Introduction

- Genetic abnormalities such as deletions in \textit{IKZF1} (70%), \textit{PAX5} (35%), and \textit{CDKN2A} \textit{p} (30%) detected in Philadelphia chromosome-positive (Ph+) B-ALL.\(^1\)
- Ph+ B-ALL patients with \textit{IKZF1} deletion have poor prognosis when treated with imatinib- or dasatinib-based regimens.\(^2\)
- \textit{IKZF1} plus- (\textit{IKZF1} deletion with additional co-occurring deletion(s)) was also shown to be prognostic in GIMEMA-led trials (dasatinib plus blinatumomab).\(^3\)
- The molecular determinants for clinical outcomes in ponatinib-treated patients remain unknown.

Aim

We systematically analyzed genetic alterations in adult Ph+ B-ALL patients treated with Hyper-CVAD plus dasatinib,\(^4\) Hyper-CMAD plus dasatinib,\(^4\) or Hyper-CVAD plus ponatinib\(^5\) and investigated the prognostic significance of the genetic alterations.

Methods

Figure 1. CONSORT diagram.

Results

Figure 2. Landscape of genetic alterations in 105 cases of Ph+ B-ALL.

Conclusion

\textit{IKZF1} plus status was an independent prognostic factor for outcome in patients with Ph+ B-ALL treated with Hyper-CVAD plus ponatinib. In contrast, in patients treated with dasatinib-based regimen, the outcomes were poor across all molecular subsets.

Reference


Conflict of interest / disclosures

Takeda Pharmaceutical Company (EJ).