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A glimpse into translocation (8;21) in acute myeloid leukemia: Profile and therapeutic outcomes from a tertiary care hematology center from East India

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ABSTRACT

Objectives: Translocation (8;21) is a RUNX1-RUNX1T1 fusion transcript, a favorable risk cytogenetic abnormality with a variable clinicopathological profile. However, there is a paucity of data on the outcomes of acute myeloid leukemia (AML) with t(8;21) from East India. This report is an analysis of data of AML with t(8;21) at our center.

Material and Methods: *De novo* AML patients with the presence of t(8;21) cytogenetic abnormality from 2015 to 2019 were analyzed for clinical, pathological, and molecular characteristics and were compared with treatment outcomes. Relapse-free survival (RFS) and overall survival (OS) were determined using Kaplan–Meier curves.

Results: Twenty-nine patients (10%) with *de novo* AML had t(8;21) with 18 male patients and a median age of 20 years. Aberrant expression of CD19, CD56, and CD7 expressions was noted in 44.8%, 17.24%, and 10.29% of patients, respectively. Additional cytogenetic abnormality was observed in 31.03%. CD19 had an 80% correlation with the occurrence of C-kit status. High-dose induction therapy had complete remission rates of 100%. The median duration of follow-up was 287.5 days. The presence of myeloid sarcoma (MS) and C-kit positivity had inferior OS and RFS ($P < 0.05$). The dose of cytosine arabinoside, given in consolidation of 3 g/m^2 and 1.5 g/m^2, had a median OS of 758 and 479 days ($P = 0.661$) and median RFS of 348 and 150 days ($P = 0.002$), respectively. In the group that received intensive therapy, by the end of 3 years, only 15.7% of patients remain in remission.

Conclusion: AML with t(8;21) is seen in young patients with a positive correlation between CD 19 with C-kit positivity. The presence of MS and C-kit positivity endowed inferior OS and RFS. Cytosine arabinoside consolidation in a dose of 3 g/m^2 offered an advantage in median RFS.

Keywords: Acute myeloid leukemia, Cytosine arabinoside, t(8;21)

INTRODUCTION

Acute myeloid leukemia (AML) with t(8;21) as per European Leukemia Network (ELN) 2017 risk stratification is favorable risk. This aberration is seen in approximately 1–5% of all newly diagnosed AML patients.[1,2] This new chimeric RUNX1-RUNX1T1 gene fusion on chromosome eight which acts as a repressor for all hematopoietic differentiation processes mediated by RUNX1 and thus causes leukemogenesis.[3,4]

As per Medical Research Council studies, the long-term disease-free survival for AML patients with t(8;21) subset has a high survival rate.[5] A study from South India described the profile

of patients with t(8;21) AML and highlighted additional chromosomal aberrations in 88% of patients. Among these group of patients, 96% of *de novo* AML patients achieved complete remission (CR) rates after induction and overall survival (OS) at 31 months was 69%.[7] None of the studies from East India in AML reviewed the clinical biological patterns of t(8;21) and we do not have any data for the clinical outcome with this translocation from this part of the country. Considering the demographic and ethnic variations in the pattern; in this study, we endeavored to throw light on the clinicopathologic, the cytogenetic profile, and the therapeutic outcomes of t(8:21) in *de novo* AML from this region.

MATERIALS AND METHODS

Patients

De novo AML was diagnosed in 287 patients by the World Health Organization (WHO) 2008 classification. This study was conducted in the hematology department of a tertiary care center in East India. This was a retrospective analysis done on AML patients between the duration of January 2015 and December 2019. Among these, 29 patients had t(8;21) and thus patients were enrolled in the study.

Diagnostic evaluation

Clinicopathological, molecular profile, treatment, and outcome details were collected from case sheets of these patients. Blood investigation such as complete blood count and peripheral smear (PS) was done on all patients using Sysmex XP100 and Leishman stain, respectively. Morphological assessment of bone marrow aspirate (BMA) slides was done with Leishman-Giemsa staining. All the BMA samples were analyzed for immunophenotyping (IPT) and cytogenetics study. IPT was assessed by flow cytometry using a three laser, 10-color FACSCalibur instrument (Beckmann Coulter) with a Navios software system using the following markers: CD45, CD34, human leukocyte antigen and DR, nuclear TdT, cytoplasmic myeloperoxidase, CD117, CD13, CD14, CD33, CD36, CD64, CD41, and CD42b; B-cell markers such as CD19, CD79a, CD10, and CD20, and T-cell markers CD 3, sCD3, CD2, CD5, and CD7. BMA for cytogenetics was processed using standard protocols followed by GTG banding (G banding with trypsin using Giemsa staining) with a banding resolution of 550 and analyzed 20 metaphases. The reporting of karyotypes was done by the ISCN in 2013 and 2016.[8] After the confirmation of diagnosis, fluorescent *in situ* hybridization panel for t(8;21), MLL rearrangement and t(16;16), and molecular markers such as NPM1, C-kit, FLT3, and CEBPA were assessed using Sanger sequencing.[9,10]

