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## Comparison of autologous stem cell transplantation versus consolidation chemotherapy for patients with cytogenetically normal acute myeloid leukemia and FLT3ITD

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### Abstract

We retrospectively analyzed clinical outcomes of patients aged 18–59 with cytogenetically normal AML (CN-AML) and mutations in the FLT3 receptor according to type of postremission therapy. Specifically, we compared the outcomes of patients who underwent autologous SCT versus consolidation chemotherapy. There were 37 patients with an ITD mutation (7 also had a TKD mutation) and 19 patients with an isolated TKD mutation at diagnosis. In all, patients with an isolated TKD (n=16) had an improved DFS and OS (p=.031 and .014, respectively) compared to ITD patients (n=21). For individuals with an isolated TKD mutation, survival outcomes were similar irrespective of the type of postremission therapy (n=7 for SCT and 9 for chemotherapy) (p=0.97 and 0.082, respectively). However, ITD positive patients who underwent an SCT in CR1 (n=10) had an improved DFS but similar OS compared to those who received consolidation chemotherapy (n=11) (p=.05 and .27). These results suggest that high dose chemotherapy with autologous SCT may be a reasonable therapeutic choice over consolidation chemotherapy for young CN-AML patients with a FLT3ITD mutation.

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Acute myeloid leukemia (AML) is a relatively resistant myeloid neoplasm commonly occurring in older patients (median age at diagnosis is 67 years).(1) AML outcomes are heterogeneous, with cure rates ranging from essentially zero in subsets of older patients to 80% in younger patients with favorable disease features.(2) Among various risk factors, karyotype is an important independent risk factor and is used to divide AML into good,

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#### Conflict of Interest

Dr. Richard M Stone has served as a consultant for Novartis

intermediate and poor prognostic groups.(3, 4) The normal karyotype group is considered to be at intermediate risk and constitutes approximately 40% of patients with AML. However, even these patients have a heterogeneous outcome, with OS rates ranging from 24% to 42%. This group may be further stratified according to the presence or absence of mutations or expression levels of various molecular markers. (5, 6)

FLT3ITD represents a segmental duplication within the juxtamembrane domain of FLT3 receptor and is found in 30–40% of CN-AML patients.(8) FLT3ITD is associated with monocytoid differentiation (FAB M5), higher white blood cell counts, increased serum lactate dehydrogenase, and higher peripheral and bone marrow blasts compared to wild-type FLT3 (wt FLT3) CN-AML patients.(9, 10) Patients with this mutation have similar complete remission (CR) rates but significantly increased relapse rates and a reduced OS as compared to patients with wt FLT3.(9–11) It has been further identified that the allelic ratio of mutant:wt transcripts impacts prognosis.(8, 10, 12–14) The length of the duplicated fragment and its specific location within FLT3 may also affect prognosis.(13, 15–17) Overall, CN-AML patients with a FLT3ITD mutation have an inferior prognosis compared to those with a wtFLT3. There is considerable debate over the best postremission treatment for FLT3ITD CN-AML patients.(18, 19)

Another mutation, FLT3TKD, is a point mutation in the tyrosine kinase domain of the receptor and is found in approximately 10% of all CN-AML patients.(8) Similar to patients with FLT3ITD mutations, patients with FLT3TKD also have an elevated white blood cell count at presentation. However, the prognostic significance of TKD mutations is unclear.(9–11, 22–24)

We analyzed patients with CN-AML and mutated FLT3 to determine the relationship between different postremission therapies and outcome.

Our study includes all consecutively treated patients at our institutions who consented for determination of their FLT3 mutation status at diagnosis on a research study. The choice of post-remission therapy was not randomized but instead reflects the availability of open protocols and physician and patient preference.

There were a total of 37 patients who were below the age of 60 years at diagnosis and had a normal karyotype with a FLT3ITD. Treatment histories of these patients are described in supplemental material (Fig S1). Patients who were positive for both the ITD and TKD mutation were included in the ITD group (n=7) because these individuals have similar outcomes as those with an isolated ITD mutation.(23, 27) The remission frequency for this patient population was 81.1% (30/37). There were 19 newly diagnosed AML patients with an isolated TKD mutation who were under the age of 60 and had a normal karyotype. Their treatment histories are shown in Supplemental Figure 2 (Fig S2). The CR frequency for this group was 89.5% (17/19).

