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# ZMYND11::MBTD1-Rearranged Acute Myeloid Leukemia in Chinese Adults: Clinicopathologic Features and Clinical Outcomes

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## Abstract

# Background

The *ZMYND11::MBTD1* fusion, resulting from t(10;17)(p15;q21), is a rare recurrent genetic alteration in acute myeloid leukemia (AML). Its clinical and prognostic features in adult patients remain poorly characterized, particularly in Chinese populations. This study aimed to delineate the clinicopathological, immunophenotypic, and molecular features, as well as treatment responses and survival outcomes, of adult AML harboring this fusion.

# Methods

We retrospectively analyzed five newly diagnosed adult AML patients with *ZMYND11::MBTD1* fusion treated at our institution between 2020 and 2024. These cases were integrated with nine previously reported adult cases for a comprehensive cohort analysis (N = 14). All institutional cases were identified via targeted RNA sequencing with PCR confirmation. Immunophenotyping, cytogenetics, treatment response, and overall survival (OS) were evaluated.

# Results

All five institutional cases exhibited a consistent RAM-like immunophenotype characterized by bright aberrant CD7 and CD56 expression, positivity for CD33 and CD117, dim CD45, and absent or reduced HLA-DR and CD38. Conventional karyotyping detected t(10;17) in only one of five cases. In the institutional cohort, the complete remission (CR) rate after one induction cycle was 40%, with a best CR rate of 80% after sequential therapy. In the integrated cohort (median age 51 years, 57% female), 85.7% of patients were classified as ELN 2022 intermediate-risk. The median OS was 34.0 months, with estimated 1-, 2-, and 3-year OS rates of 83.3%, 60.8%, and 48.6%, respectively. Patients who underwent allogeneic hematopoietic cell transplantation (allo-HCT) showed numerically longer median OS (37.0 vs. 23.0 months) and 3-year OS (80.0% vs. 33.3%), though not statistically significant ($P$ = 0.472).

# Conclusion

Adult AML with *ZMYND11::MBTD1* fusion displays a distinctive RAM-like immunophenotype and an unfavorable clinical course that may not be fully captured by current ELN risk stratification. Recognition of this entity, particularly in cases with bright CD7/CD56 expression, is clinically important. Early consideration of allo-HCT may be warranted in eligible patients, and larger prospective studies are needed to validate these findings and refine risk assessment.

## Background

Acute myeloid leukemia (AML) is a biologically heterogeneous hematologic malignancy increasingly defined by recurrent genomic alterations that inform disease classification, prognostic stratification, and therapeutic management. Recent classification systems, including the 2022 World Health Organization (WHO) classification, the International Consensus Classification (ICC), and the European LeukemiaNet (ELN) recommendations all emphasize the pivotal role of molecular genetics in AML nosology and risk assessment (1–3).

*ZMYND11::MBTD1* fusion, generated by t(10;17)(p15;q21), has emerged as a rare but recurrent genetic alteration in AML (4–10). Current reports suggest that this rearrangement is enriched in poorly differentiated or minimally differentiated AML and is often accompanied by aberrant CD7 and CD56 expression, weak or absent myeloperoxidase (MPO), and absent or reduced HLA-DR expression (5–10). These features overlap with the RAM immunophenotype, a high-risk flow cytometric pattern originally described in pediatric AML (11–13).

Data on adult AML harboring *ZMYND11::MBTD1* rearrangement remain limited, particularly among Chinese patients. We hypothesized that *ZMYND11::MBTD1*-rearranged AML in adults exhibits a recognizable RAM-like immunophenotype and adverse outcomes not fully captured by ELN 2022 criteria. Here, we describe five adult patients from a single Chinese center and integrate them with nine previously reported adult cases. We focused on clinicopathologic, immunophenotypic, cytogenetic and molecular features of this entity, together with treatment responses and survival outcomes.

