

i(17q) in myeloid malignancies

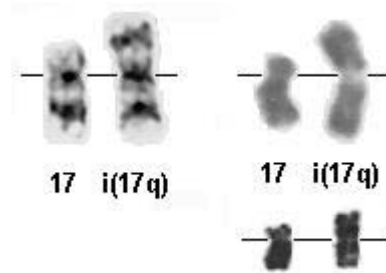
Identity

Note

an isochromosome 17 results in a loss of the short arm (17p) and duplication of the long arm (17q) leading to a single copy of 17p and three copies of 17q

an i(17q), usually observed in a complex karyotype, has been reported in solid tumors and in various types of hematological diseases: acute and chronic myeloid leukemias, acute lymphoid leukemias and chronic lymphoid leukemias, and Hodgkin and non-Hodgkin lymphomas

in [chronic myeloid leukemia](#), i(17q) is a frequent and well known secondary anomaly, either solely in 10% of cases, or with other additional anomalies, in at least another 10% of cases, in particular with [+8](#).



i(17q) G- banding (left) - Courtesy Jean-Luc Lai; and R- banding (right) - top: Editor, bottom: Courtesy Jacques Boyer.

Clinics and Pathology

Disease myeloproliferative/myelodysplastic diseases (MPD/MDS)

Phenotype / cell stem origin previous studies on isolated i(17q) have suggested this aberration was associated with chronic myeloid abnormalities with a high rate of progression to ANLL; a new clinico-pathological entity in which i(17q) is the sole abnormality has been reported in a mixed myeloproliferative disorder / myelodysplastic syndrome with an aggressive course; fifteen patients were included in this study classified as chronic myeloid malignancy at initial presentation: these features were not confirmed after a negative molecular BCR-ABL analysis in all cases studied (eleven patients)

Etiology i(17q) as sole cytogenetic aberration represents only 1% of cases in myeloid malignancies

Cytology a severe hyposegmentation of neutrophil nuclei (pseudo-Pelger Huet

neutrophils (PHH)) and a prominence of the monocyte/macrophage lineage has been noted; other studies have identified an association between hyposegmented neutrophils and loss of 17p (called [17p-syndrome](#)), always included in complex karyotypes; the i(17q) appeared to be a part of the malignant clone as demonstrated in cases available for a FISH analysis: all myeloid cell lines observed contained the abnormal i(17q), whereas none of the lymphocytes were affected

Prognosis by standard Kaplan-Meier analysis, the median survival was 2.5 years (range 0.85-5.25 years)

Genes involved and Proteins

Note the underlying molecular defect that produces the isolated i(17q) is unknown : breakage of the proximal p arm (17p11.2) with rejoining of both centromere-containing chromatids and subsequent inactivation of one centromere; breakpoints could involve important genetic material whose disruption could result in oncogene or tumor suppression gene deregulation

in understanding the specific i(17q) phenotype, loss of genes localized on 17p were suggested as [p53](#) (17p13.1); a direct correlation between p53 loss and PHH neutrophils was found in a series of MDS and ANLL with 17p- syndrome

External links

Other database [i\(17q\) in myeloid malignancies](#) [Mitelman database \(CGAP - NCBI\)](#)

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Citation

This paper should be referenced as such :

Bilhou-Nabera C . i(17q) in myeloid malignancies. Atlas Genet Cytogenet Oncol Haematol. February 2000 .

URL : <http://AtlasGeneticsOncology.org/Anomalies/i17qID1038.html>

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