Clinical outcomes of myeloid/lymphoid neoplasms with fibroblast growth factor receptor-1 (FGFR1) rearrangement

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ABSTRACT

Objective: Myeloid/lymphoid neoplasms with fibroblast growth factor receptor-1 (FGFR1) rearrangement are hematopoietic stem cell disorders with a poor prognosis, but no established standard therapy. Methods: We experienced a patient with T-lymphoblastic lymphoma (LBL) associated with FGFR1 rearrangement who underwent cord blood transplantation, but died of pulmonary complication. We collected the clinical data of patients with FGFR1 rearrangement from the medical literature and analyzed 45 patients, including our patient. Results: The primary diagnoses were myeloproliferative neoplasm (MPN) or myelodysplastic syndromes (MDS) in 14 and acute leukemia or LBL in 31. In MPN and MDS patients, the cumulative incidence of transformation to blast phase (BP) at 12 months was 46.2%. The 1-year overall survival (OS) from diagnosis in all cases was 43.1%. With regard to the impact of treatment response on survival, the achievement of complete response with a landmark at 2 months after diagnosis of BP was associated with a superior OS (40.0% vs. 26.0% P = 0.011 for 1-year OS from BP). Allogeneic hematopoietic stem cell transplantation (HSCT) was performed in 13 patients, and the 1-year OS from allogeneic HSCT was 61.5%. The hazard ratio for mortality was 0.34 (95% CI, 0.08–1.51, P = 0.15) for allogeneic HSCT treated as a time-dependent covariate, which suggests that allogeneic HSCT may confer a clinical benefit. Conclusion: The further accumulation of clinical data is needed to determine the optimal therapeutic approach for these neoplasms.

KEYWORDS
Fibroblast growth factor receptor-1 rearrangement; hematopoietic stem cell transplantation; myeloproliferative neoplasm; 8p11 myeloproliferative syndrome; T-lymphoblastic lymphoma; leukemia; lymphoma; cord blood transplantation

Introduction

Myeloid/lymphoid neoplasms with fibroblast growth factor receptor-1 (FGFR1) rearrangement are rare hematological malignancies associated with chromosomal rearrangement of the short arm of chromosome 8 [1]. This category of neoplasms was listed in the 2008 World Health Organization classification of tumors of hematopoietic and lymphoid tissues. Approximately 15 cases of FGFR1 translocation partner genes have been identified, and the most common gene partner is ZNF198 on chromosome 13q12 [2]. The term 8p11 myeloproliferative syndrome (EMS) was coined to explain this different entity.

Hematopoietic neoplasms with rearrangement of FGFR1 are most commonly diagnosed as myeloproliferative neoplasms (MPN). Its clinical course varies widely, and rapid transformation, most often to lymphoblastic lymphoma (LBL), acute lymphoblastic leukemia (ALL), or acute myeloid leukemia (AML), occurs within several months after diagnosis [3]. The prognosis is very poor despite intensive chemotherapy. Several reports have indicated that allogeneic hematopoietic stem cell transplantation (HSCT) achieved cytogenetic remission and is the only curative treatment option [4–11].

In a literature review of 65 cases of EMS [3], although various therapeutic regimens were used, the complete clinical remission rate was 27% and overall survival (OS) was 15 months. The median survival time after transformation for patients who received stem cell transplantation was 24 months. In this review, chromosome 8q11 abnormality was evaluated by conventional chromosomal analysis and FGFR1 rearrangement was not necessarily detected by a molecular analysis in all cases. An analysis limited to cases with confirmed FGFR1 rearrangement has not yet been reported.

Here we describe a patient with T-LBL associated with FGFR1 rearrangement who underwent allogeneic HSCT and achieved cytogenetic remission. Furthermore, we collected a case series by reviewing the literature to evaluate the clinical outcomes and therapeutic strategies, including allogeneic HSCT, for patients with hematologic neoplasms associated with confirmed FGFR1.
Patients and methods

Case presentation

A 34-year-old female visited another hospital for leukocytosis discovered during a medical checkup in July 2015. She had no subjective symptoms. Bone marrow (BM) aspirate and biopsy showed hypercellularity with hypereosinophilia, but no increase in the number of blasts. A G-banding chromosomal analysis revealed 46,XX,t(8:13)(p12;q12) karyotype in all 20 BM-derived metaphases. Reverse transcription-polymerase chain reaction (RT-PCR) detected a gene rearrangement of ZNF198-FGFR1. Therefore, she was diagnosed with MPN associated with FGFR1 rearrangement.

