childhood ALL (2% to 6%), and rarely in acute myeloid leukemia (AML)[2] . The BCR-ABL fusion product is an abnormal tyrosine kinase that plays the role of a driver mutation in the leukemogenesis of Ph positive hematological malignancies[3]. In patients with ALL, the presence of Ph chromosome is associated with a worse outcome, and usually leads to a relapse within 2 years in the absence of an aggressive treatment. While the occurrence of M-BCR-ABL is equal in both childhood and adult Ph-positive ALL than in adult cases[2].

The translocation of the same part of a donor to several recipient chromosomes is referred to as a jumping translocation (JT) and was first described in a case of Prader–Willi syndrome[4]. Although the aberrations of chromosome 1 are very common in most hematologic malignancies including multiple myeloma, myeloproliferative disorders, and myelodysplastic syndrome[5], JTs of chromosome 1 are rare chromosomal aberrations. The most common donor region of chromosome 1 is the region distal of 1q21[6-8]. The translocated portion is usually attached to the telomeric ends of the acceptors[9]. The derivative of 1q may be either a ''primary'' or ''secondary'' chromosomal abnormality[10]. The presence of an extra copy of 1q provides a proliferative advantage and drives clonal evolution at the genetic level and is clinically associated with disease progression and poor prognosis[11-13].

The aberrations of 1q may be triggered by various mechanisms such as viral infections, immunodeficiency, DNA hypomethylation, and chromosomal instability. In case of viral infections, there may be fusions between homologous virus-related sequences of different chromosomes. 1q instability and hypomethylation is observed in cases of multiple myeloma[9]. Regardless of the underlying mechanism, the genetic abnormality is the same, which is a trisomy, partial trisomy, or tetrasomy of the donor region which in turn acts as a driver for the proliferation of the hematologic neoplasm[14].
Our case exhibited a novel cytogenetic finding in an ALL patient. Unbalanced translocations between chromosome 1q and chromosomes Y, 7, 14, 15, 16, and 19 have been previously reported[9], and the incidence of JT between 1q and chromosome 13 is extremely rare. There is only one report of the concurrent presence of Ph chromosome and 1q in ALL, the coexistence of a Ph chromosome with the partial trisomy of chromosome 1q involving chromosome 13 as the acceptor has never been reported in literature to the best of our knowledge.

In conclusion, dismal prognosis associated with synchronous presence of a Ph chromosome and JT leading to a partial trisomy of chromosome 1q may carry significant prognostic and therapeutic implications.