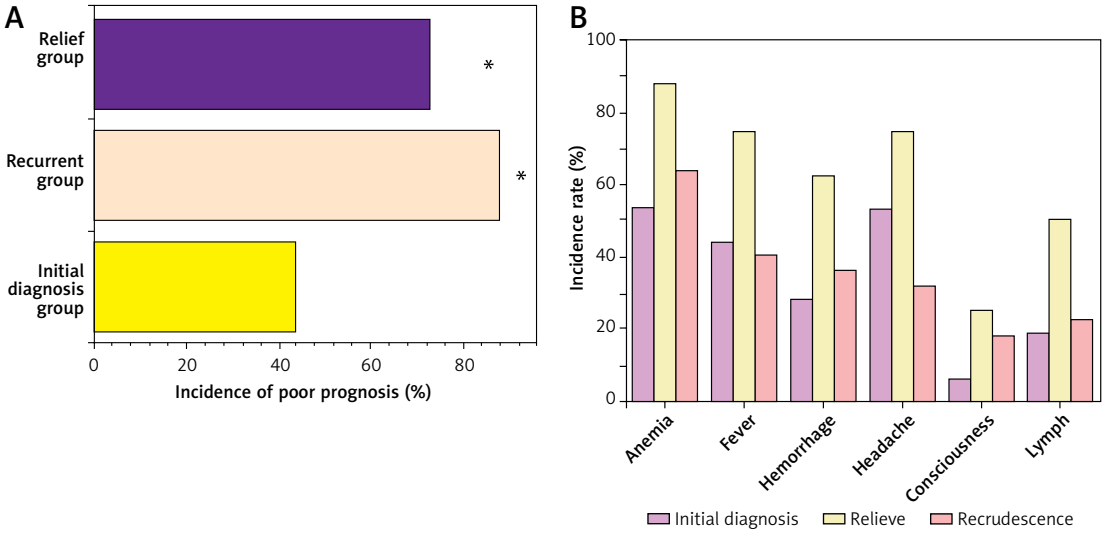


Figure 3. Comparison of poor prognosis. **A** – Comparison of the incidence of poor prognosis. **B** – The specific incidence of adverse conditions

**Represents relapse group or remission group versus initial diagnosis group, $p < 0.05$.*

test demonstrated that the differences among the groups were highly significant (χ^2 = 18.72, $p < 0.001$) (Figure 3).

U937 transfection results

After transfection in sh-SENP1 group, the SENP1 expression level in U937 cells decreased by about 70% as compared with the sh-NC group, which indicated that the transfected cell line was stable with knockdown of SENP1 (Figure 4).

Effect of SENP1 on AML cell multiplication

Under normal serum culture conditions, a stable transfection cell line was constructed to explore the role of SENP1 in AML cell proliferation. The results showed that SENP1 knockdown in U937 cells significantly reduced cell proliferation, suggesting that SENP1 may play a role in promoting AML cell proliferation (Figure 5).

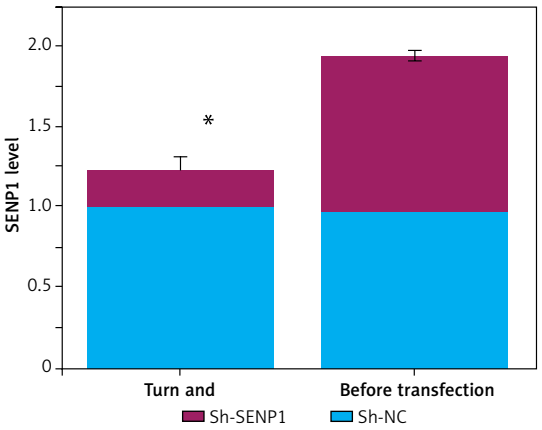


Figure 4. Results of U937 transfection

**Means sh-SENP1 versus sh-NC, $p < 0.05$.*

Effect of SENP1 on AML apoptosis

The effect of SENP1 on spontaneous apoptosis of AML cells was investigated using the constructed stable transfected cell lines under normal culture conditions. The cells were collected, and the difference in the apoptotic rate between the sh-SENP1 and sh-NC groups was determined using the Annexin V/PI method. The apoptotic rate of U937 cells increased after SENP1 knockdown (Figure 6).