Excluded from our research question were 4/37 patients with FLT3ITD and 1/19 with FLT3TKD who received allogeneic SCT in CR1. However, we included patients who received an allogeneic SCT after first relapse (ITD: n=5; TKD: n=3). Time dependent covariate analysis indicated that receiving an allogeneic SCT after relapse did not significantly improve OS (p=0.62 for the ITD group and 0.83 for the entire cohort of patients; there were too few patients in the TKD group for this analysis to be conducted).

Patient characteristics such as age at diagnosis, time to CR1, WBC at diagnosis and gender are in presented in Table 1. These features were balanced between those who were assigned to autologous SCT or consolidation chemotherapy in both ITD and TKD patient groups. Also, there was no significant difference in the distribution of allelic ratio and ITD length

within the two treatment groups for ITD patients ( $p=.22$  and  $.2$ , respectively). We then studied the relationship between baseline characteristics and clinical outcome and we found that among the characteristics listed in Table 1, only female gender was associated with a better DFS in patients with an isolated TKD mutation ( $p=.03$ ).

We also found that an allelic ratio of  $>.2$  was associated with a worse DFS and OS in ITD mutation positive patients who had initiated postremission autologous SCT or chemotherapy ( $n=21$ ) ( $p=0.01$  and  $0.04$ ). A value of  $0.2$  was determined as the optimal cut point by recursive partitioning analysis. The median length of the ITD in patients who initiated postremission autologous SCT or chemotherapy was  $33\text{bps}$  ( $n=21$ ) (Range= $17-210$ ). For patients with more than one mutation ( $n=5$ ), the length of the most dominant mutation was chosen for analysis. In our study, the ITD length was not associated with DFS or OS. No other variable was noted to influence DFS or OS.

We next analyzed the survival of patients with an isolated TKD mutation according to type of postremission therapy. For the 7 patients who initiated autologous SCT compared to the 9 patients who initiated consolidation chemotherapy, there was no significant difference in DFS or OS ( $p=0.97$  and  $0.082$ , respectively). Similarly, there was no significant difference in DFS or OS for patients who received a stem cell infusion or at least 3 cycles of consolidation chemotherapy ( $n=6$  and  $8$ , respectively;  $p=0.78$  and  $0.25$ , respectively).

On the other hand, patients with an ITD mutation who initiated autologous SCT ( $n=10$ ) had an improved DFS compared to those who initiated consolidation chemotherapy ( $n=11$ ) ( $p=0.05$ ) (Figure IA), however there was no difference in OS ( $p=.27$ ) (Figure IB). Since some patients in each cohort ( $n=2$  for autologous SCT and  $n=2$  for consolidation chemotherapy) did not complete the initiated postremission therapy, defined as having received infusion of stem cells or at least 3 cycles of chemotherapy, respectively, we then compared the survival in patients who had completed their postremission therapy as defined above. Again, we found that autologous SCT ( $n=8$ ) was significantly associated with an improved DFS compared to consolidation chemotherapy ( $n=9$ ) ( $p=.03$ ) while no significant difference in OS was observed ( $p=.19$ ).

Lastly we compared the survival outcomes of patients with ITD versus TKD who achieved CR and progressed to postremission therapy, excluding those who underwent allogeneic SCT in CR1. Patients with an isolated TKD mutation ( $n=16$ ) had a significantly longer DFS and OS compared to patients with ITD ( $n=21$ ) ( $p=.031$  and  $.014$ ).

In summary, we analyzed the outcome of AML patients under 60 years with normal cytogenetics and FLT3 mutations according to the type of postremission therapy they initiated or received. Younger adults with ITD who initiated or received autologous SCT had an improved DFS compared to consolidation chemotherapy. OS, however, was not different for the two treatment modalities. For patients with isolated TKD, there was no difference in DFS or OS of patients who received autologous SCT versus consolidation chemotherapy.

