Reply to the letter by Yang et al. RE: acute myeloid leukemia associated with *FGFR1* abnormalities

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To the Editor

We would like to thank for Yang and his colleagues' insightful comments on our work. In our previous manuscript, we described three cases of 8p11 myeloproliferative syndrome (EMS) that initially presented as acute myeloid leukemia (AML). Two of these—t(8;13)(p11.2;q12) and t(8;9)(p11.2;q33)—were cytogenetically identified, but one with variant inv(8)(p11.2q13) was detected only by fluorescence in situ hybridization (FISH) [1]. A number of other EMS cases presenting as AML have been reported, and new therapeutic strategies are being introduced [2–4]. Many EMS cases are initially recognized by conventional cytogenetics. FISH can confirm the majority of EMS and determine submicroscopic deletions, but in a few cases did not detect the rearrangement [5]. Identification and characterization of fusion genes are clinically important to the understanding of disease pathogenesis and selection of the monitoring method [3]. FISH using home-brew probes and 5' rapid amplification of cDNA end PCR with sequences analysis have been used [6, 7]. Long-distance PCR and long-distance inverse PCR were introduced in a previous report as diagnostic tools capable of identifying the corresponding partner genes of *FGFR1* [8].

Clearly, international collaborative studies, including distinct ethnic entities, aimed at elucidating poorly understood aspects of EMS such as incidence, clinical and laboratory manifestation, genetic aberrations including submicroscopic deletion and their association with pathogenesis and prognosis, are warranted.

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