## Methods

# Patients and study design

This retrospective study included five newly diagnosed adult AML patients harboring *ZMYND11::MBTD1* fusion who were treated at our center between January 2020 and December 2024. AML diagnosis was established according to the 2022 WHO classification. Eligibility criteria for the institutional cohort included: age ≥ 16 years; newly diagnosed AML; receipt of at least one course of therapy with evaluable response; and identification of *ZMYND11::MBTD1* fusion by targeted transcriptome sequencing followed by polymerase chain reaction (PCR) confirmation.

For the contextual cohort, we reviewed 12 previously published AML cases associated with t(10;17)(p15;q21)/*ZMYND11::MBTD1* and included nine adult patients (9). One published adult case lacked follow-up data and was included in baseline analyses but excluded from survival analysis. The integrated cohort therefore comprised 14 adult patients for clinicopathologic analysis and 13 patients for OS analysis.

## Flow cytometry

Fresh heparinized bone marrow aspirates were analyzed by multiparameter flow cytometry. Diagnostic samples were gated on blast populations with low side scatter and dim CD45 expression, with acquisition of ≥ 1 × 10^5 events; ≥ 1 × 10^6 total events were acquired for post-treatment and remission samples. Ten-color immunophenotyping was performed using antibodies against CD34, CD38, CD117, HLA-DR, CD13, CD33, CD14, CD15, CD64, CD11b, CD7, CD56, CD2, CD4, CD19, MPO, TdT, cytoplasmic CD3, cytoplasmic CD79a, and CD45. Data acquisition was performed on a NAVIOS cytometer (Beckman Coulter, Brea, California) and analyzed using KALUZA software (Beckman Coulter, Brea, California) (14).

### Cytogenetic and molecular analyses

Conventional chromosome analysis was performed on heparinized bone marrow aspirates following culture in RPMI 1640 medium, colchicine exposure, hypotonic treatment, fixation, and R-banding. At least 20 metaphases were analyzed when available. Karyotypes were interpreted according to the International System for Human Cytogenomic Nomenclature 2020.

DNA and RNA were extracted from bone marrow specimens. Targeted next-generation sequencing and RNA sequencing were performed according to institutional protocols (15, 16). *ZMYND11::MBTD1* fusion transcripts identified by RNA sequencing were confirmed by PCR in all cases. Concurrent somatic mutations were reported based on clinically available sequencing results.

### Treatment, follow-up, and endpoints

Treatment decisions were individualized according to age, performance status, comorbidities, and physician assessment. Patients considered eligible for intensive chemotherapy received idarubicin plus cytarabine induction followed by high-dose cytarabine consolidation. Patients considered unfit for intensive induction received azacitidine plus venetoclax. Re-induction or salvage regimens were selected according to response, measurable residual disease (MRD), and overall clinical condition. Allogeneic hematopoietic cell transplantation (allo-HCT) was considered after remission based on ELN 2022 risk stratification, MRD, donor availability, and patient fitness.

Follow-up data were obtained from medical records and telephone interviews. The data cutoff was April 30, 2026. Overall survival (OS) was defined as the time from diagnosis to death from any cause or last follow-up.

## Statistical analysis

Continuous variables are presented as medians with interquartile ranges (IQRs), and categorical variables as frequencies and percentages. Continuous variables were compared between institutional and reference cohorts using the Mann-Whitney U test, and categorical variables using Fisher's exact test. OS was estimated using the Kaplan-Meier method and compared using the log-rank test. Given the small sample size, all analyses were exploratory.