Two months later, she presented with bilateral cervical lymphadenopathy and was referred to our hospital for further management. Physical examinations showed diffuse cervical and axial lymphadenopathy. A computed tomography scan detected tonsillar swelling, systemic lymphadenopathy, and mild hepatosplenomegaly. Complete blood count showed a hemoglobin level of 13.5 g/dL, platelet count of 108 × 10^9/L, and white blood cell count of 65.9 × 10^9/L. Differential counts showed 0% blasts, 5.6% lymphocytes, 3.8% metamyelocytes, 58.0% neutrophils, 5.0% lymphocytes, 7.2% monocytes, 18.0% eosinophils, and 2.4% basophils. There were no increased blasts in the BM. We performed a biopsy of the cervical lymph node and the pathology showed the infiltration of lymphoblastic cells. Chromosome analysis of the lymph node cells revealed the 46,XX,t(8:13)(p12;q12) karyotype in 20/20 cells. Immunohistochemical stains and flow cytometric analysis demonstrated that the lymphoma cells were positive for CD3, CD4, CD5, CD7, terminal deoxyribonucleotidyl transferase. Based on the above findings, the patient was diagnosed with transformation to T-LBL.

The patient underwent intensive chemotherapy according to the ALL regimen. The induction regimen consisted of prednisolone, daunorubicin, vincristine, cyclophosphamide, and L-asparaginase. After induction therapy, 18F-fluorodeoxyglucose–positron emission tomography (FDG-PET) showed a complete response (CR); however, BM cells still showed the 46,XX,t(8:13)(p12;q12) karyotype. The patient then received consolidation therapy, including dexamethasone, etoposide, cytarabine, and L-asparaginase, and maintained CR. She planned to undergo allogeneic HSCT, but, unfortunately, a healthy family donor was not available. During coordination to identify an unrelated donor, she relapsed in January 2016 with bilateral cervical lymphadenopathy. She received reinduction chemotherapy consisting of dexamethasone, daunorubicin, cytarabine, cyclophosphamide, mercaptopurine, and L-asparaginase, but failed to achieve CR. She received a cord blood transplantation (CBT) in March 2016 after a conditioning regimen that included cyclophosphamide (60 mg/kg) for 2 days and total body irradiation (TBI) of 2 Gy twice daily for 3 days. However, neutrophil recovery was not achieved at 28 days after transplantation, and a chimerism analysis of BM cells at that time showed that all cells were of recipient origin and FGFR1 rearrangement was detected by fluorescent in situ hybridization (FISH) analysis. She was diagnosed as primary graft failure and a second CBT was carried out on day 43 after the first CBT, after a conditioning regimen consisting of fludarabine (30 mg/kg) for 4 days, cyclophosphamide (30 mg/kg), and TBI 2 Gy. She developed stage 1 acute cutaneous graft-vs-host disease (GVHD) on day 21 after the second CBT and was treated with topical corticosteroids. The neutrophil count surpassed 500/µL on day 28 after the second CBT, and a chimerism analysis of BM showed that 99.7% of cells were derived from the second cord blood donor. Furthermore, FISH analysis detected no FGFR1 rearrangement in the BM cells, which confirmed the achievement of cytogenetic remission. However, she died in July 2016 on day 73 after the second CBT due to the acute exacerbation of interstitial lung disease.

Literature selection

We collected cases from a computerized search of the medical literature using PubMed. We searched with words (#1: FGFR1 OR 8p11 and #2: leukemia OR lymphoma OR myeloproliferative), and extracted literature that satisfied both #1 AND #2 on January 2017. The following clinical data were obtained from the literature. This study was conducted in approach with the Declaration of Helsinki.

Statistical methods

Survival time was calculated by the Kaplan-Meier method and compared using the log-rank test. All statistical analyses were performed with EZR version 1.30 (Saitama Medical Center, Jichi Medical University), which is a graphical user interface for R (The R Foundation for Statistical Computing, version 3.2.2). More precisely, it is a modified version of R commander (version 2.2–0) that includes statistical functions that are frequently used in biostatistics [12].