There is considerable debate about the optimal postremission therapy for CN-AML patients with a FLT3ITD mutation. Current options vary among allogeneic SCT, autologous SCT, and/or chemotherapy along with use of novel FLT3R inhibitors. In general, comparisons of autologous SCT and chemotherapy have not shown a clear difference in outcome when all AML patients are included. The potential benefit of autologous SCT in AML subgroups has not been extensively studied. Yoshimoto *et al.*, in a retrospective analysis, demonstrated a similar DFS and OS for patients with a wtFLT3 versus a FLT3ITD who underwent an autologous SCT in CR1.(28) Bornhauser *et al.* also noted a decrease in probability of relapse and an improvement in OS for patients with FLT3ITD who received an autologous SCT in

CR1 compared to those receiving chemotherapy alone.(19) In contrast, Schlenk *et al.* found no difference in relapse free survival and OS between normal cytogenetic patients receiving autologous SCT or conventional consolidation chemotherapy in both FLT3ITD and wt FLT3 patient groups.(18)

Our data raise the question as to whether autologous SCT may be considered over consolidation chemotherapy for patients with ITD who are not undergoing allogeneic SCT or therapy with a FLT3 inhibitor.

## MATERIALS and METHODS

### Patients

We conducted a retrospective analysis of all patients who presented to the Dana Farber Cancer Institute/Brigham and Women Hospital and Massachusetts General Hospital between 2002 and 2008 who underwent testing for FLT3 mutations. It was the policy to test all patients with newly diagnosed AML in this fashion. Patients had consented to an IRB approved research protocol prior to obtaining samples for FLT3 analysis.

We analyzed outcomes for all adult patients with FLT3 mutations who were newly diagnosed, had a normal karyotype and were < 60 years of age. CR rates, disease free survival, overall survival and type of treatment received were assessed by medical record review.

### Mutations Analysis

FLT3 mutations, ITD length, sequence and allelic ratio, were determined as previously described.(25) Nucleophosmin gene (NPM1) mutation status is not available.

### End Points and Definitions

CR was defined by the presence of a normocellular bone marrow containing < 5 percent blasts and showing trilineage maturation with an absolute neutrophil count of more than 1000/ul and a platelet count of more than 100,000/ul.(26) DFS was defined as the time from the date of first CR to an event (death in first CR or relapse). OS was defined as the time from diagnosis to date of death. Patients lost to follow-up were censored at the date of last documented contact.

### Statistics

The two sample t-test and the Fisher exact test were used to assess the significance of differences in clinical parameters between the patient groups. The method of Kaplan and Meier was used to estimate DFS and OS; groups were compared using the log-rank test. The Cox proportional hazards regression model was used to explore associations between therapeutic and demographic characteristics and DFS or OS in a multivariable setting. Receipt of allogeneic transplant in the setting of relapse or CR2 or higher was analyzed as a time-varying covariate in these models. A p-value less than 0.05 was interpreted as statistically significant. There was no adjustment for multiple comparisons.

