











Long-Term Results of the Dasatinib-Blinatumomab Protocol for Adult Philadelphia-Positive ALL

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ABSTRACT

Clinical trials frequently include multiple end points that mature at different times. The initial report, typically based on the primary end point, may be published when key planned coprimary or secondary analyses are not yet available. Clinical trial updates provide an opportunity to disseminate additional results from studies, published in JCO or elsewhere, for which the primary end point has already been reported.

We report the long-term results of the frontline trial with dasatinib and blinatumomab in induction/consolidation (GIMEMA LAL2116, D-ALBA) for adult Philadelphia-positive ALL (Ph+ ALL), which enrolled 63 patients of all ages. At a median follow-up of 53 months, disease-free survival, overall survival, and event-free survival are 75.8%, 80.7%, and 74.6%, respectively. No events have occurred among early molecular responders. A significantly worse outcome was recorded for *IKZF1*^{plus} patients. Twenty-nine patients—93.1% being in molecular response (ie, complete molecular response or positive nonquantifiable) after dasatinib/blinatumomab—never received chemotherapy/transplant and continued with a tyrosine kinase inhibitor only; 28 patients remain in long-term complete hematologic response (CHR). An allogeneic transplant was carried out in first CHR mainly in patients with persistent minimal residual disease; 83.3% of patients are in continuous CHR. The transplant-related mortality was 12.5% for patients transplanted in first CHR and 13.7% overall. Nine relapses and six deaths have occurred. *ABL1* mutations were found in seven cases. The final analysis of the D-ALBA study shows that a chemotherapy-free induction/consolidation regimen on the basis of a targeted strategy (dasatinib) and immunotherapy (blinatumomab) is effective in inducing durable long-term hematologic and molecular responses in adult Ph+ ALL, paving the way for a new era in the management of these patients.

ACCOMPANYING CONTENT

 [Data Supplement](#)

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INTRODUCTION

In Philadelphia-positive ALL (Ph+ ALL), tyrosine kinase inhibitors (TKIs) with or without chemotherapy, generally followed by an allogeneic stem cell transplant (SCT), have led to survival rates approaching 50% at 1-5 years.¹⁻¹⁵ Since 2000, the GIMEMA group pioneered a chemotherapy-free induction strategy for patients with newly diagnosed adult Ph+ ALL.¹⁶⁻²⁰ This approach led to complete hematologic responses (CHR) in 94%-100% of cases and virtually no deaths in induction, irrespective of age. A further advancement has come by adding blinatumomab. In the D-ALBA (GIMEMA LAL2116, ClinicalTrials.gov identifier: [NCT02744768](#)) frontline protocol for adult Ph+ ALL with no upper age limit, dasatinib plus steroids was administered in induction followed by at least two blinatumomab cycles as consolidation.²¹⁻²³ The initial report²¹ showed 60% of molecular responses (52% by intention to treat) at the primary end point (after two cycles of blinatumomab) and an overall survival (OS) and disease-free survival (DFS) of 95% and 88%, respectively, at 18 months. Post-consolidation treatment was left to the investigators' choice

(Data Supplement, Fig S1 [online only]). We report the long-term follow-up of the study.

PATIENTS AND METHODS

The treatment received after blinatumomab consolidation and the long-term follow-up were collected in the GIMEMA LAL2217 (ClinicalTrials.gov identifier: [NCT03318770](#)) ancillary trial after patients signed a written informed consent. The trial was approved by the Sapienza University Institutional Review Board. Demographics of the 63 patients enrolled (median age 54 years, 24-82), genomic characterization, response criteria, minimal residual disease (MRD) determination, and statistical analyses are detailed in the Data Supplement.

RESULTS

Molecular Responses and Survival

Molecular responses increased over time, being 75%, 77%, 70.1%, 85%, 90%, and 93% at 2, 4, 6, 8, 10, and 12 months,

respectively, from the last blinatumomab cycle. The percentage of patients with molecular responses increased further at a later follow-up, with the caution that molecular findings were often lacking for allografted patients or those who were lost to follow-up (not shown).

