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ORIGINAL ARTICLE

Genetic Mutations in Patients with Acute Myeloid Leukemia and Their Impact on Prognosis

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ABSTRACT

Background: **This study aimed to analyze the mutant gene profile of patients with acute myeloid leukemia (AML) and discuss the value of genetic mutations in disease prognosis stratification.**
Methods: **Clinical data of patients newly diagnosed with AML (excluding acute promyelocytic leukemia, APL), who were admitted to the Department of Hematology of our hospital from October 2018 through October 2023, were analyzed. Bone marrow fluid was collected from patients, and RNA and DNA were extracted. Leukemia fusion genes and myeloid-related gene mutation panels were used for detection. The results of genetic mutations, patients in different age groups and different karyotype groups, were analyzed and compared.**
Results: **A total of 190 newly diagnosed AML patients were included, with a male-to-female ratio of 1:1.06 and a median age of 56 years (range: 8 - 91 years). A total of 357 genetic mutations were detected in 157 patients, among whom 99 (52.1%) had two or more genetic mutations. Genetic mutations (mutation frequency ≥ 10%) were NPM1 (42 cases, 27.45%), FLT3::ITD (34 cases, 22.22%), CEBPA (31 cases, 20.26%), DNMT3A (30 cases, 19.6%), NRAS (24 cases, 15.69%), ASXL1 (23 cases, 15.03%), IDH2 (20 cases, 13.07%), and RUNX1 (17 cases, 11.11%). The most common mutations in males were RUNX1 and ASXL1, in females, it was NPM1. The common mutations in the ≤ 64 years group were NPM1, FLT3::ITD, CEBPA, DNMT3A, NRAS, ASXL1, IDH2, RUNX1, TET2, PTPN11, FLT3, IDH1, TP53, FLT3::TKD, and SRSF2, and in the > 64 years group, it was KIT. According to the cytogenetic prognostic risk stratification, there were 84 patients (44.2%) in the low-risk karyotype group (with a high incidence of CEBPA double mutations), 56 patients (29.5%) in the intermediate-risk karyotype group (with high incidences of NPM1, CEBPA double mutations, FLT3::ITD, TET2, DNMT3A, IDH2, NRAS, IDH1, and PTPN11), and 50 patients (26.3%) in the high-risk karyotype group (with high incidences of RUNX1, ASXL1, NRAS, FLT3::ITD, TP53, CEBPA, DNMT3A, and NPM1). In total, there were 157 cases (82.63%) with genetic mutations (OS: 14.03 months) and 33 cases (17.37%) with no genetic mutations (OS: 17.73 months). There was no statistically significant difference between two groups (p > 0.05).**
Conclusions: **The incidence of genetic mutations varies among AML patients in different age groups and karyotype groups.**
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KEYWORDS

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INTRODUCTION

Acute myeloid leukemia (AML) is a disease caused by uncontrolled proliferation of clonal hematopoietic cells [1], characterized by infiltration of abnormal hematopoietic progenitor cells into the bone marrow, and its uncontrolled proliferation and impaired differentiation enhance the survival advantage of leukemia cells, thereby affecting normal hematopoiesis [2]. Currently, the clinical risk stratification system (for diagnosis and treatment) of AML mainly relies on the 2022 ELN risk stratification [3], which classifies patients into low-risk, intermediate-risk, and high-risk groups based on their cytogenetic characteristics. However, in clinical practice, it has been found that patients in the same risk stratification vary in disease biological characteristics, response to treatment, and disease outcome. With the development of genetic testing technology, scholars have studied the mechanism of acute leukemia-related genetic mutations in disease occurrence and development and identified some specific types of genetic mutations or fusion genes to guide clinical treatment plans [4].

This study used gene sequencing technology to detect genetic mutations in newly diagnosed AML patients (excluding APL). It aimed to understand the distribution of AML-related genetic mutations in patients, analyze their correlation with age at onset, gender, white blood cell count, platelet count, hemoglobin count, chromosome karyotype, and genetic abnormalities, and discuss the value of genetic mutations in disease diagnosis and prognosis stratification.