Relationship of SENP1 with DFS and OS in AML

Kaplan–Meier analysis (Figure 7) demonstrated that the 95% confidence intervals for the 1-year OS rates were 85.2–98.8% in the control group and 18.2–56.8% in the relapse group, with a sig-

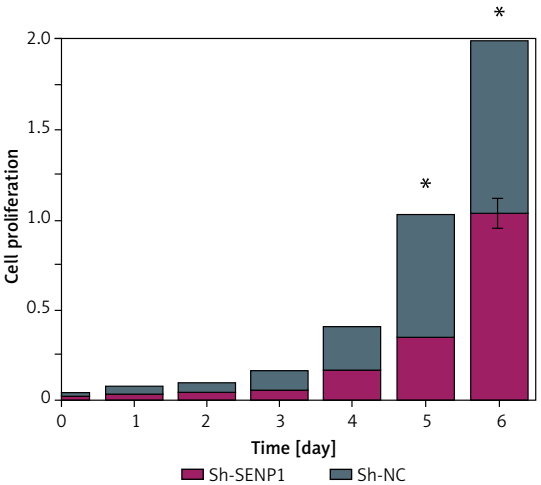


Figure 5. Comparison of cell multiplication ability between sh-SENP1 and sh-NC groups after SENP1 knockdown

**Means sh-SENP1 versus sh-NC, $p < 0.05$.*

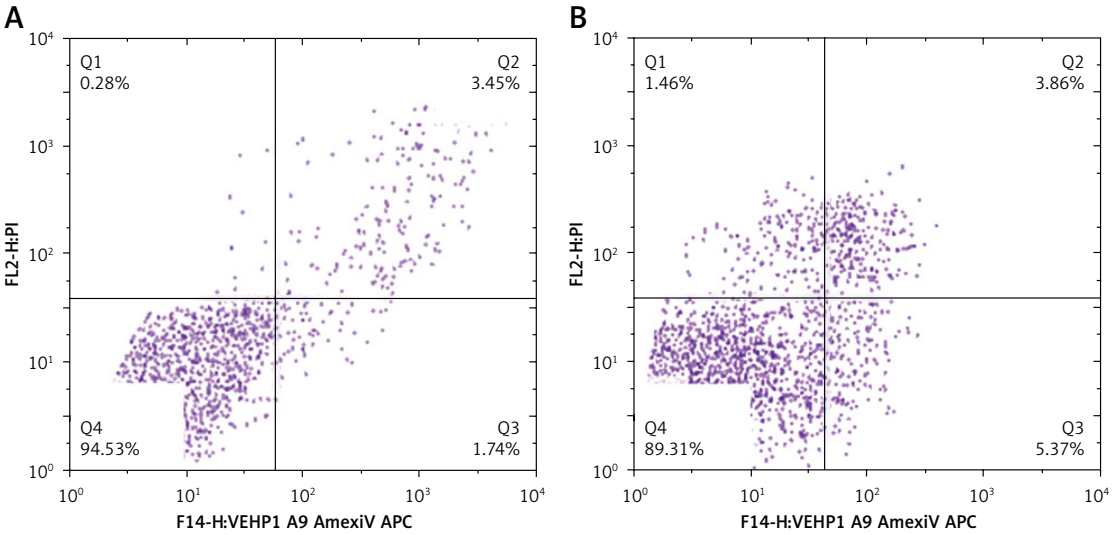


Figure 6. Spontaneous apoptosis rates in sh-SENP1 and sh-NC groups. **A** – spontaneous apoptosis rate in sh-NC, **B** – spontaneous apoptosis rate in sh-SENP1. Note: Q1, Q2, Q3, and Q4 represent G0/G1, S, G2, and M phases, respectively

nificant difference between the survival curves (log-rank test, $p < 0.001$). The 95% confidence intervals for the 1-year OS rates in the newly diagnosed and remission groups were 68.3–87.9% and 72.5–91.3%, respectively, with a significant difference between these groups ($p = 0.021$).

Discussion

AML is a common hematological malignancy with an incidence of approximately 4 per 100,000 and is more common in middle-aged and elderly men. The accumulation of immature myeloid precursor cells in patients with AML leads to impaired hematopoietic function, resulting in symptoms such as infection, bleeding, and anemia, and eventually may lead to death [22]. With the increase of the elderly population in China, the incidence of AML is increasing and posing a threat to public health. Studies have shown that SENP1 is involved in tumor development, and previous studies have reported relevant findings [23, 24]. Studies have found high SENP1 expression in AML patients' tissues. This is consistent with the results of previous studies, suggesting that SENP1 may be involved in the occurrence and progression of AML. SENP1 is an important ubiquitin-like modifying enzyme involved in the regulation of a variety of cellular processes, including gene transcription, DNA repair, and apoptosis. The high expression of SENP1 may be related to the abnormal proliferation, anti-apoptosis, and decreased DNA damage repair ability of AML cells.