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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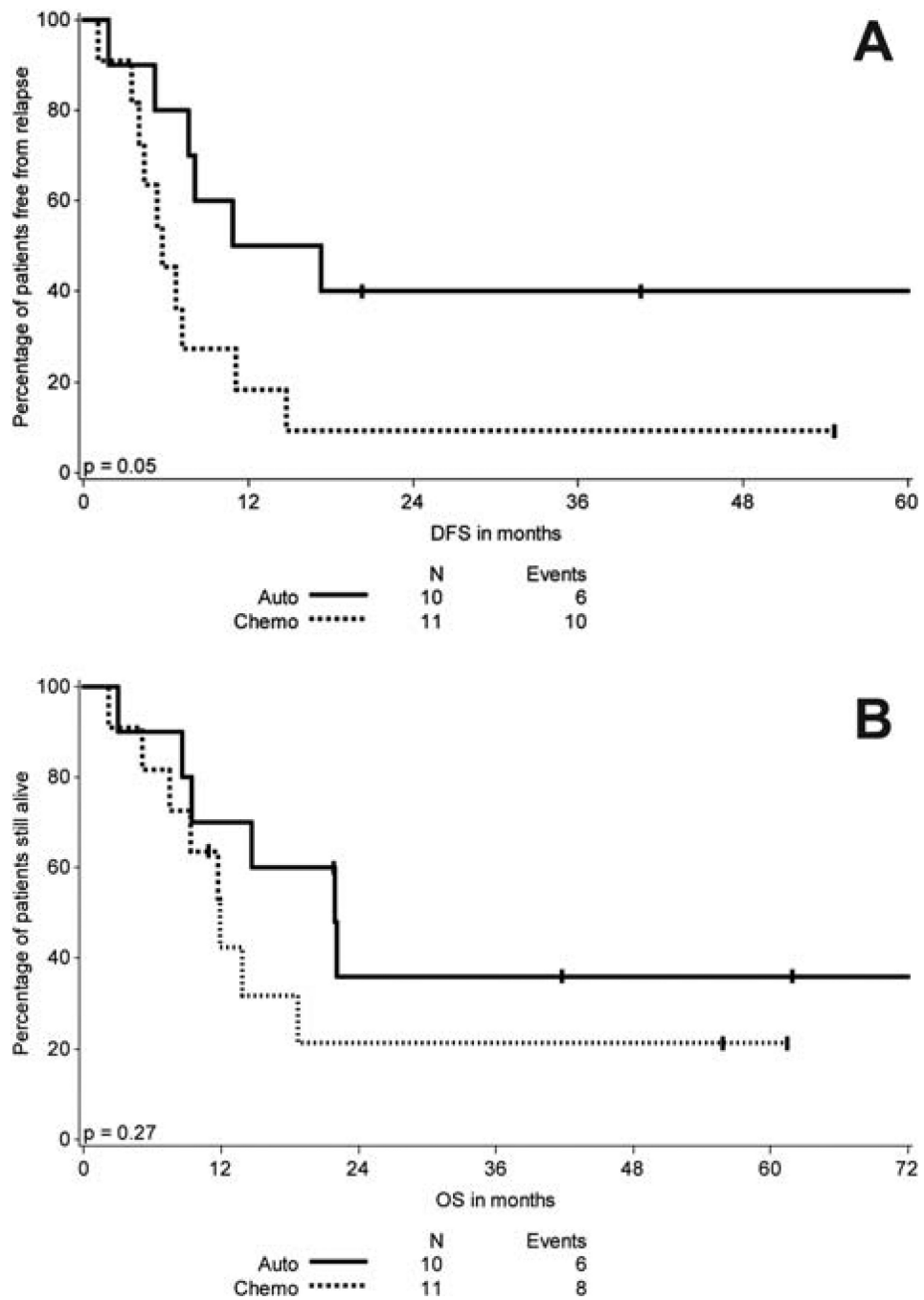
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**Figure 1.**

A. DFS in months of patients with FLT3R ITD according to type of postremission therapy initiated

B. OS in months of patients with FLT3R ITD according to type of postremission therapy initiated



**Table 1**

Patient characteristics for ITD and TKD positive patients who initiated autologous SCT or consolidation chemotherapy. P value corresponds to comparisons within each type of mutation and not across FLT3 mutation types.

	ITD positive patients			TKD positive patients			P value
	Autologous SCT	Chemotherapy	P value	Autologous SCT	Chemotherapy	P value	
	N	Median, range	N	Median, range	N	Median, range	
Age at dx	10	48 (29, 59)	11	47 (21, 60)	7	49 (39, 60)	0.22
Duration from diagnosis to CR1 (months)	10	1.35 (0.99, 2.07)	11	1.18 (0.95, 2.65)	7	1.2 (0.9, 1.8)	0.97
WBC	9	58.7 (0.9, 150.2)	11	52.9 (13.1, 149.9)	7	27 (16, 238.6)	0.96
Gender							0.65
Female	5		7		3		
Male	5		3		4		
ITD length	10	40.5 (18, 210)	11	27 (17–165)			0.2
Allelic Ratio	10	.27 (.02,1.17)	11	.32 (.16–1.32)			0.22
					9	10.1 (2.0, 53.9)	0.16
					8		0.1
					1		