At a median follow-up of 53 months, DFS, OS, and event-free survival (EFS) were 75.8%, 80.7% and 74.6%, respectively (Fig 1). A significantly better DFS was observed in the 17 molecular responders at the end of induction (EOI; 100%) versus nonmolecular responders (n = 42; 68.6%; P = .018). After two blinatumomab cycles, the difference in DFS and OS was no longer significant between molecular responders (n = 33) and MRD+ patients (n = 22): 82.9% versus 75.7% and 89.4% versus 84.2%, respectively (Fig 2).

A significantly worse DFS was recorded for *IKZF1*^{plus} patients (n = 11, 45.5%) compared with those without *IKZF1* deletion (n = 25, 82.3%) or with *IKZF1* only deletions (n = 13, 74%; P = .029). OS results were similar (Data Supplement, Fig S2; Table S1).

Postconsolidation Treatment

Twenty-nine patients (median age 58 years, 30-70) neither received chemotherapy nor transplant and remained on TKI treatment. Twenty-seven patients (93.1%) had obtained a molecular response (ie, complete molecular response or positive nonquantifiable) after dasatinib/blinatumomab, 20 after two blinatumomab cycles, and seven after further blinatumomab cycles. Twenty-eight patients (96.5%) remain in persistent CHR at a median follow-up of 48 months (22-64). Further details are given in the Data Supplement (Fig S1).

Twenty-four patients were allografted in first CHR (median age 51.5 years, 24-67; Data Supplement, Fig S3). Thirteen patients (54.2%) were nonmolecular responders at the

primary end point; only two became molecularly negative after further blinatumomab cycles. One patient relapsed 1 month after SCT, and three died of complications. The remaining 20 patients (83.3%) are alive in CHR at a median follow-up of 49 (17-66) months.

Six patients were transplanted in second CHR; three died while three are alive in second CHR at 30, 40, and 52 months, respectively, from the second CHR.

The transplant-related mortality (TRM) in first CHR was 12.5% and 13.7% overall.

Relapses, Deaths, and Toxicity

Overall, nine relapses occurred—four hematologic, four involving the CNS, and one nodal (Table 1)—at a median of 4.4 (1.9-25.8) months. Three patients are alive after salvage treatment and SCT. The *IKZF1*^{plus} signature was present at diagnosis in four of eight evaluable cases; *ABL1* mutations, tested at relapse or at a concomitant MRD increase in extra-hematologic recurrences, were found in seven patients (six T315I and one E255K). Four patients harbored both the *IKZF1*^{plus} signature and T315I mutation. Although relapses occurred at a similar rate in p190 and p210 cases (14.6% and 13.6%), time to recurrence was shorter in p190 versus p210 cases (3.9 v 24.3 months). Five deaths occurred in first CHR (one pneumonia during induction, one endocarditis in a mechanical aortic valve carrier, and three after SCT). Toxicity is reported in the Data Supplement (Table S2).

DISCUSSION

Our group showed for the first time the advantage of treating adult/elderly Ph+ ALL frontline with dasatinib followed by blinatumomab.²¹⁻²³ Similar results were recently reported combining ponatinib and blinatumomab.²⁵ The final analysis of the D-ALBA study shows that at a median follow-up of