Research suggests that SENP1 may play a role in AML. Experiments have found that highly expressed SENP1 is associated with poor prognosis of people with AML. Specifically, highly expressed SENP1 was associated with shorter OS and DFS in people with AML. This may be because high SENP1 expression promotes AML cell proliferation and inhibits apoptosis and immune responses. As a deubiquitylation enzyme, SENP1 can regulate the activity of various of signaling pathways involved in processes such as apoptosis and DNA repair. Thus, high SENP1 expression may promote AML cell proliferation and inhibit apoptosis [25]. The proliferation rate of AML cells decreased after SENP1 knockdown. Through cell multiplication assay and cycle analysis, it was found that SENP1 knockdown prolonged the G1 phase of AML cells, thereby limiting cell multiplication. In addition, SENP1 knockdown promoted apoptosis of AML

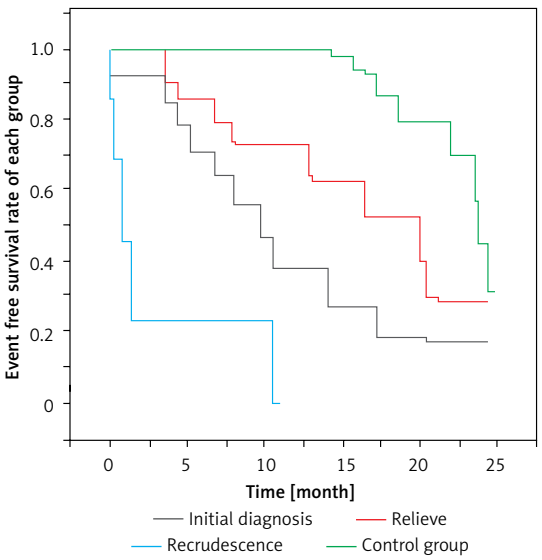


Figure 7. Relationship between SENP1 and OS and DFS in AML

cells, as demonstrated by the difference in the apoptotic rates between the sh-SENP1 and sh-NC groups detected by the Annexin V/PI method. Given the potential role of SENP1 in the occurrence and development of AML, inhibition of SENP1 could become a new treatment strategy. It has been shown that SENP1 inhibitors can suppress the proliferation and induce apoptosis of AML cells [26]. Therefore, further studies should investigate the potential of SENP1 inhibitors as therapeutic agents for AML and evaluate their safety and efficacy in the clinic.

Secondly, the relationship between SENP1 expression and clinical characteristics of AML patients was further explored. It was found that high SENP1 expression was associated with poor prognostic indicators, including lower OS and DFS. This suggests that the high expression of SENP1 may be closely related to the poor prognosis of AML patients, suggesting that SENP1 may serve as a potential prognostic marker. This finding holds potential clinical translational value. From a prognostic assessment perspective, measuring SENP1 expression levels in bone marrow samples may serve as an independent prognostic indicator: patients with high SENP1 expression at initial diagnosis may require intensified induction chemotherapy regimens, while SENP1 expression levels in relapsed patients could be utilized to monitor disease progression. In terms of therapeutic strategy, as a deSUMOylating enzyme, SENP1 represents a promising target for AML treatment. The development of SENP1 inhibitors may offer a novel targeted therapy approach by specifically suppressing SENP1 activity to arrest the AML cell cycle and induce apoptosis, particularly in relapsed or refractory cases. Notably, the inhibitory effect of SENP1 silencing on U937 cell proliferation observed in this study provides preliminary evidence supporting further investigation of SENP1 as a therapeutic target.

In contrast to other experiments [27–29], this study aimed to explore SENP1 expression and significance in AML patients. Although there have been many studies on SENP1 in other tumors, the role and significance of SENP1 in AML have not been fully explored. Therefore, this study provides preliminary evidence regarding the role of SENP1 in AML and may contribute to a better understanding of its potential biological significance. This study also explored the relationship between SENP1 expression and the clinical characteristics of patients with AML. By analyzing the association between high SENP1 expression and poor prognostic indicators, such as lower OS and DFS, this study provides preliminary evidence for the potential value of SENP1 as a prognostic marker. However, there are some limitations, including the relatively small sample size. The limited number of eligible patients may have reduced the statistical power and affected the precision of the results. Larger, multicenter studies are needed to increase the sample size and further validate these findings.