MATERIALS AND METHODS

Study subjects

This was a retrospective study. Clinical data of patients newly diagnosed with AML (excluding APL), who were admitted to the Department of Hematology, Affiliated Hospital of Chengde Medical College, from October 2018 through October 2023, were collected. All patients underwent routine blood tests, bone marrow cytomorphology, cellular immunophenotyping, chromosome karyotype analysis, fusion gene detection, and myeloid-related genetic mutation detection, and met Acute myeloid leukemia: 2023 update on diagnosis, risk-stratification, and management [5].

Detection Methods for Bone Marrow Samples

A total of 4 - 6 mL of bone marrow fluid was collected from patients, anticoagulated with EDTA tubes for RNA and DNA detection. RNA was used for real-time quantitative PCR to screen for AML fusion genes such as AML::ETO, BCR::ABL, CBFβ::MYH11, MLL::PTD, and NUP98::NSD1. DNA was used for next-generation sequencing (NGS) detection of AML-related genetic mutations, covering hot mutation regions of genes such as ASXL1, FLT3, NPM1, KIT, CEBPA, DNMT-3A, IDH, TET2, EZH2, RUNX1, PHF6, TP53, SF3B1, SRSF2, U2AF1, RAS, and JAK.

Treatment methods

Patients ≤ 60 years old received induction chemotherapy based on the DA3+7 regimen (demethoxydaunorubicin 8 - 12 mg/m² × 3 days + cytarabine 100 mg/m² × 7 days); patients aged > 60 but ≤ 70 years received induction chemotherapy with the DA3+7 regimen or priming regimens such as CAG (aclarubicin 10 - 14 mg/m² × 4 days + cytarabine 10 mg/m², twice daily × 14 days + colony-stimulating factor 300 μg/day); patients > 70 years old received induction chemotherapy with regimen A (cytarabine 100 mg/m² × 7 days).

AML patients who achieved complete remission (CR) after induction chemotherapy entered the consolidation treatment stage, receiving 4 courses of medium- to high-dose cytarabine monotherapy. Patients with transplantation conditions, suitable donors, and classified as high-risk based on cytogenetic typing combined with molecular biological markers underwent allogeneic hematopoietic stem cell transplantation.

Follow-up observation

Overall survival (OS) was defined as the time from diagnosis to patient death or the end of follow-up (January 31, 2024).

Statistical methods

SPSS software was used for data analysis. Count data were expressed as cases (%), and comparisons were performed using the χ^2 test. A p-value < 0.05 was considered statistically significant.

RESULTS

General information

The study consists of a total of 190 newly-diagnosed AML (non-APL) patients, including 92 males (48%) and 98 females (52%), with a male-to-female ratio of 1:1.06. The median age of patients was 56 years (range: 8 - 91 years), with 63% of patients ≤ 60 years old and 37% > 60 years old. According to the WHO (2022 edition) classification criteria for myeloid leukemia, among the 190 enrolled patients, 84 patients had AML with recurrent genetic abnormalities, including 16 cases (8.42%) with t(8;21)/RUNX1::RUNX1T1, 2 cases (1.05%) with inv(16)/CBFβ::MYH11, 2 cases (1.05%) with BCR::ABL1, 39 cases (20.53%) with NPM1 mutation, and 25 cases (13.16%) with CEBPA biallelic mutation, 106 patients diagnosed AML not otherwise specified (AML NOS). Patients were grouped according to cytogenetic prognostic risk stratification in the Chinese Guidelines for the Diagnosis and Treatment of Adult Acute Myeloid Leukemia (non-acute promyelocytic leukemia) (2022 edition) [6]: 84 cases (44.21%) were in the favorable prognosis group, 56 cases (29.47%) were in the intermediate prognosis group, and 50 cases

Table 1. The frequency of genetic mutations in 190 patients with AML in different karyotype groups.

Group (n = 190)	Gene mutation (n, %)
Low-risk karyotype group (n = 84)	*RUNX1*::*RUNX1T1*(13, 15.47%), CEBPA double mutation (12, 14.28%), no mutation (33, 39.28%)
Intermediate-risk karyotype group (n = 56)	NPM1 (26, 46.42%), CEBPA double mutation (8, 14.28%), FLT3::ITD (22, 39.28%), TET2 (7, 12.69%), DNMT3A (17 cases, 30.35%), IDH2 (6, 10.71%), NRAS (6, 10.71%), IDH1 (6, 10.71%), and PTPN11 (8, 14.28%)
High-risk karyotype group (n = 50)	RUNX1 (11, 22%), ASXL1 (15, 30%), NRAS (6, 12%), FLT3::ITD (10, 20%), TP53 (9, 18%), CEBPA (5, 10%), DNMT3A (6, 12%), and NPM1 (5, 10%)

Table 2. Response rate of patients with acute myeloid leukemia with different mutant genes to induction chemotherapy.