Treatment

After counseling about the disease, the various treatment choices, and outcomes to the patients and their attendants, consent was sought for therapy. Therapy plans were made according to the age, frailty scores, comorbidities, and baseline Eastern Cooperative Oncology Group score.[11] Patients eligible for intensive chemotherapy were induced with daunorubicin (DA) 60 mg/m^2 for 3 days plus cytosine arabinoside (Ara C) 100 mg/m^2 infusion for 7 days. Once patients achieved hematological recovery, they underwent a repeat bone marrow examination, and therefore, patients were assessed for remission. The patients who attained complete hematological remission post-induction were consolidated with three cycles of high-dose cytosine arabinoside (HiDAC) at a dosage of 3 g/m^2 or 1.5 g/m^2. These dose cohorts are in agreement with the revision in department policy regarding the dosage of HiDAC in the consolidation regimens. The patient's ineligible or not willing for intensive therapy was given hypomethylating agents (HMAs).[12-15] All the patients were monitored rigorously and as per the clinical requirements, they received supportive care. The patients who received HiDAC consolidation therapy received no further treatment until hematological relapse. After achieving remission, these patients were planned for allogeneic transplants; however, they dropped out of transplant due to logistic issues.[16-18]

Statistical analysis

This study was powered to see the clinical, pathological characters, and molecular characteristics of patients with AML. It also aimed to see the impact of the clinicopathological parameters and therapy options including the dose of HIDAC in consolidation therapy on outcomes. Kaplan–Meier curves were applied to estimate the relapse-free survival (RFS) and OS; differences in survival distributions were evaluated by a log-rank test. Two-sided $P < 0.05$ was considered statistically significant. Non-parametric tests were used for correlational analysis. Software SPSS version 25 (SPSS Inc.) was used for analysis.

RESULTS

During the study period between January 2015 and December 2019, 287 patients with *de novo* AML underwent cytogenetic and molecular analysis. t(8:21) was seen in 29 (10.10%) patients among all the newly registered patients of *de novo* AML. Among these patients, 18 were males and 11 females, median age of the study group was 20 years (range: 3–62 years). The baseline patient characteristics of patients enrolled in the study are summarized in [Table 1]. Extramedullary disease (EM) in the form of myeloid sarcoma (MS) has been observed in nine patients (33.74%) and the most common site of presentation of MS was orbit (66.74%).

Table 1: Patient characteristics.

Patient variables	All patients (*n*=29)
Age (years) median (range)	20 (3–62)
Male: female	18:11
Myeloid sarcoma *n*, (%)	9 (31.03)
Hemoglobin (g/L) median (range)	69.5 (37–122)
Total WBC (×10^9/L) median (range)	6.75 (1–60)
Total platelet count (×10^9/L) median (range)	10 (2–113)
Peripheral smear blast percentage (%) median (range)	31 (0–90)
Marrow blast percentage (%) median (range)	70 (14–95)
Aberrant phenotypic expression	
CD19 positivity *n*, (%)	13 (44.8)
CD 7 positivity *n*, (%)	3 (10.29)
CD 56 positivity *n*, (%)	5 (17.24)
C-kit exon 17 and exon 8 mutations *n*, (%)	10 (34.48)
Additional cytogenetics *n*, (%)	9 (31.03)
Monosomy of sex chromosomes n, (%)	8 (27.5)
9 q deletion n, (%)	1 (3.4)
Trisomy 4 n, (%)	1 (3.4)
Risk category	
Good risk category *n*, (%)	20 (68.9)
Intermediate risk category *n*, (%)	9 (31.03)
Therapy used	
Induction with 3 plus 7 *n*, (%)	19 (65.51)
Demethylating agent *n*, (%)	5 (17.24)
Consolidation	
Three HIDACs *n*, (%)	15 (51.72)
Dose of cytosine arabinoside	
1.5 g/m^2 *n*, (%)	6 (20.68)
3 g/m^2 *n*, (%)	13 (44.8)

AML: Acute myeloid leukemia, CR: Complete remission, CD: Cluster differentiation, HIDAC: High-dose cytarabine

The median blast percentage was 70% (range: 14–95%). Blast percentage of <20% in the marrow was seen in one patient, and here, this translocation served as a disease-defining abnormality. Auer rods were seen in a PS of five patients of AML with t(8;21) (q22; q22). On karyotyping, additional chromosomal abnormalities were seen in 9 patients (31.03%) and the most frequently observed additional chromosomal abnormality seen in the study group which was the loss of sex chromosome (LSC) followed by deletion (del) of the long arm (q) of chromosome 9 and trisomy 4 which was seen in 8 (27.5%), 1 (3.4%), and 1 (3.4%) patients, respectively.