Results

Review of the literature

We obtained 356 articles after a primary search of the title and abstract. We selected cases that provided sufficient information on survival times from diagnosis or allogeneic HSCT. Cases without a confirmation of FGFR1 abnormalities by FISH or RT-PCR, even if clinically
diagnosed as EMS, were excluded. As a result, we identified 39 reports (44 cases) with hematological neoplasms associated with FGFR1 abnormalities [4–43]. Finally, 45 cases including our patient were included in the following analyses.

**Patient characteristics**

The clinical characteristics of the patients are summarized in Table 1. The median age at diagnosis was 46 years (range, 0–87), and 13 patients (28.9%) were older than 60 years. The primary diagnosis was MPN (n = 13), AML (n = 12), T-ALL/LBL (n = 10), B-ALL/LBL (n = 4), acute leukemia (AL) of ambiguous lineage (n = 5), and myelodysplastic syndrome (MDS) (n = 1). In a RT-PCR analysis, ZNF198 (n = 12) was the most frequently detected FGFR1 translocation partner gene.

**Survival analysis**

The OS data from diagnosis were available for 41 patients. The median follow-up time was 10 months (range, 1–66 months). The 1-year OS from diagnosis was 43.1% (95% confidence interval [CI], 26.8–58.4%) (Figure 1A). There was no statistically significant difference in OS between patients with (n = 11) and without (n = 30) ZNF198-FGFR1 translocation (P = 0.472) (Figure 1B).

**Patients with MPN and MDS**

Fourteen patients with MPN (n = 13) or MDS (n = 1) were treated with hydroxyurea (HU) (n = 7), HU and prednisolone (n = 1), HU and mercaptopurine (n = 1), or imatinib (n = 1). The remaining 4 were observed without treatments. Of the 13 patients with clinical data regarding the duration to blast phase (BP), transformation to BP, including AML (n = 5), B-ALL (n = 1), and T-LBL (n = 1), was observed in seven patients with a cumulative incidence of transformation to BP at 1 year of 46.2% (95% CI, 17.6–70.9%), considering death (n = 2) or allogeneic HSCT (n = 2) as competing risks (Figure 2). The median OS from the diagnosis was only 9.0 months (range, 2–66 months) in patients who did not receive allogeneic HSCT before the transformation to BP, whereas two of the three patients who received allogeneic HSCT before the transformation to BP achieved long-term remission of 30 and 57 months, respectively, after diagnosis.

**Patients with BP**

Next, we analyzed 38 patients including patients with BP at diagnosis (primary BP, n = 31) and those with BP transformation from MPN or MDS (n = 7). The diseases included ALL/LBL (n = 15), AML (n = 18), and AL of ambiguous lineage (n = 5).

Induction therapy data for BP were available for 32 patients. CR was achieved in 15 of 32 patients (46.9%), including 11 (84.6%) of 13 with ALL/LBL, 2 of 14 (14.3%) with AML, and 2 of 5 (40%) with AL of ambiguous lineage. There were no significant differences in CR rates between patients with primary BP (n = 27, 51.9%) and those with transformed BP (n = 5, 20%) (P = 0.338).

Survival data for BP were available for 35 patients, including 28 with primary BP and seven with transformed BP. The 1-year OS from BP was 29.8% (95% CI, 14.4–46.8%) (Figure 3A). There were no significant differences in 1-year OS from the diagnosis of BP between patients with primary BP and those with transformed BP (P = 0.859) (Figure 3B). With regard to the impact of treatment response on survival, the achievement of CR with a landmark at 2 months after diagnosis of BP was associated with a superior OS (40.0% [95% CI, 12.3–67.0%] vs. 26.0% [95% CI, 7.3–50.1%], P = 0.011 for 1-year OS from BP) (Figure 3C). In 14 patients with CR after induction therapy, four patients received allogeneic HSCT. Three of these four patients remained in remission at 7, 23, and 28 months, respectively, from the diagnosis of BP.