Genetic mutation	Total cases	Complete remission (%)	Partial remission (%)
NPM1	39	71.8	28.2
FLT3::ITD	34	52.9	38.3
CEBPA	25	76	20
DNMT3A	25	60	40
ASXL1	16	62.5	37.5
PTPN11	13	84.6	15.4

(26.31%) were in the high-risk karyotype group.

Incidence of genetic mutations

Among the 190 patients, 33 had no genetic mutations, while 157 patients had a total of 357 genetic mutations, among whom 99 (52.1%) had two or more genetic mutations. Genetic mutations with a mutation frequency ≥ 10% were NPM1 (42 cases, 27.45%), FLT3::ITD (34 cases, 22.22%), CEBPA (31 cases, 20.26%), DNMT3A (30 cases, 19.6%), NRAS (24 cases, 15.69%), ASXL1 (23 cases, 15.03%), IDH2 (20 cases, 13.07%), and RUNX1 (17 cases, 11.11%). Other common genetic mutations include PTPN11 (14 cases, 9.15%), TP53 (9 cases, 5.88%), FLT3 (14 cases, 9.15%), FLT3::TKD (7 cases, 5.88%), TET2 (15 cases, 9.8%), SRSF2 (9 cases, 5.8%), IDH1 (11 cases, 7.19%), and KIT (10 cases, 7.69%).

Further, 29.1% (99/190) patients had two or more mutations. Among them, DNMT3A and NPM1 as well as FLT3::ITD and NPM1 were the most common concurrent mutations, followed by DNMT3A and FLT3::ITD. In 42 patients with NPM1 mutation, 18 cases (42.86%) had concurrent FLT3::ITD mutation, 18 cases (42.86%) had concurrent DNMT3A mutation, 8 cases (19.05%) had concurrent PTPN11 mutation, 6 cases (14.29%) had concurrent TET2 mutation, and 6 cases (14.29%) had concurrent IDH1 mutation. In 34 patients with FLT3::ITD mutation, 10 cases (29.50%) had concurrent DNMT3A mutation. For DNMT3A mutation, the most common coexisting mutations were FLT3-ITD, NPM1, IDH2, TET2, CEBPA, and PTPN11. The most frequently detected NPM1 mutation did not co-express with U2AF1, JAK2, CBFB, CSF3R, PHF6, SH2B3, CBL, EZH2, or ZRSR2 in the same patient (Figure 1).

The Circos plot depicts the incidence and co-expression of mutant genes and fusion genes in 190 newly treated AML patients in this study. The length of the arc corresponds to the mutation frequency of the gene, and the width of the band corresponds to the mutation frequency of patients with concurrent co-expressed genetic mutations.

Relationship between incidence of genetic mutations and patient gender

The most common mutations in males were RUNX1 and ASXL1, while in females, it was NPM1, with a statistically significant difference ($p < 0.05$).

Relationship between incidence of genetic mutations and patient age

X-tile software was used to determine that the optimal cutoff age for overall survival rate was 64.0 years. The incidence of mutant genes in the age > 64 years group was different from that in the age ≤ 64 years. The common mutations in the ≤ 64 years group were NPM1 (42 cases, 27.45%), FLT3::ITD (34 cases, 22.22%), CEBPA (31 cases, 20.26%), DNMT3A (30 cases, 19.6%), NRAS (24 cases, 15.69%), ASXL1 (23 cases, 15.03%),