IPT was performed on all patients. The aberrant expression of CD19, CD56, and CD7 expressions was noted in 44.8%, 17.24%, and 10.29% of patients, respectively. NPM1, FLT3, and CEBPA were performed in all patients and none of the patients in the study group were positive for this mutation. C-kit mutations were performed on all the patients with t(8:21) and C-kit mutation was detected in 10 patients (34.4%) from the study group. A correlational test was applied to see an association between CD19 and C-kit and a positive correlation of 0.801 was seen between the two groups.

Therapy was initiated in all 29 *de novo* AML patients with t(8;21). Among these, 19 patients received a conventional 7-day cytosine arabinoside and a 3-day DA induction regimen. All the patients (19/19) 100% attained CR after completion of the first induction. No induction-related mortality was observed in our study. Followed by induction, all the patients received consolidation therapy with HiDAC. Thirteen patients received consolidation with HiDAC (3 g/m^2) and intermediate-dose cytosine arabinoside (1.5 g/m^2) was given to six patients. Therapy with demethylating agent azacitidine was given to five patients (17.24%). Only 1 patient (20%) was in CR after 6 cycles of azacitidine with OS of 291 days. Rest 5 patients (17.24%) opted for the best supportive care.

The median duration of follow-up in the study group was 287.5 days (11–1642 days). The Kaplan–Meier curves are shown in [Figures 1 and 2]. In the subgroup analysis, the OS and RFS were analyzed using a log-rank (Mantel-Cox test) with a level of significance taken as 0.05. Patients with MS had poorer OS and RFS in comparison to those with the absence of MS ($P < 0.05$). At 3 years, none of the patients with the presence of MS was alive. The OS and RFS were also analyzed for the presence and absence of CD 19 status. The median OS with the presence of CD19 and absence of CD 19 was 479 and 911 days, respectively, and the median RFS in the presence of CD19 and absence of CD 19 was 240 and 436 days, respectively. The calculated *P*-value for the OS and RFS for CD19 was 0.606 and 0.628, respectively, which highlighted that there was no significant difference in survival based on CD19 status.

Then, the molecular marker C-kit was also analyzed for the survival rates, the median OS with the presence and absence of C-kit in patients was 176 and 548 days, respectively, and the median RFS in the presence and absence of C-kit was 449 and 994 days, respectively ($P = 0.017$), and at 3 years follow-up, none of the patients with C-kit mutation was alive. In terms of choice of therapy, 3+7 induction therapy had a better median OS and RFS in comparison to HMA-based therapy ($P < 0.05$). The dose of HiDAC, given in consolidation, was compared between 3 g/m^2 and 1.5 g/m^2 which had a median OS of 758 and 479 days, respectively ($P = 0.661$) and median RFS of 348 and 150 days, respectively ($P = 0.002$). Among those who received intensive therapy at a median follow-up of 3 years only 15.7% of patients (3/19) were in continuous remission.

DISCUSSION

The t(8;21) is a distinct entity and is well documented to have a favorable outcome. It has been categorized as a separate

