INTRODUCTION

Acute lymphoblastic leukemia (ALL) is a clonal malignant disease of the bone marrow in which early lymphoid precursors proliferate and replace the normal hematopoietic cells of the marrow ***(Pui et al., 2004).***

The complexity of the leukemogenic process, together with our limited understanding of the biology of this disease, presents a challenge to developing novel therapeutic approaches ***(Cobaleda et al., 2000).***

Precursor B-cell acute lymphoblastic leukaemia (B-ALL)/ Lymphoblastic lymphoma occurs primarily in children and comprises about 80–85% of ALL cases ***(Jaffe et al., 2001).***

Recently, the World Health Organization (WHO) characterized cytogenetic subgroups of B-ALL by the t(9;22)(q34;q11.2), t(11q23;var), t(12;21)(p13;q22), t(1;19)(q23;p13.3) [abbreviated to t(1;19)], hypodiploidy and hyperdiploidy (>50 chromosomes) ***(Jaffe et al., 2001).***

The second most common translocation observed in approximately 6% of precursor B-ALL cases and 20–25% of all pre-B-ALL cases is the t(1;19) ***(Troussard et al., 1995 and Wiemels et al., 2002).***

The (1;19) translocation involves breakpoints in the PBX1 (Previously cited as prl) gene on chromosome 1q23 and in the E2A (also known as TCF3) gene on chromosome 19p13.3 ***(Mellentin et al., 1990 and Hunger et al., 1991).***

Detection of the cytogenetic subgroups in B-ALL is important for prognostic stratification into low, intermediate and high risk groups, and correspondingly vital for the selection of appropriate therapeutic regimens ***(Brandon et al., 2005).***

Typically, conventional cytogenetic, Fluorescence In Situ Hybridization (FISH) and/or Reverse-Transcription Polymerase Chain Reaction (RT-PCR) are utilized for the detection of the (1;19) translocation ***(Brandon et al., 2005).***