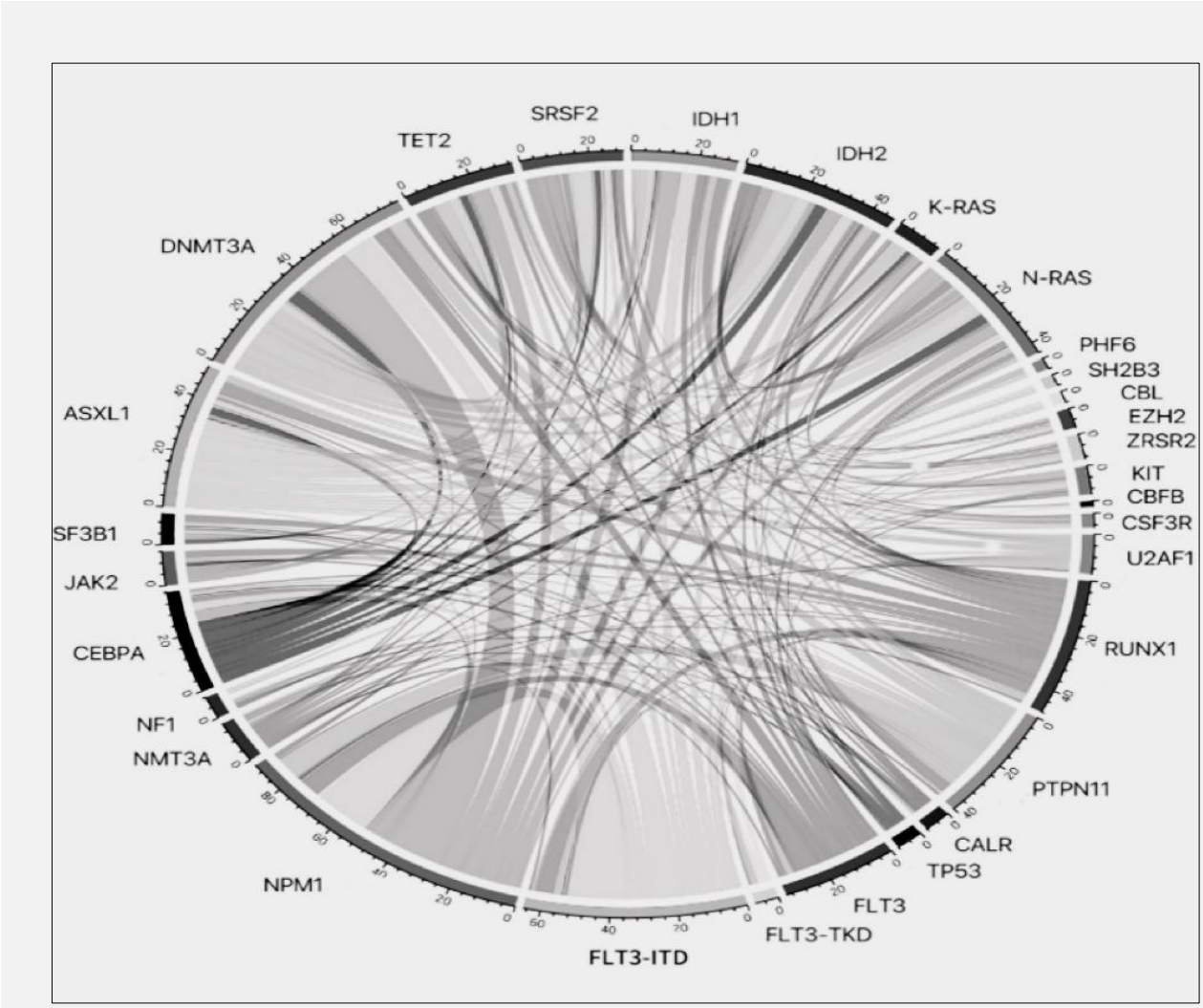

Figure 1. Circos plot of mutated genes in 190 AML patients.

IDH2 (20 cases, 13.07%), RUNX1 (17 cases, 11.11%), TET2 (15 cases, 9.8%), PTPN11 (14 cases, 9.15%), FLT3 (14 cases, 9.15%), IDH1 (11 cases, 7.19%), TP53 (9 cases, 5.88%), FLT3::TKD (7 cases, 5.88%), and SRSF2 (9 cases, 5.8%). The common mutation in the > 64 years group was KIT (10 cases, 7.69%) (Figure 2).

