MLL gene rearrangements are seen in both B-ALL and T-ALL, and in acute myelogenous leukemia (AML), especially therapy-related AML. Infantile B-ALL, generally pro B-ALL type, has a poor prognosis and is frequently associated with rearrangement of the MLL gene. In addition, myeloid and natural killer cell antigens, such as CD15 and CD56 are frequently expressed. Reciprocal translocations are most common and the following are most common in decreasing order of frequency: t(4;11)(q21;q23), t(11;19)(q23;p13.3) and t(9;11)(p21–22;q23). Clonal rearrangements of MLL gene are usually seen in infants' leukemias as well as a majority of therapy-related leukemias, in which it always represents a hallmark for aggressive disease, poor clinical outcome and bad response to treatment with high risk of relapse. Those rearrangements include chromosomal translocations, internal gene duplications, chromosome 11q deletions or inversions, MLL gene insertions into other chromosomes, and insertion of genetic material into the MLL gene. In gene expression profiling studies, MLL+ precursor B-ALL shows a profile consistent with an early hematopoietic progenitor that is distinct from conventional B-ALL and AML, suggesting that MLL+ precursor B-ALL represents a clinically and molecularly unique entity. These cases reveal a mature B-ALL immunophenotype without FAB-L3 morphology and MYC rearrangement and were reported in children, very few of these present with MLL gene rearrangements and especially the t(9;11) translocation.

In gene expression profile studies, MLL+precursor B-ALL shows a profile consistent with an early hematopoietic progenitor that is distinct from conventional B-ALL and AML, suggesting that MLL+precursor B-ALL is a clinically and molecularly unique entity. These MLL+pro B-ALL are rarely seen in adults specially t(9;11). B-ALL with t(9;11) are reported in infants but only rarely described in adult. These reported cases had mature B-ALL subtype on morphology and/or immunophenotyping and presented with an aggressive manner. To our knowledge the index case is the first case of pre-B-ALL with t(9;11) in an elderly female who had presented with a relatively indolent course of disease.

Infantile B-ALL associated with 11q23 chromosomal abnormality and rearrangement of the MLL gene, frequently show t(4;11) and t(11;19). The t(9;11) translocation, although the most common MLL rearrangement associated with de novo AML with monocytic differentiation (FAB M4 and M5) and DNA-topoisomerase inhibitor therapy related AML, is only rarely seen in precursor B-ALL. The t(9;11) translocation most commonly results in a fusion of the MLL and AF9 genes and the resultant MLL/AF9 fusion gene is involved in myeloproliferation and leukemogenesis. Various studies in mice and human have suggested t(9;11) fusion gene develop predominantly AML with only a minority show features of ALL. However, the exact mechanism of leukemogenesis is still unclear, as is its role in MLL positive B-ALL. It has also been