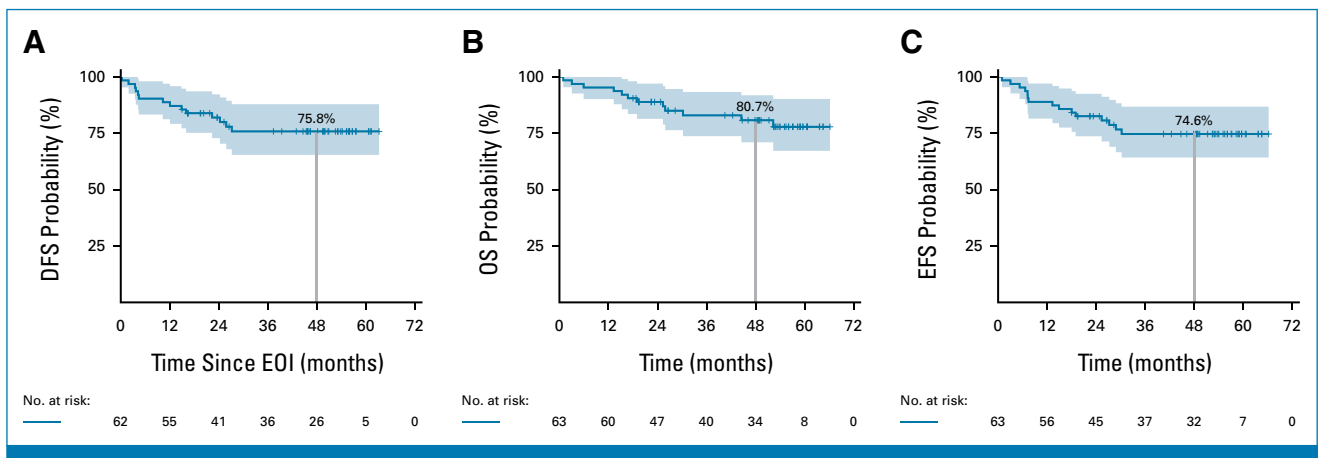


FIG 1. (A) DFS, (B) OS, and (C) EFS calculated from the EOI (d +85) and diagnosis/treatment initiation. The shadings of each curve show the 95% CI at a median follow-up of 53 months. DFS, disease-free survival; EFS, event-free survival; EOI, end of induction; OS, overall survival.

