

# A Novel Jumping Translocation in a Case of Acute Myeloid Leukemia

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## Abstract

Jumping translocations (JTs) involve one donor chromosome segment and multiple recipient chromosomes in different cell lines in an individual. They are rarely observed as congenital abnormalities but have been reported in a variety of malignant disorders (myeloid and lymphoid disorders and solid tumors). This abnormality may reflect increased genomic instability and has been observed to be associated with aggressive disease. The long arm of chromosome 1 (1q) is most frequently observed as the donor segment. JTs result in trisomy for the donor segment and loss of material from the recipient chromosomes.

We report a case of JT with the long arm of chromosome 17 (17q) as the donor segment in a case of acute myelogenous leukemia in a 79 year-old female patient. In addition to a t(6;11)(q27;q23) which is present in all 20 cells analyzed, translocations between 17p22 and 7q23, 8q24.3, 17q23, 19p13.3, 20q13.3 and 21q22 were observed. The patient died shortly after the cytogenetic studies. To our knowledge, this is the first report in which 17q is the donor segment in a JT. The biological consequences of the JT in this case, partial trisomy for the long arm of chromosome 17 as well as loss of material from the recipient chromosomes, may have contributed towards the aggressive nature of the disease in this patient.

## Introduction

Jumping translocations (JTs) involve one donor chromosome segment and multiple recipient chromosomes in different cell lines in an individual. They have been observed in solid tumors and hematologic malignant disorders including follicular lymphomas, Burkitt lymphoma, multiple myelomas, acute promyelocytic leukemia and acute myelomonocytic leukemia (AMMoL). The donor segment most commonly involved in the JTs is the long arm of chromosome 1. However, other donor chromosome segments, including those from chromosomes 3, 7, 8, 9, 14 and 15, have been reported.

Jumping translocations in malignant disorders involve interstitial telomeric sequences. The biologic consequences of the unbalanced JTs are trisomy of the donor segment with amplification of genes on the duplicated segment and loss of material from the recipient chromosomes.

We report a case of JT with the long arm of chromosome 17 (17q21-qter) as the donor segment to six recipient chromosomes (7, 8, 17, 19, 20 and 21) in a case of AML.

## Conclusion

1. A jumping translocation involving the segment distal to 17q21 was observed as a secondary abnormality.
2. This JT results in trisomy of the segment 17q21 to 17qter and the loss of material from six recipient chromosomes.

## Discussion

1. To our knowledge, this is the first report where 17q is the donor chromosome.
2. The biological consequences of the JT in this case, partial trisomy for the long arm of chromosome 17 as well as loss of material from the recipient chromosomes, may have contributed towards the aggressive nature of the disease in this patient.

## Clinical Report and Karyotype

A 79 year old patient was diagnosed with AML. Cytogenetic analysis was performed on metaphase cells from 24 and 48 hour unstimulated cultures of peripheral blood lymphocytes. The patient died shortly after the diagnosis.

### KARYOTYPE

46,XX,t(6;11)(q27;q23)[1]/  
47,idem,+6[7]/  
47,idem,+6,der(8)t(8;17)(q24.3;q21)[7]/  
47,idem,+6,der(7)t(7;17)(p22;q21)[1]/  
47,idem,+6,der(17)t(17;17)(q21;q23)[1]/  
47,idem,+6,der(19)t(17;19)(q21;p13.3)[1]/  
47,idem,+6,der(20)t(17;20)(q21;q13.3)[1]/  
47,idem,+6,der(21)t(17;21)(q21;q22)[1]