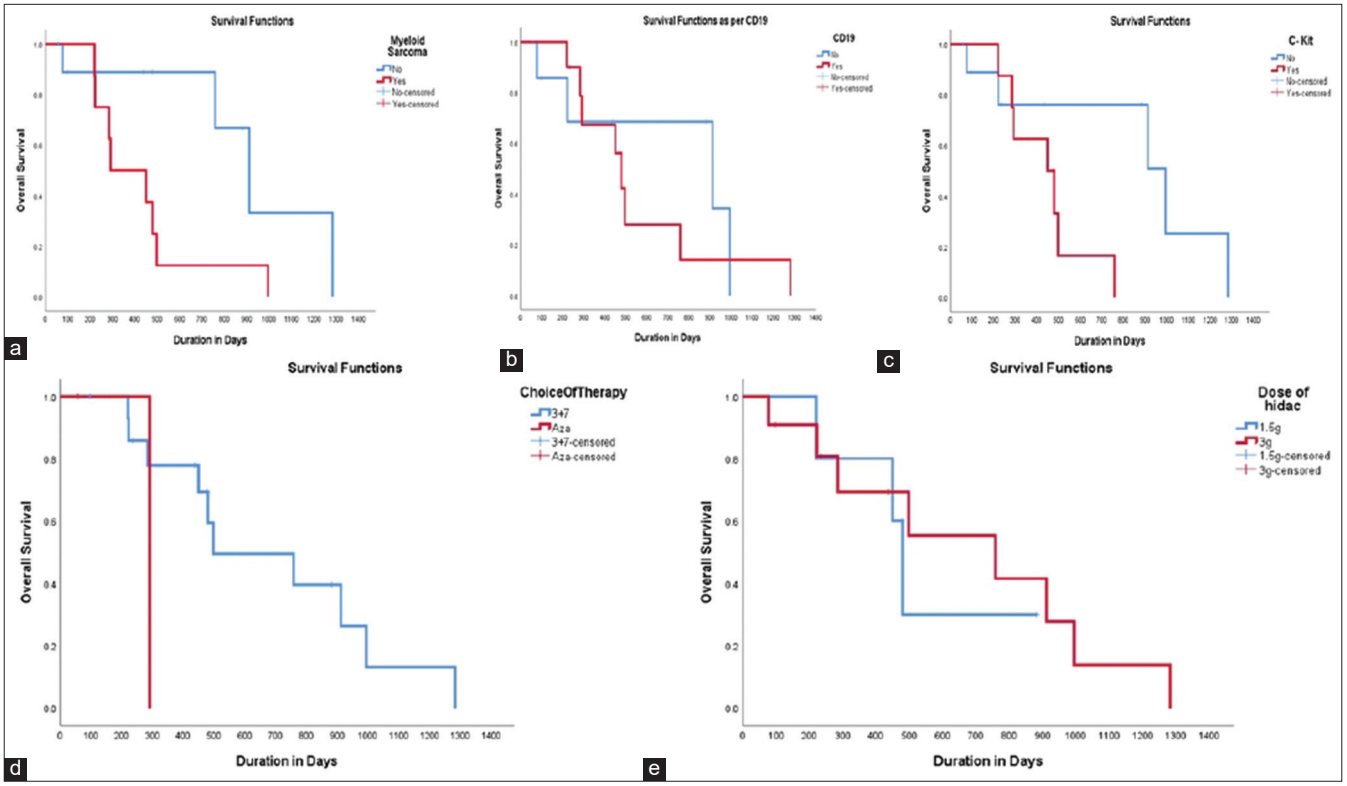

Figure 1: Overall survival on the basis of (a) myeloid sarcoma, (b) CD19 status, (c) C-kit status, (d) choice of therapy, and (e) dose of cytosine arabinoside.

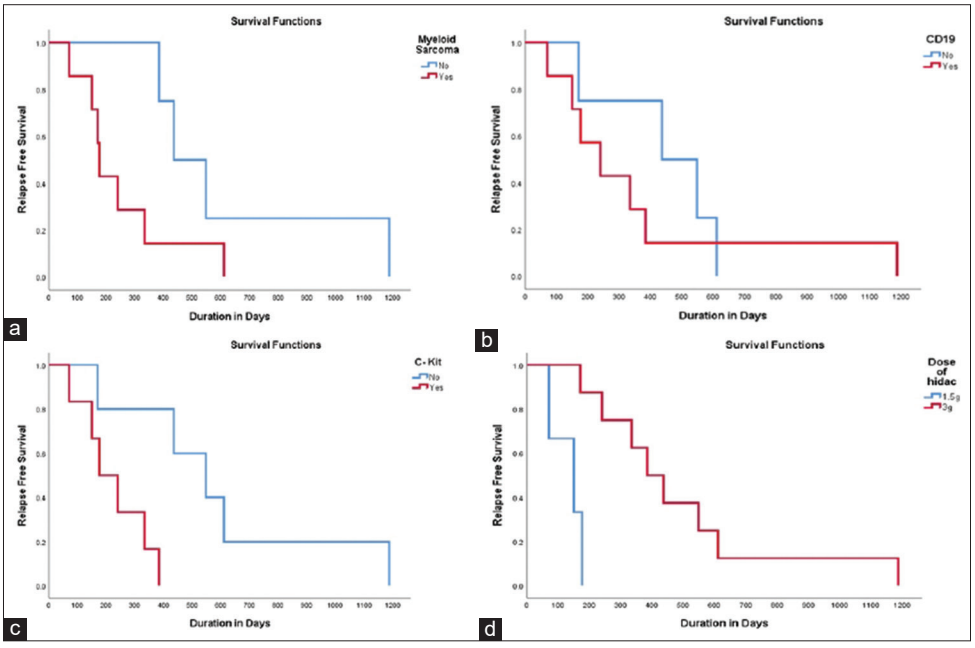

Figure 2: Relapse-free survival based on (a) myeloid sarcoma, (b) CD19 status, (c) C-kit status, and (d) dose of cytosine arabinoside.