### Results

## Baseline features, integrated cohort and risk stratification

The five institutional patients had a median age of 41 years (IQR, 31–61), and three were female. Median white blood cell (WBC) count at diagnosis was 2.83 × $10^9$/L (IQR, 2.50–3.10), hemoglobin level was 6.3 g/dL (IQR, 5.1–7.6), platelet count was 51 × $10^9$//L (IQR, 35–84), and bone marrow (BM) blast percentage was 72.0% (IQR, 68.0-77.5). Morphologic findings were heterogeneous which included FAB M5, M7, and unclassified cases. All five institutional cases harbored *ZMYND11::MBTD1* fusion transcripts detected by RNA sequencing and confirmed by PCR, yet conventional cytogenetics identified t(10;17)(p15;q21) in only one patient, and the remaining 4 cases showed either normal karyotypes or other non-specific abnormalities. Concurrent somatic mutations were identified, including *SMC2*, *ARID1A*, *GATA2*, *NRAS*, *SRSF2*, and *TET2* (Table 1).

Table 1
MICM features, treatment response and outcomes of the five institutional patients.

| Case | Sex/age | FAB/ BM blasts | Immunophenotype | Karyotype | Co-mutations | First-line therapy and response | Sequential therapy / best response | Relapse | Allo-HCT | OS, months | S a f u |
|---|---|---|---|---|---|---|---|---|---|---|---|
| P1 | Male/61y | M5a/ 72% | CD33, CD34, CD117, CD45dim, CD123; partial CD38 (24.6%); aberrant CD7 (98.8%) and CD56 (99.5%) | 42 ~ 44,X,-Y,-21[cp6]/46,XY | SMC2 | IA, NR | HAD, CR | Yes | No | 65.5 | A |
| P2 | Female/31y | Unclassified/ 68% | CD33, CD34, CD117, CD71, CD45dim; aberrant CD7 (99.44%) and CD56 (99.6%) | 46,XX | ARID1A, GATA2 | IA, PR | IA, PR; later proceeded to transplant | No | Yes | 14.6 | A |
| P3 | Female/41y | Unclassified/ 78% | CD33, CD34, CD71, CD117, CD123, CD45dim; partial CD13, HLA-DR, CD36; aberrant CD7 (100.0%) and CD56 (100.0%) | 44 ~ 46,XX,t(10;17)(p15;q21) | NRAS | IA, CR | CR | No | Yes | 36.3 | A |
| P4 | Male/68y | M5/ 60% | CD33, CD34, CD117, CD123, CD45dim; partial CD38, HLA-DR; aberrant CD56 (92.1%) and CD7 (98.3%); low MPO and cCD79a | 44–46,XY,add(10)(p15)[cp4]/46,XY | SRSF2, TET2 | VA, NR | CGA, CR | Yes | No | 23.1 | D |
| P5 | Female/17y | M7/ 95% | CD13, CD15, CD117, CD33, CD36, CD45dim; aberrant CD56 (97.1%) and CD7 (100.0%) | 46,XX | None | IA, CR | CR | No | Yes | 21.4 | A |

WBC, white blood cell count (x$10^9$/L); Hb, hemoglobin (g/dL); PLT, platelet count (x$10^9$/L); BM, bone marrow; FAB, French-American-British classification; IA, idarubicin plus cytarabine; VA, venetoclax plus azacitidine; HAD, homoharringtonine, cytarabine, and daunorubicin; CGA, cladribine, granulocyte colony-stimulating factor, and cytarabine; CR, complete remission; PR, partial remission; NR, non-remission; OS, overall survival; allo-HCT, allogeneic hematopoietic transplantation; NA, not available.

We integrated nine previously reported adult *ZMYND11::MBTD1*-rearranged AML cases from the literature, yielding an integrated cohort of 14 patients Median age was 51 years (IQR, 40–67), and a female predominance (8/14, 57.1%). Median WBC, hemoglobin level, platelet count, and BM blast percentage were 3.35 × 10^9/L (IQR, 2.55–4.07), 8.3 g/dL (IQR, 6.3–9.6), 69.0 × 10^9/L (IQR, 35.5–85.5), and 64.3% (IQR, 57.3–77.1), respectively (Table 2).

