individuals. About 86% of MDS cases are diagnosed at age ≥ 60 and less than 10% of patients are younger than 50. Recurrent cytogenetic abnormalities are present in 40% to 60% of cases. Cytogenetic analysis plays an important role, whether in the diagnosis or classification of the disease. Recently, the new international molecular prognostic score system (IPSS-M) reinforces the valuable prognostic contribution of cytogenetics in MDS in both survival and leukemic evolution. **Objective:** To report a chronic chloroform exposure as a trigger capable of inducing DNA damage. **Case report:** Chronic exposure to a genotoxic agent has been linked to MDS chromosomal abnormality. Among these, chloroform, and an inorganic halide derivative of hydrocarbons can induce the DNA lesion. A 24-year-old male patient with a marked history of 10-year chloroform exposure sought medical attention for anemia, fainting, and unintentional weight loss. He was diagnosed with Myelodysplastic Syndrome and refractory anemia with excess blasts (RAEB II) with initial tests: Hb = 5 g/dL; neutrophils=320/mm3; platelets= 18 x 109/L. Bone marrow aspiration showed 15% myeloblasts, immunohistochemistry profile showed CD34+, CD117+ in blasts and cytogenetic analysis showed a very complex karyotype: 45,XY,del(6)(q22),-7,add(9) (q12),del(12)(p11.2),del(17)(p13),del(20)(q12),add(21(p13)[17]/46, XY[3]. Chemotherapy with azacitidine was performed as a bridge to bone marrow transplantation, but the patient evolved into sepsis, dying eight months after diagnosis. **Conclusion:** The pathogenesis of MDS reflects the heterogeneity of the disease, thus, comprises multiple steps and includes factors that can be endogenous and/or exogenous. Therefore, this unusual case shows the extreme importance of investigating possible recurrent and/or occupational exposures in these patients combined with cytogenetics analyses.

https://doi.org/10.1016/j.htct.2022.09.350

CYTOGENETIC BICLONAL ABNORMALITIES IN PEDIATRIC AND ADULT PATIENTS WITH MYELODYSPLASTIC SYNDROME: CLINICAL IMPLICATIONS

BF Silva [a], GF Lima [a], EF Rodrigues [a], APS Bueno [b], AS Fonte [c], VL Lovatel [a], TS Fernandez [a]

[a] *Laboratório de Citogenética, Instituto Nacional de Câncer (INCA), Rio de Janeiro, RJ, Brasil*
[b] *Instituto de Puericultura e Pediatria Martagão Gesteira (IPPMG), Universidade Federal do Rio de Janeiro (UFRJ), Rio de Janeiro, RJ, Brasil*
[c] *Hematologia Pediátrica, Hospital Federal da Lagoa, Rio de Janeiro, RJ, Brasil*

Objective: Myelodysplastic syndrome (MDS) comprises a heterogeneous group of clonal hematopoietic stem cell diseases. MDS is characterized by dysplasias, cytopenias and an increased risk of acute myeloid leukemia (AML) evolution. MDS has a higher incidence in elderly patients, and is rare in pediatric patients. Cytogenetic analysis is an important laboratory tool for diagnosis, prognosis and clinical decision-making for MDS patients. Chromosome alterations can be detected in 30-50% in MDS. The most frequent chromosomal abnormalities are: del(5q), del(7q)/-7, +8, del(11q), del(12p), del(17p), del(20q) -Y. Rare cytogenetic abnormalities usually are considered as intermediate group in the Revised International Prognostic Scoring System (IPSS-R), as the cytogenetic biclonal abnormalities. However, this group of chromosomal abnormalities may be reported to help to elucidate its clinical implications in *de novo* MDS. The aim of this study was to report rare cases of cytogenetic biclonal alterations in pediatric and adult patients with *de novo* MDS and their clinical implications. **Materials and methods:** This study included 7 patients with MDS, being 3 pediatric patients and 4 adult patients from a cohort of 1056 *de novo* MDS patients. Chromosomal and clinical studies were carried out in 1056 MDS patients, being 200 pediatric patients and 856 adult patients, between 2000-2022. Cytogenetic analyses were performed in bone marrow cells by G-banding and fluorescence *in situ* hybridization (FISH). **Results:** A total of 7 (0.66%) patients with *de novo* MDS (7/1056) had biclonal cytogenetic abnormalities, being 4 (0.48%) adult patients (4/856), and 3 (1.5%) pediatric patients (3/200). The mean age of adult patients was 67.5 (60-73 years old), and pediatric patients were 10 (6-13 years old). Most of the patients (57%) were classified as advanced stages (MDS-EB-1, MDS-EB-2 and MDS-EB-t) and dysplasias in more than one lineage (71%). The cytogenetic biclonality in adult patients involved the clones with the chromosomal alterations: del(11q),del(20q)/del(12p); Y/del(17p); del(11q),+21/del(6q); i(17q)/hyperdiploidy karyotype; and in pediatric patients del(17p)/del(12p); +X,+8/add(7p); +8/+21. **Discussion:** Biclonal cytogenetic alterations are uncommon findings, with a frequency between 1.6%-6.5% in MDS. Although we studied a large cohort of MDS patients the frequency of these alterations was lower than literature (0.66%). It is important to note that most studies included secondary or therapy-related MDS and AML which generally have a higher chromosomal instability. In our results, the chromosomal instability in these clones involved common cytogenetic alterations: del(11q), del(12p), del(17p) and +8, but the prognostic value of this distinct cytogenetic clones still needs to be elucidated. The mechanisms for the development of unrelated cytogenetic clones remain to be elucidated, especially because it is unclear if these clones are in fact two independent clones, representing a true biclonality, or a clonal evolution derived from a common precursor with a submicroscopic molecular genetic change. **Conclusion:** Our study shows new insights into biclonal cytogenetic abnormalities in *de novo* MDS, demonstrating for the first time, from a large cohort of *de novo* MDS patients, the frequency of cytogenetic biclonality in pediatric and adult patients.

https://doi.org/10.1016/j.htct.2022.09.351

EVOLUÇÃO DA SÍNDROME DE FOURNIER, EM PACIENTE COM SÍNDROME MIELODISPLÁSICA: RELATO DE CASO

VMS Morais [a,b,c], LEP Pereira [b], JASA Barros [b], VG Silva [b], CGS Duarte [b], DWS Araújo [b], FMB Lavra [b], ARDS Ayres [b], EG Silva [a,b], KMLP Silva [a]