Relationship between incidence of genetic mutations and initial white blood cell count, platelet count, and hemoglobin level

X-tile software was used to determine the optimal cutoff values of initial white blood cell count, hemoglobin level, and platelet count for overall survival rate as 26.7 × 10^9/L, 88 g/L, and 41 × 10^9/L, respectively, based on which patients were grouped independently. The most common mutations in the WBC < 26.7 × 10^9/L group were U2AF1 and TP53, with statistically significant difference ($p < 0.05$). The more common mutations in the WBC ≥ 26.7 × 10^9/L group were FLT3::ITD, NPM1, and CBFB, with statistically significant difference ($p < 0.05$). No significant correlation was found between other genetic mutations and initial white blood cell count.

The most common mutations in the initial HB < 88 g/L group were CEBPA and KIT, with statistically significant difference ($p < 0.05$). No significant correlation was found between other genetic mutations and initial HB level.

The most common mutation in the initial PLT < 41 × 10^9/L group was KIT, with statistically significant difference ($p < 0.05$). The most common mutations in the initial PLT ≥ 41 × 10^9/L group were CALR, NPM1, NMT3A, JAK2, DNMT3A, IDH2, and CSF3R, with statistically significant difference ($p < 0.05$). No signifi-

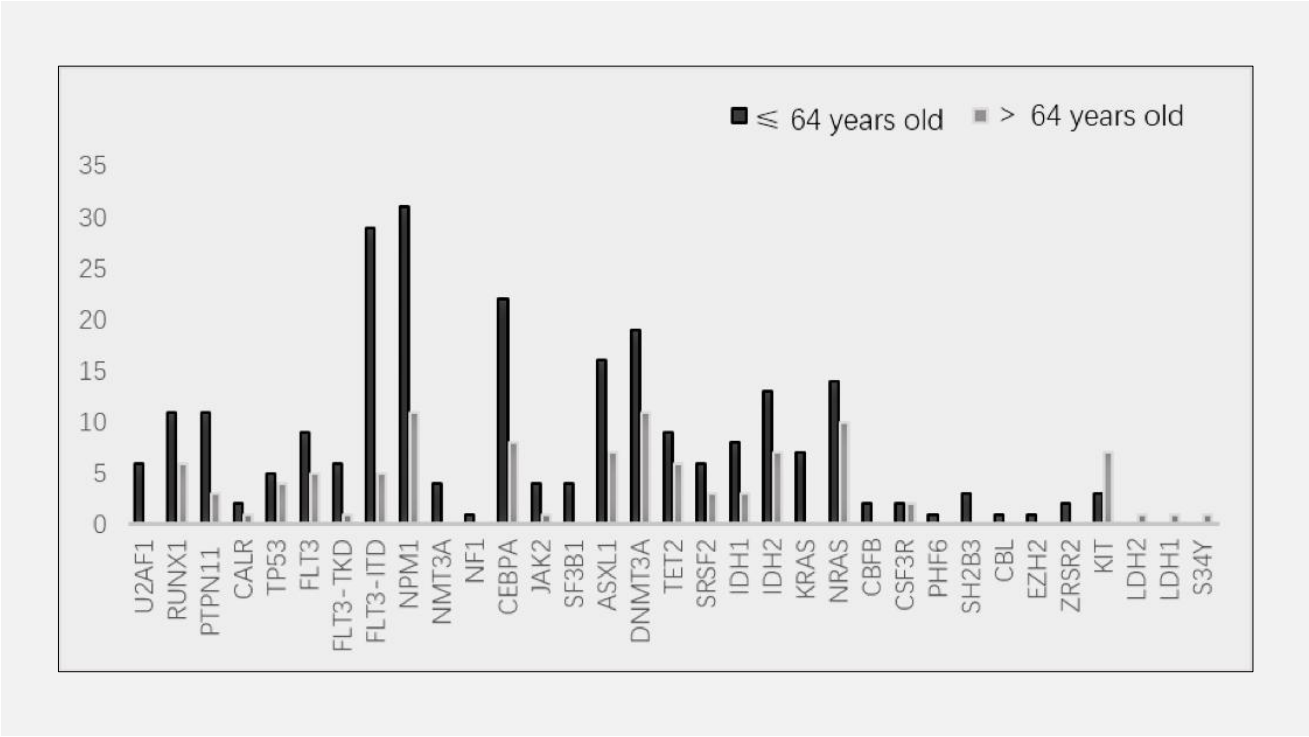

Figure 2. Distribution of genetic mutations in patients with acute myeloid leukemia in different age groups.