Table 2
Baseline characteristics of the institutional, reference, and integrated adult AML cohorts with *ZMYND11::MBTD1* fusion.

| Characteristic | Institutional cohort (n = 5) | Reference cohort (n = 9) | Integrated (n = 14) | P value |
|---|---|---|---|---|
| Age, years | 41 (31–61) | 60 (40–67) | 51 (40–67) | 0.423 |
| Female sex, n (%) | 3 (60.0) | 5 (55.6) | 8 (57.1) | 1.000 |
| WBC, x10^9/L | 2.83 (2.50–3.10) | 3.8 (2.7–4.1) | 3.35 (2.55–4.07) | 0.518 |
| Hemoglobin, g/dL | 6.3 (5.1–7.6) | 9.5 (8.2–9.9) | 8.3 (6.3–9.6) | **0.045*** |
| Platelets, x10^9/L | 51.0 (35.0–84.0) | 77.0 (28.0–81.0) | 69.0 (35.5–85.5) | 1.000 |
| Bone marrow blasts, % | 72.0 (68.0-77.5) | 58.0 (50.0–76.0) | 64.3 (57.3–77.1) | 0.147 |
| ELN 2022 favorable risk | 0/5 | 0/9 | 0/14 (0.0) | 1.000 |
| ELN 2022 intermediate risk | 4/5 | 8/9 | 12/14 (85.7) | 1.000 |
| ELN 2022 adverse risk | 1/5 | 1/9 | 2/14 (14.3) | 1.000 |
| CR after first induction | 2/5 | 7/9 | 9/14 | 0.266 |
| Allo-HCT | 3/5 | 3/9 | 6/14 | 0.592 |

Data are median (IQR), n (%), or n/N unless otherwise indicated. P values compare institutional and reference cohorts. AML, acute myeloid leukemia; WBC, white blood cell count; CR, complete remission; allo-HCT, allogeneic hematopoietic cell transplantation.

By ELN 2022 criteria, 12/14 patients (85.7%) were classified as the intermediate-risk group and 2 as the adverse-risk group. The adverse-risk group comprised one institutional case with *SRSF2* mutation and one published case with complex karyotype.

### Consistent RAM-like immunophenotype in institutional cases

As depicted in Table 1, all institutional cases demonstrated a highly consistent aberrant immunophenotype. Blasts showed strong expression of CD7 and CD56, together with positivity for CD33 and CD117 and dim CD45. CD34 was expressed in four cases, whereas HLA-DR, CD38, and MPO were absent, low, or partially expressed. Overall, the immunophenotype exhibited a modified RAM-like phenotype characterized by additional CD7 expression, which may represent a distinctive feature of *ZMYND11*::*MBTD1*-rearranged AML. The morphology, karyotype and immunophenotype of case P3 were showed in Fig. 1.

## Treatment response

Four institutional patients received standard IA (3 + 7) induction, and one received azacitidine plus venetoclax. After first induction, two patients achieved complete remission (CR), one achieved partial remission, and two were refractory, yielding a CR rate of 40.0% and an overall response rate of 60.0%. After second induction, four of five patients ultimately achieved CR. Three patients underwent allo-HCT and remained alive at last follow-up, whereas both non-transplanted patients experienced hematologic relapse, including one death (Table 1).

In the integrated cohort of 14 adult patients, 10 received standard 3 + 7 induction, with eight achieving CR after the first cycle. Three patients received venetoclax-based induction, with one achieving CR after one cycle. One elderly patient received azacitidine alone and failed to respond. The overall first-cycle CR rate was 64.3% (9/14), and the overall response rate was 71.4% (10/14). Six patients underwent allo-HCT, among whom three had died at last follow-up.

## Long-term survival

In the institutional cohort, median OS was not reached. Estimated 1-, 2-, and 3-year OS rates were 100.0%, 66.7%, and 66.7%, respectively. Eight cases in the reference cohort were evaluable for OS, median OS was 34.0 months, and the 1-, 2-, and 3-year OS rates were 71.4%, 53.6%, and 35.7%, respectively. The OS difference was not statistically significant between institutional and reference cohorts (log-rank $P = 0.107$; Fig. 2A).