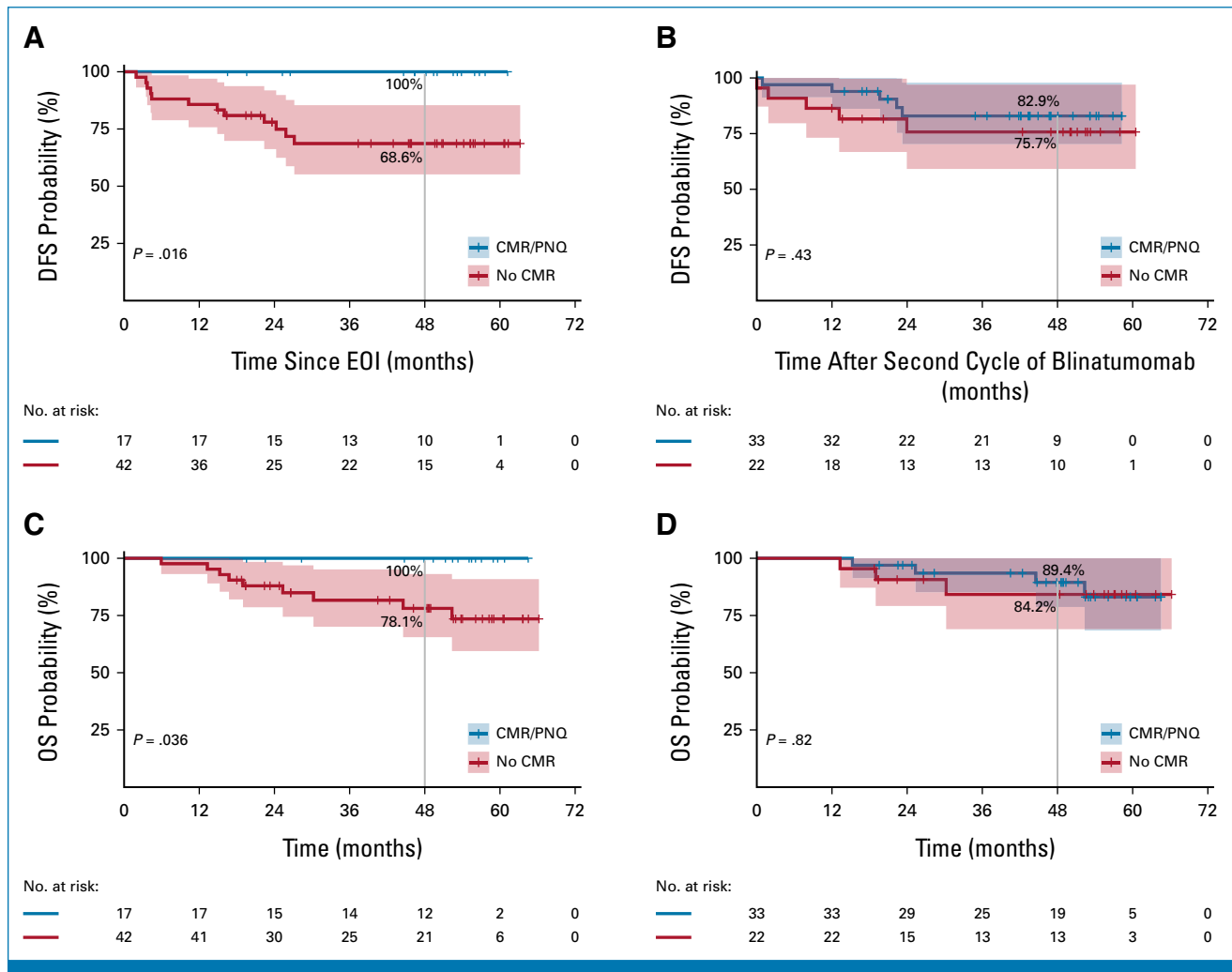


FIG 2. (A and B) DFS and (C and D) OS according to molecular response. DFS was calculated from (A) EOI (d +85) and after the (B) second cycle of blinatumomab (primary end point); OS was calculated from diagnosis/treatment initiation stratifying patients according to molecular response at the (C) EOI (d +85) and (D) after the second cycle of blinatumomab. Blue line: molecular responders; red line: non-molecular responders. The shadings of each curve show the 95% CI. CMR, complete molecular response; DFS, disease-free survival; EOI, end of induction; OS, overall survival; PNQ, positive nonquantifiable.

53 months DFS and OS are 75.8% and 80.7%, respectively, which is significantly better than in our previous dasatinib-based trial (Data Supplement, Figs S4–S5).¹⁸ DFS is 100% in molecular responders at the EOI, confirming the prognostic role of an early molecular response in a chemotherapy-free setting.²⁶ After two blinatumomab cycles, no significant long-term survival differences between molecular and nonmolecular responders were found, indicating that blinatumomab is effective in preventing a relapse also in patients with a suboptimal early molecular response, possibly also through a host immune modulation.^{21,24}

At a prolonged follow-up, *IKZF1*^{plus} patients continue to show a significantly worse outcome (Data Supplement, Fig S2), particularly when associated with T315I mutations.

Half of our patients remained on treatment only with a TKI; most (93.3%) were in molecular response after

dasatinib/blinatumomab and 96.5% are in CHR at 4 years. Instead, most patients transplanted in first CHR were MRD+, and 83.3% are alive in CHR (median follow-up 49 months). Although the trial was not designed to define the role of SCT, a clear benefit was seen in MRD+ patients while virtually all MRD– patients remain in long-term CHR without SCT. At a prolonged follow-up, we confirm the low TRM, likely due to the lack of systemic chemotherapy and good general conditions of patients undergoing transplant.

These long-term results compare favorably with more intensive treatments combining ponatinib and hypercyclophosphamide, vincristine, doxorubicin,²⁷ with an estimated 6-year OS of 75% and EFS of 65% in a younger population (median 46 years) and with the PONALFIL trial,²⁸ where younger patients (18–60 years) received ponatinib, chemotherapy, and transplant, with a 3-year OS and EFS of 96% and 70%, respectively.