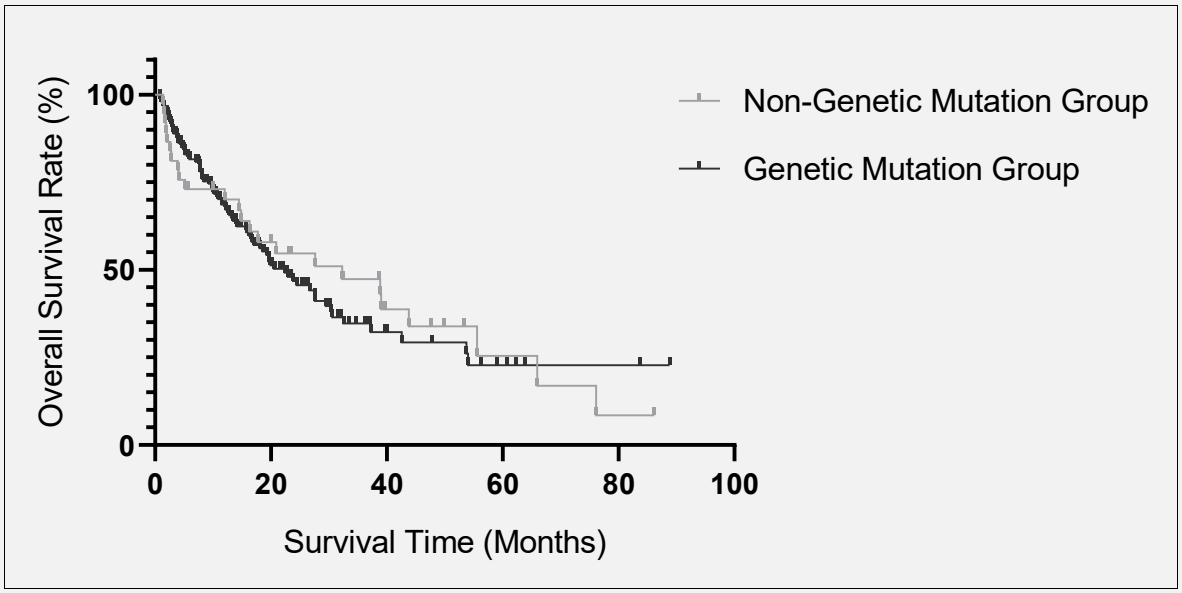

Figure 3. The total survival curve of patients with acute myeloid leukemia in the genetic mutation group and the non-genetic mutation group.

cant correlation was found between other genetic mutations and initial platelet count.

Relationship between genetic mutations and chromosome karyotype

Analysis was performed according to chromosome karyotype groups and genetic mutations with a mutation frequency ≥ 10% (Table 1).

Impact of genetic mutations on induction chemotherapy response rate

Among 190 AML patients, 33 cases (17.37%) had no genetic mutations involved in this study, and 157 cases (82.63%) had 357 mutant genes. Further, 124 cases (65.26%) achieved CR after 1 - 3 courses of induction chemotherapy, 1 case achieved CR after 4 courses of chemotherapy, 6 cases (3.16%) had no response (NR) after ≥ 4 courses of chemotherapy, 6 cases did not receive chemotherapy, 33 cases received only one course of chemotherapy and did not receive further hospitalization, 27 cases completed maintenance chemotherapy, and 11 cases died within 60 days after chemotherapy (Table 2).

Impact of genetic mutations on OS

Follow-up was conducted for 190 AML patients, with a median OS of 36.4 months. In total, 157 cases (157/190, 82.63%) had genetic mutations, with a median OS of 14.03 months; 33 cases (33/190, 17.37%) had no genetic mutations, with a median survival time of 17.73 months, which was better than that of the mutation group (14.03 months), with no statistically significant difference ($p > 0.05$) (Figure 3).