Among 13 evaluable patients in the integrated cohort, median OS was 34.0 months. Estimated 1-, 2-, and 3-year OS rates were 83.3%, 60.8%, and 48.6%, respectively (Fig. 2B). Patients who underwent allo-HCT had numerically longer survival than non-transplanted patients, with a median OS of 37.0 versus 23.0 months and estimated 3-year OS rates of 80.0% versus 33.3%, respectively. The transplant comparison was markedly underpowered (log-rank $P = 0.472$, Fig. 2C) and confounded by non-random treatment assignment; these data should be considered exploratory.

### Discussion

This study expands the clinicopathologic spectrum of adult AML with *ZMYND11*::*MBTD1* rearrangement through a retrospective study of a Chinese single-center cohort integrated with previously published adult cases. Several findings showed important diagnostic and prognostic implications. First, institutional cases demonstrated a consistent RAM-like immunophenotype, characterized by bright CD7 and CD56 expression, CD33/CD117 positivity, dim CD45, and absent or reduced MPO, HLA-DR, and CD38. Second, conventional cytogenetics identified t(10;17)(p15;q21) in only one of five institutional cases, indicating the importance of RNA-based fusion testing for reliable detection. Third, survival outcomes appeared unfavorable and relapse was common in the whole cohort, suggesting that *ZMYND11*::*MBTD1* rearrangement may define a rare AML subgroup whose clinical aggressiveness is not fully reflected in current risk stratification systems.

Mechanistically, *ZMYND11*::*MBTD1* fusion provides a mechanistic framework for understanding the distinctive biology of this AML subgroup. ZMYND11 contains reader domains, including PHD, bromodomain, and PWWP modules, that recognize H3.3K36me3-decorated chromatin (17), while MBTD1 is a core subunit of the NuA4/TIP60 histion acetyltransferase (HAT) complex and contributes to hematopoietic stem cell maintenance (18). In experimental studies, the ZMYND11-MBTD1 fusion protein retargets the NuA4/TIP60 complex to the gene bodies of transcriptionally active loci through its PWWP-mediated chromatin tethering, leading to aberrant acetylation of H4 and H2A and consequent dysregulation of genes involved in stemness and differentiation blockade (19, 20). These data support the concept that *ZMYND11*::*MBTD1* is not merely a diagnostic marker but a driver lesion with biologic and clinical relevance. This mode of action — hijacking a chromatin-modifying complex to enforce a leukemogenic transcriptional program — parallels the mechanism described for CBFA2T3::GLIS2, another fusion oncoprotein that drives pediatric AML with a RAM immunophenotype through epigenetic reprogramming (PMID: 23153540).

The classical RAM immunophenotype was first identified in pediatric AML patients from the COG-AAML0531 trial, characterized by the unique combination of bright CD56 expression, dim-to-negative CD45 and CD38, and lack of HLA-DR (11). Our cohort of adult *ZMYND11*::*MBTD1*-rearranged AML demonstrated uniform CD7 expression in addition to CD56, CD33, and CD117 positivity, therefore, we favor the term “RAM-like” rather than RAM phenotype. Notably, both CBFA2T3::GLIS2 and *ZMYND11*::*MBTD1* fusions are associated with a strikingly similar RAM immunophenotypic signature, suggesting that the aberrant epigenetic landscape imposed by these oncogenic fusions may converge on a shared transcriptional program that includes upregulation of the adhesion molecule CD56 and the T-cell marker CD7, and has been associated with poor prognosis (11–13).

RNA-based fusion detection has increasing clinical importance for rare rearrangements. In the institutional cohort, *ZMYND11*::*MBTD1* was present in all cases, yet only one case showed the classic t(10;17)(p15;q21) by cytogenetic analysis. This may reflect technical limitations, cryptic or submicroscopic rearrangements, complex structural variants, or insufficient metaphases. For rare fusion-defined AML subgroups, reliance on conventional karyotyping alone may lead to underdiagnosis (21). Incorporating RNA sequencing or targeted fusion assays into diagnostic workflows may improve detection, especially in cases with a suggestive immunophenotypic signature (22, 23).