TABLE 1. Clinical and Biological Features of Relapsed Patients

Age, Years	WBC (10 ⁹ /L) ^a	Type of Relapse	Fusion Type	<i>IKZF1</i>	Mutation	Time From CHR to Relapse, ms	Allo-SCT	Last Follow-Up	Previously Reported in Puzzolo et al ²⁴
71	157.9	Hematologic	p190	<i>IKZF1</i> ^{plus}	T315I	1.9	No	Dead	Yes
52	0.7	Hematologic	p190	ND	T315I	3.6	Second CHR	Dead	Yes
54	11.2	Nodal	p190	<i>IKZF1</i> ^{plus}	T315I	3.6	Second CHR	Dead	Yes
30	35.9	CNS + molecular	p190	<i>IKZF1</i> loss	E255K	4.2	Second CHR	Alive	Yes
40	10.5	CNS + molecular	p190	<i>IKZF1</i> ^{plus}	T315I	4	Second CHR	Alive	Yes
81	4.9	Hematologic	p190	No	T315I	12	No	Dead	Yes
53	7.2	CNS + molecular	p210	No	NE	24.3	Second CHR	Dead	No
45	23.9	CNS + molecular	p210	<i>IKZF1</i> loss	wt	25.8	Second CHR	Alive	No
67	2.9	Hematologic	p210	<i>IKZF1</i> ^{plus}	T315I	14.9	First CHR	Dead	No

Abbreviations: CHR, complete hematologic response; ND, not done; NE, not evaluable; SCT, allogenic stem cell transplant; wt, wild-type.

^aAt diagnosis.

Few relapses occurred (9): four had an *IKZF1*^{plus} signature at presentation and developed a T315I mutation during treatment, confirming our previous suggestion that *IKZF1*^{plus} might lead to an increased genomic instability. It appears that *IKZF1*^{plus}, if not coupled with the T315I mutation, may not per se induce a relapse. This has prompted to investigate T315I mutations by digital droplet polymerase chain reaction.²⁹ T315I mutations are a detrimental event, whose effect should be abrogated by using ponatinib, as explored by the PETHEMA²⁸ and GIMEMA²⁰ groups and the MD Anderson Cancer Center.²⁵ These trials did not report T315I mutations, except for one case.²⁰ Further results will come from the ongoing GIMEMA ALL2820 protocol, where ponatinib is used in place of dasatinib in the experimental arm. An emerging issue is represented by CNS relapses, recorded in four of nine cases. Although it is reassuring that three patients were salvaged by alternative treatments followed by transplant, a signature predictive of a CNS localization/recurrence is being investigated.³⁰

The role of p190 and p210 has been widely discussed: p210 cases generally show a slower MRD clearance, but this does ultimately not affect outcome. Although the noninferior

outcome is confirmed in our study (not shown), in p210 cases, relapses occurred at later time points.

The treatment was also well tolerated in the long-term follow-up, with only six patients requiring a TKI switch: Pleural effusion was the most common adverse events.

Taken together, the final analysis of the D-ALBA study (1) confirms the very high DFS and OS rates; (2) documents that early molecular responders are likely not to relapse; (3) shows that patients can remain in sustained long-term CHR without systemic chemotherapy and transplant; (4) shows that the protocol is associated with few relapses, predominantly in cases with an *IKZF1*^{plus} signature and T315I mutations; (5) points to the beneficial effect of transplant in MRD+ patients; and (6) confirms the low TRM in allografted patients. These results support the design of the ongoing GIMEMA ALL2820 trial where dasatinib is substituted by ponatinib, CNS prophylaxis has been strengthened, MRD has been further refined, and transplant allocation relies on the genetic profile at diagnosis and by the depth/persistence of molecular response.

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