DISCUSSION

Over the past few decades, significant progress has been made in the treatment of AML. Our in-depth understanding of the genetic and molecular mechanisms of this disease is crucial for the development of new therapies and the integration of new biomarkers into routine patient assessment [7]. Currently, in clinical practice, the European Leukemia Net (ELN) genetic risk classification is a reference document in hematology, widely used in clinical practice or clinical trials [3,8]. However, some scholars have pointed out the limitations of this classification, as it is only based on data from AML patients receiving intensive chemotherapy and cannot be applied to unfit/frail and/or elderly patients receiving less intensive treatment, especially those in the poor-risk category [9]. Based on these considerations, some scholars believe that a new genetic classification is needed for accurate prognosis stratification of patients receiving less intensive treatment [10]. With the continuous discovery of the molecular genetic pathogenesis of AML, combined with refined treatment paths adapted to age, genetic risk stratification, and body tolerance, the treatment of AML patients has become more standardized and refined, and the efficacy and prognosis of AML patients have also been improved. In the past decade, there has been limited large-sample research data on exploring the genetic mutation profile of AML patients using NGS technology, and most reports with large sample sizes are from European multi-center data. However, in recent years, more and more data from major medical centers in China have been reported.

The results of this study showed that the detection rates of RUNX1 and ASXL1 mutations were higher in male patients than in female patients, while the detection rate of NPM1 mutation was higher in female patients than in male patients, with statistically significant differences. The distribution of detected mutations varied significantly by age: the genetic mutation profiles of people under 64 years old and those aged 64 years and above were significantly different, with statistically significant differences. TP53 mutation was more common in people aged 64 years and above, while DNMT3A, FLT3::ITD, and CEBPA mutations were more common in young patients. Among initial blood parameters, white blood cell count had the most significant impact on genetic mutations: the mutation frequencies of FLT3-ITD, NPM1, and CBFB in the initial WBC ≥ 26.7 × 10⁹/L group were significantly higher than those in the initial WBC < 26.7 × 10⁹/L group, with statistically significant differences; the mutation frequencies of U2AF1 and TP53 in the initial WBC < 26.7 × 10⁹/L group were significantly higher than those in the initial WBC ≥ 26.7 × 10⁹/L group, with statistically significant differences. Hemoglobin level had a greater impact on CEBPA and KIT mutations: the mutation frequencies of CEBPA and KIT in the HB < 88 g/L group were significantly higher than those in the HB > 88 g/L group, with statistically significant differences. Platelet count had a significant impact on genetic mutations: the mutation frequency of KIT in the initial PLT < 41 × 10⁹/L group was significantly higher than that in the initial PLT > 41 × 10⁹/L group, with a statistically significant difference; the mutation frequencies of CSF3R, IDH2, CALR, NPM1, NMT3A, JAK2, and DNMT3A in the initial PLT ≥ 41 × 10⁹/L group were significantly higher than those in the initial PLT < 41 × 10⁹/L group, with statistically significant differences. FAB classification was significantly associated with the mutation frequencies of FLT3::ITD, NPM1, CEBPA, ASXL1, DNMT3A, IDH2, and NRAS (≥ 10.0%): FLT3::ITD, NPM1, and CEBPA were more common in M1; ASXL1 and NRAS were more common in M4; DNMT3A was more common in M5; and IDH2 was more common in M0.

In terms of genetic mutation results, the genetic mutations with an incidence rate of over 10% in this study were RUNX1, FLT3::ITD, NPM1, CEBPA, ASXL1, DNMT3A, IDH2, and NRAS. The incidence rates of most genes were basically consistent with previous domestic reports. TP53 mutation is more common in European and American AML patients, while the proportion of TP53 mutation in domestic patients is not high. Current studies believe that this mutation is related to