CR was achievable in a substantial proportion of patients, including a best CR rate of 80.0% in the institutional cohort after two cycles of induction. However, relapse occurred in both non-transplanted institutional patients, whereas all transplanted cases remained alive at last follow-up. In the integrated cohort, allo-HCT was associated with numerically better 3-year OS, but the difference was not statistically significant. While the small sample precludes definitive conclusions, the pattern of universal relapse in non-transplanted patients and sustained remission in transplanted patients supports consideration of allo-HCT in CR1 for eligible patients. Meanwhile, larger multicenter datasets with standardized MRD assessment are needed to determine whether transplantation can overcome the adverse biology of this subgroup.

The long-term survival data further suggest that *ZMYND11::MBTD1*-rearranged AML may carry prognostic implications beyond current ELN 2022 classification. In the integrated cohort, 85.7% of patients were assigned to the intermediate-risk group, yet median OS was only 34.0 months, and the estimated 3-year OS rate was below 50%. The intermediate-risk classification was driven largely by the absence of recognized adverse-risk features rather than by the presence of favorable ones, which raises the question of whether current criteria adequately capture the biological risk of this rare rearrangement. Although the findings in this small cohort are insufficient to support immediate revision, they raise the possibility that *ZMYND11::MBTD1* rearrangement may identify a subset of nominally intermediate-risk AML whose true clinical behavior aligns with an adverse-risk profile.

This study has some limitations. Because of the rarity of the disease and the small sample size, all analyses were exploratory and descriptive. The integrated cohort analysis was based on published case-level data rather than a systematic review with individual patient datasets, and treatment regimens, transplant eligibility, MRD monitoring, and follow-up duration were heterogeneous across the study. In addition, the limited number of death events within the institutional cohort restricted statistical power for survival analyses. Nevertheless, the consistency of the immunophenotypic profile and the signals of unfavorable prognosis support further prospective investigation.

In summary, adult AML with *ZMYND11::MBTD1* rearrangement appears to represent a rare but clinically recognizable molecular subgroup characterized by a distinctive CD7/CD56-bright RAM-like immunophenotype, frequent ELN 2022 intermediate-risk assignment, and unfavorable clinical outcomes. Recognition of this entity has important diagnostic and potential therapeutic implications and may justify early consideration of allo-HCT in eligible patients. Larger multicenter studies integrating genomic, measurable residual disease and standardized treatment will be required to clarify its biologic and prognostic significance and to determine whether this lesion warrants incorporation into future AML classification and risk-stratification systems.

## Conclusions

This study provides the first systematic characterization of adult *ZMYND11::MBTD1*-rearranged AML in a Chinese cohort. We identify a consistent bright CD7/CD56-positive RAM-like immunophenotype as a readily recognizable diagnostic clue for this rare fusion. Importantly, despite predominant ELN 2022 intermediate-risk classification, the observed survival outcomes suggest an adverse prognosis not adequately captured by current risk stratification, highlighting the need to reconsider this fusion in future prognostic models. The relapse pattern and numerical survival benefit associated with allo-HCT support early transplantation consideration in eligible patients. These findings underscore the clinical importance of recognizing this entity and call for larger prospective studies to refine risk assessment and therapeutic strategies.