**Patients receiving allogeneic HSCT**

Allogeneic HSCT was performed in 14 patients: 3 with MPN, three with transformed BP, and eight with primary BP. Survival data from diagnosis, BP, and allogeneic HSCT were available for 10, 8 and 13 patients, respectively. The 1-year OS from diagnosis, BP, and allogeneic HSCT was 68.6% (95% CI, 30.5–88.7%), 56.2%
(95% CI, 14.7–84.2%), and 61.5% (95% CI, 30.8–81.8%), respectively (Figure 4A). The hazard ratio of mortality for allogeneic HSCT as a time-dependent covariate was 0.34 (95% CI, 0.08–1.51, \( P = 0.15 \)). With regard to the effect of disease status at HSCT, the 1-year OS from allogeneic HSCT of those with MPN (n = 3), those in CR of BP (n = 3), and those in NR of BP (n = 7) was 66.7% (95% CI, 5.4–94.5%), 100% (95% CI, 100–100%), and 42.9% (95% CI, 9.8–73.4%), respectively (Figure 4B).

**Discussion**

The aim of the present study was to evaluate the clinical outcomes and therapeutic strategies for hematological malignancies associated with \( FGFR1 \) rearrangement. In previous reports, EMS was reported to have a poor prognosis [3]. However, no previous report has provided detailed survival data from patients with the \( FGFR1 \) rearrangement. We conducted a retrospective review of a case series from the published literature. This study showed that the prognosis in patients with \( FGFR1 \) rearrangement was poor, but allogeneic HSCT would provide a clinical benefit in selected patients.

Compared to a previous report of 65 cases with EMS [3], 16 of whom were also included in the current analysis, this study showed high CR rates (46.9% vs. 27%) and a prolonged survival from allogeneic HSCT (median OS; not reached vs. 15 months) in patients with \( FGFR1 \) rearrangement. A possible explanation for this difference is that the previous study included cases reported between 1970s and 2000s, and improvements in induction therapies and supportive care might have improved the treatment outcome. On the other hand, the 1-year OS was not improved in this study (median OS; 11 months vs. 15 months). This was possibly due to the lower proportion of cases diagnosed as MPN in the current study (31% vs. 41%).

There is no established induction therapy for myeloid/lymphoid neoplasms with \( FGFR1 \) rearrangement, and therefore various treatments, including ALL and AML regimens, were used in this cohort. The present study showed that the CR rate of patients with ALL/LBL associated with \( FGFR1 \) rearrangement was equivalent to that of patients with de novo ALL/LBL, but the CR rate for AML and AL of ambiguous lineage was extremely low. These results suggested that \( FGFR1 \) rearrangement may be associated with chemoresistance in myeloid neoplasms. In a mouse model, BCR-\( FGFR1 \)-driven AML blasts remarkably increased the phosphoactivation of FLT3, MAPK3/1,
and STAT/3/5, as well as the expression of anti-apoptotic BCL2 [44]. For myeloid neoplasms with FGFR1 rearrangement, it is worth trying to target these molecules. In fact, one patient with AL of ambiguous lineage showed chemotherapy resistance, but achieved CR with the use of ponatinib [4]. Sorafenib, a multikinase inhibitor, has also been used for the treatment of myeloproliferative disorders with FGFR1 abnormalities, but the hematological response was short term [45]. TKIs alone may be insufficient to produce a sufficient response, and therefore TKIs could possibly serve as a bridge to allogeneic HSCT.

In this study, approximately half of the patients who were diagnosed with MPN transformed to BP within one year. However, two of three patients who received allogeneic HSCT before BP achieved long-term remission. These results suggest that allogeneic HSCT may improve the prognosis of patients with MPN associated with FGFR1 abnormalities, when performed before BP transformation.

This study has several limitations. For example, this study included a small number of patients with heterogeneous characteristics. The retrospective nature of this study also limits the reliability of statistical analyses. In addition, a potential publication bias can not be excluded, since patients who successfully underwent allogenic HSCT might have been more likely to be reported. However, hematological malignancies associated with FGFR1 are very rare, and our analysis provides the current best evidence that may help physicians.

In conclusion, we demonstrated that patients with FGFR1 rearrangement were associated with a poor prognosis. The available literature suggested that allogeneic HSCT could provide a potential clinical benefit in selected patients. The further accumulation of clinical data is needed to determine whether allogeneic

Figure 3. OS from the diagnosis of BP in all patients (n = 35) (A). OS from BP in patients who transformed to BP with a previous diagnosis of MPN or MDS (n = 7) and in those with a diagnosis of primary BP (n = 28) (B). OS from BP grouped according to the response with a landmark of 2 months after BP (C).
HSCT could be an optimal therapeutic approach and improve clinical outcomes.

**Disclosure statement**

The authors declare that they have no conflict of interest.

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