entity in the WHO 1998 classification of myeloid neoplasia. In the 2008 WHO revision, the presence of this abnormality is defined as disease defining abnormality even in the absence of bone marrow blasts <20%.[19] The overall incidence of this

abnormality in our series was 10.10%, which was comparable to the reports globally.[20] The translocation was more common in males (sex ratio 1.63) and younger individuals (below 30), with a solitary patient above 60 years of age.[21] EM disease has been reported in 10–25% of patients with t(8;21) AML in the literature and we observed them in nine of our patients.[22] Patients with MS had poorer OS and RFS in comparison to those with an absence of MS ($P < 0.05$). The presence of EM disease is usually associated with a poor prognosis and shorter survival 5-year survival rates for patients with MS range between 20% and 30%, which appear similar to AML in general.[27]

The cytogenetic findings in this series are divergent from the results in the literature. The incidence of additional abnormalities in our study was 31.03% and other Indian studies have reported it to be 65%.[23] On IPT, aberrant CD19 positivity and CD56 positivity rate in this study were 44.8% and 17.24%, respectively, which are relayed in other studies also, with CD19 and CD56 positivity were seen in 54% and 20% of patients, respectively.[23] Among patients with t(8;21) AML, C-kit mutations have been described in 6.3–12.7% of patients; however, in our study, it was observed in 34.24%.[24] There was a correlation of 80% between the presence of CD19 and C-kit. This shows that cases, that is, of all the patients who were C-kit positive, had positivity of CD19 in IPT. We do not have many studies to support our results. The median RFS and OS in KIT-mutated patients were inferior to that in unmutated patients ($P < 0.05$), and similarly, poor outcomes have been observed in other studies also.[26,28]

In patients treated with intensive induction therapy, the CR rate was 100% and the OS at end of 3 years was 15.7%. The median duration of follow-up in the study group was 287.5 days (range 11–1642 days). Overall disease-free survival is about 60% in t(8;21) AML in some studies.[25] However, 30–40% of cases usually relapse after standard intensive chemotherapy, among this half of them become resistant to treatment which has been reported in numerous studies.[25] The median OS was 19.8 months in the favorable risk which has been reported from low-income countries.[30] Although our work did not examine the factors responsible for the inferior survival seen in our cohort when compared to other centers, the presence of other molecular markers or epidemiological factors could have some influence for which more studies are warranted.

However, we observed that the median RFS was higher with the dose of HiDAC 3 g/m^2 but with no difference in outcomes in terms of median OS. EORTC-GIMEMA AML-12 trial improves the outcome of adult patients younger than age 46 years with acute myeloid leukemia with a higher dose of HiDAC.[29] In our study group, the patient received intensive therapy at a median follow-up of 3 years, only 15.7% of patients were in continuous remission unlike in other studies. These variations could be due to the heterogenicity in AML and the demographic variation. All our patients received treatment according to the ELN recommendations. However, the frequent use of next-generation sequencing in such cases may help in further identification of the other risk factors.

Although we have many studies globally which have been conducted to define t(8;21) abnormality and its clinicopathological behavior and outcomes, we do not have many reported from Indian studies, especially for this part of the country. This appears to be the only series of t(8;21) from East India with survival data in this subset of AML.

CONCLUSION

Our findings are similar to the literature for the overall incidence of t(8;21) in AML, the median age, male predominance, and similar incidence of LSC. We noted a higher incidence of extramedullary leukemia, but we noted a similar incidence of aberrant CD19 expression and CD56 expression as reported. The incidence of C-kit mutations was higher than what has been reported in previous studies. We noted to have a positive correlation between CD 19 with C-kit positivity. Poor outcomes and C-kit status have been established in previous studies. We also noted in this study cohort that the presence of MS and C-kit positivity showed lower RFS. The median relapse-free survival was higher in case the dose of HiDAC was 3 g/m^2; however, the median OS remains the same irrespective of the dose of cytosine arabinoside. This appears to be the only series of t(8;21) AML from East India. However, studies with large sample sizes and longer follow-ups are required to enrich our understanding.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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