## Abbreviations

AML
Acute myeloid leukemia
allo-HCT
Allogeneic hematopoietic cell transplantation
BM
Bone marrow
CR
Complete remission
ELN
European LeukemiaNet
FAB
French-American-British classification
HAT
Histone acetyltransferase
Hb
Hemoglobin
IA
Idarubicin plus cytarabine
ICC
International Consensus Classification
IQR
Interquartile range
MRD
Measurable residual disease
MPO

Myeloperoxidase
NR
Non-remission
OS
Overall survival
PCR
Polymerase chain reaction
PLT
Platelet count
PR
Partial remission
RAM
$CD56^+$/CD45□□□/CD38□□□/$HLA\text{-}DR^-$ immunophenotype
VA
Venetoclax plus azacytidine
WBC
White blood cell count
WHO
World Health Organization

## Declarations

**Ethics approval and consent to participate**

This study was approved by the Ruijin Hospital Ethics Committee of Shanghai JiaoTong University School of Medicine (2026 – 201). Due to the retrospective design of the study and the use of anonymized data, the requirement for informed consent was waived.

**Consent for publication**

Not applicable, as the study used anonymized retrospective data and did not include identifiable patient information.

# Competing interests

The authors declare that they have no competing interests.

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# Author Contribution

SL and YC contributed equally to this work. YZ and JW conceived and designed the study. SL, YC, XF, and XW collected and assembled the clinical data. SL and YC performed the statistical analysis and interpreted the data. XH and YS provided administrative support and supervised patient management. SL and YC drafted the manuscript. XF, XW, XH, YS, JW, and YZ critically revised the manuscript for important intellectual content. All authors read and approved the final manuscript.

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Not applicable.

# Availability of data and material

The datasets generated and analyzed during the current study are available from the corresponding author upon reasonable request, subject to institutional and ethical restrictions.

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## Figures

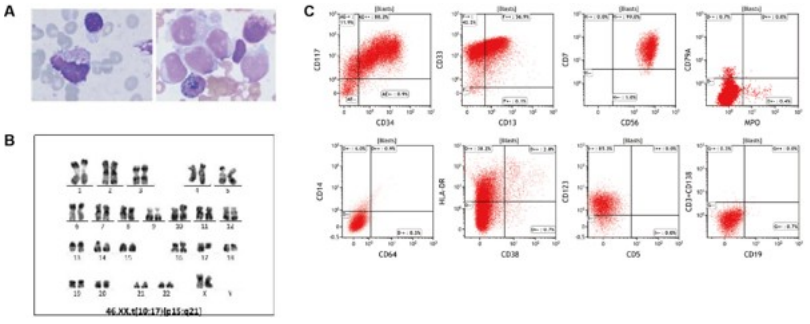



**Figure 1**

The morphology, karyotype and immunophenotype of case P3. (A) Wright-Giemsa–stained bone marrow smear (original magnification ×1000, oil immersion). (B) G-banded karyotype of a metaphase spread (original magnification ×1000). (C) Immunophenotyping by flow cytometric analysis of P3 bone marrow revealed a distinct leukemic population (in red), illustrating expression of CD33, CD34, CD117, CD123 , CD7 and CD56, partial expression of CD13 and HLA-DR. The leukemic cells were negative for CD5, CD14, CD38, CD19, CD64, CD138, cCD79A and MPO.

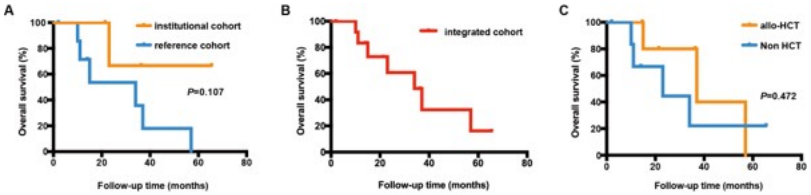



**Figure 2**

Overall survival in adult AML with *ZMYND11*::*MBTD1*fusion. (A) Overall survival of the institutional cohort compared with the reference cohort. (B) Overall survival of the integrated cohort. (C) Overall survival of patients who underwent allo-HCT compared with those who did not undergo allo-HCT. Curves were generated using 13 patients evaluable for OS. The institutional cohort had only one death event at data cutoff; therefore, between-group comparisons should be interpreted cautiously. OS, overall survival; allo-HCT, allogeneic hematopoietic cell transplantation.