This study mainly relied on tissue samples for assessing SENP1 expression and lacked analysis of other biological specimens. SENP1 expressions in blood samples may better reflect the overall disease status and progression of AML. Therefore, future studies could use blood samples as research subjects to further validate and extend these results. In addition, the mechanisms underlying SENP1 expression and function were not investigated in detail in this study. Understanding the regulatory mechanism of SENP1 may help better understand its role and significance in AML. Exploring the upstream regulators and downstream targets of SENP1, as well as possible signaling pathways and molecular mechanisms, may help reveal the functional mechanism of SENP1 in AML. Although this article discussed the significance of SENP1 in AML and potential therapeutic targets, more experimental and clinical research are needed to verify the results. Especially for the clinical application of SENP1 inhibitors, more in-depth studies are needed, including the evaluation of drug safety, tolerability, and efficacy. Current research on SENP1 in hematologic malignancies has largely focused on acute lymphoblastic leukemia (ALL) and multiple myeloma (MM), with relatively limited exploration of its function in AML. Studies have shown that in acute B-lymphoblastic leukemia cells, SENP1 promotes cell proliferation by activating the c-Myc signaling pathway, and its high expression is associated with chemotherapy resistance. In multiple myeloma, SENP1 regulates ribosomal protein function through deSUMOylation, thereby influencing tumor cell invasiveness [30–32]. In this research, high SENP1 expression was associated with disease relapse and poor prognosis in patients with AML. Furthermore, SENP1 silencing was associated with reduced AML cell proliferation, an increased proportion of cells in the G1 phase, and increased apoptosis. This finding broadens the evidence regarding the role of SENP1 in hematologic malignancies and provides new insights for AML prognostic evaluation and targeted therapy. It is important to note that the mechanism of SENP1 in different types of hematologic cancers may differ, suggesting that its function could be tumor-type specific, which warrants further validation in future studies.

This study indicated that high SENP1 expression is associated with disease relapse and poor prognosis in AML patients, suggesting potential clinical relevance. From a prognostic assessment perspective, measuring SENP1 expression lev-

els in bone marrow samples may have potential prognostic value, but whether SENP1 is an independent prognostic indicator requires validation in larger, appropriately adjusted studies. In terms of therapeutic strategies, as a deSUMOylating enzyme, SENP1 may represent a potential target for therapeutic development in AML. Inhibiting SENP1 activity may reduce AML cell proliferation and increase apoptosis, although this requires validation in more clinically relevant models. It is noteworthy that the inhibitory effect of SENP1 silencing on U937 cell proliferation in this study provides preliminary evidence for further preclinical investigation. Future research should focus on *in vivo* studies of SENP1 inhibitors, such as AML mouse models, to evaluate their antitumor activity and safety. Additionally, exploring the relationship between SENP1 expression levels and drug sensitivity could provide insights into potential treatment strategies. Combination strategies involving chemotherapy and SENP1 inhibitors could also be investigated in preclinical studies, particularly in patients with high SENP1 expression. Furthermore, the combined detection of SENP1 with other prognostic markers, such as NPM1 mutations and FLT3-ITD, could be explored as a potential approach to improve AML prognostic stratification and support personalized treatment strategies.

In conclusion, the results suggested that in AML patients, high SENP1 expression was associated with clinicopathological features, and SENP1 knockdown was associated with reduced AML cell proliferation and increased apoptosis. Therefore, SENP1 may represent a potential biomarker for AML and a potential therapeutic target. Further studies are needed to explore mechanisms underlying the role of SENP1 in AML and to evaluate SENP1-targeted therapies and their potential to improve patient outcomes.

This study has several limitations. First, the sample size is relatively small. he study included only 32 newly diagnosed patients and 8 relapsed patients, which may have limited the statistical power and precision of the analyses. Future studies should aim to increase the sample size or conduct multi-center collaborative research to further validate the results. Second, the types of biological specimens used were relatively limited. The study only analyzed SENP1 expression in bone marrow samples, without validation in other biological specimens such as blood. Blood samples could provide additional information on the overall disease status. Third, the mechanistic investigation is not sufficiently in-depth. The study only observed the effects of SENP1 knockdown on cell proliferation and apoptosis, without exploring its upstream regulatory factors and downstream signaling pathways, such as the SENP1/c-Myc signaling axis. Future research should incorporate additional molecular biology techniques to further elucidate the mechanisms underlying the role of SENP1 in AML.

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Ethical approval

This study was approved by the Ethics Committee of Xianyang Central Hospital (Approval No. 2022-IRB-63) and was conducted in accordance with the principles of the Declaration of Helsinki.

Conflict of interest

The authors declare no conflict of interest.

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