A reciprocal translocation between chromosomes 9 and 22 creates oncogenic BCR/ABL fusion in the breakpoint region of the derivative chromosome 22. The aim of this study was to evaluate the importance of atypical fluorescence in situ hybridization (FISH) signal patterns in pediatric and adult acute lymphoblastic leukemia (ALL) cases. We evaluated t(9;22) translocation in 208 cases with ALL (294 tests), including 139 childhood and 69 adult cases by FISH technique using BCR/ABL extra signal (ES) probe. FISH signal patterns observed in pediatric ALL cases were as follows; Major-BCR/ABL (M-BCR/ABL) (1.4%), minor-BCR/ABL (m-BCR/ABL) (3.6%), trisomy 9 (4.3%), trisomy 22 (4.3%), trisomy or tetrasomy of both chromosomes 9 and 22 (2.9%), monosomy 9 (1.4%), monosomy 22 (0.7%), ABL gene amplification (1.4%), derivative chromosome 9 deletion (1.4%), and extra copies of the Philadelphia chromosome (1.4%). FISH signal patterns observed in adult ALL cases were as follows; M-BCR/ABL (5.8%), m-BCR/ABL (11.6%), two different cell clones with major and minor BCR/ABL signal pattern (2.9%), extra copies of Philadelphia chromosome (4.3%), derivative chromosome 9 deletion (1.4%), trisomy 9 (2.9%), tetraploidy (1.4%), monosomy 9 (1.4%), trisomy 22 (1.4%), and coexistence of both trisomy 22 and monosomy 9 (1.4%). Trisomy 9, trisomy 22, and polyploidy of chromosomes 9 and 22 were specific atypical FISH signal patterns for childhood B cell acute lymphoblastic leukemia (B-ALL) patients. However, monosomy 9 and ABL gene amplification were highly specific for childhood T cell acute lymphoblastic leukemia (T-ALL) patients. Our report presents the correlation between atypical FISH signal patterns and clinical findings of a large group of ALL cases.