complex karyotypes and is an independent poor prognostic factor [11]. In this study, AML patients with TP53 mutation accounted for 5.88%; the proportion was 2.6% in 7 general hospitals in Shanghai [4], 4.3% in the First Clinical College of Xinjiang Medical University, 8% in the American population [12], and 13% in the European population [13]. Compared with domestic medical center data, the frequency of TP53 mutation in this study was lower than that in foreign results. Meanwhile, TP53 mutation in this study was associated with initial age ≥ 64 years. The proportion of AML patients with NPM1 mutation in this study was 27.45%, which was higher than that in other domestic centers, similar to 28% in the European population and 27% in the American population. Meanwhile, NPM1 mutation in this study was associated with high white blood cell count at diagnosis. The proportion of patients with DNMT3A mutation in this study was 19.6%, which was higher than that in other domestic centers (12.3% in Ruijin Hospital and the First Affiliated Hospital of Zhejiang University [14,15]), and lower than the multicenter data of 26% in the United States. DNMT3A mutation was associated with initial PLT ≥ 41 × 10^9/L and initial age < 64 years. The incidence of CEBPA double mutation in this study was 20.26%, compared with 9.49% in 7 general hospitals in Shanghai, 20% in the First Affiliated Hospital of Soochow University, 5.69% in the First Clinical College of Xinjiang Medical University, and 15.84% in the Affiliated Hospital of Hainan Medical University. The domestic research results are basically consistent, but the proportion is much higher than 4% in Europe and 6% in the United States. CEBPA mutation in this study was associated with initial age < 64 years. This study also found that the incidence of ASXL1 genetic mutation was 15.03%, which was lower than 60% in the First Affiliated Hospital of Nanjing Medical University and Jiangsu Provincial People's Hospital, while the incidence of genetic mutations such as CBFB, CSF3R, PHF6, SH2B3, CBL, NF1, EZH2, and ZRSR2 was low. The reasons for the abovementioned differences are not clear, which may be due to two factors: 1) regional differences, including ethnic minorities (the region of this study has Manchu ethnic minorities); 2) sample size, which requires more data support.

This study also analyzed the incidence of AML-related genetic mutations among different groups according to different chromosome karyotypes and found that there were differences in mutant genes among patients in different karyotype groups. There were 84 patients (44.2%) in the low-risk karyotype group, 56 patients (29.5%) in the intermediate-risk karyotype group, and 50 patients (26.3%) in the high-risk karyotype group. There were certain differences compared with the data reported by 7 general hospitals in Shanghai and the First Clinical College of Xinjiang Medical University. These differences may be due to regional differences and sample size, which require more data support.

There are some coexistence or mutual exclusion phenomena between mutant genes, which may be related to different biological functions. The results of this study showed that 63.06% of AML patients had two or more mutant genes, among whom 38.21% (60/157) had three or more mutations. The most common co-expressed genes were NPM1 and FLT3::ITD, NPM1 and DNMT3A, and DNMT3A and FLT3::ITD, and there was also an overlapping synergistic relationship among the three genes. A study showed that the most common coexisting mutant genes were ASXL1 and RUNX1, TET2, or NRAS [16]; while another study found that RUNX1, SRSF2, ASXL1, STAG2, and BCOR were often co-expressed in the same patient, and strong co-expression was also found between IDH2 and SRSF2, RUNX1 and SF3B1, and double CEBPA and GATA2 mutations. In addition, TP53 may be mutually exclusive with IDH2, NRAS, NPM1, and SRSF2; IDH2 may be mutually exclusive with TET2 and TP53. NPM1 and RUNX1 mutations, as well as DNMT3A and SRSF2 mutations, may also be mutually exclusive [17]. A study showed that the most common co-expressed genes were ASXL1 and TET2, DNMT3A, or WT1, while NPM1 mutation and ETO, CEBPA mutation were mutually exclusive, WT1 mutation and RAS family member mutations were mutually exclusive, and DNA methylation-related genes such as IDH1/2 and TET2 mutations were rarely co-expressed in the same AML patient. However, this study showed that TP53 was co-expressed with IDH2, NRAS, NPM1, and RUNX1 mutations, as well as DNMT3A and SRSF2 mutations. This finding can be explained by the biological functions of mutant genes: mutations that occur synergistically often belong to different types of genes, while mutually exclusive mutations exhibit a common mechanism of hematopoietic transformation in their biological functions [18]. Such co-expression and mutual exclusion patterns suggest that various mutations and their combinations may play a role in the pathogenesis of AML, and the patterns and relationships of these mutant genes collectively promote the occurrence of leukemia, which has a certain inherent relationship with the pathogenesis of leukemia.

Follow-up was conducted for 190 AML patients, with a median OS of 36.4 months. All in all, 157 cases (157/190, 82.63%) had genetic mutations, with a median OS of 14.03 months; 33 cases (33/190, 17.37%) had no genetic mutations, with a median survival time of 17.73 months, which was better than that of the mutation group (14.03 months), but the difference was statistically nonsignificant.

The goal of this study was to explore the impact of genetic mutations in AML patients on treatment decisions and patient prognosis. By providing valuable insights, it may help clinicians make informed clinical decisions and improve patient prognosis in AML management.

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Declaration of Interest:
None of the authors have a conflict of interest to disclose.

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