

A Rare Case of t(8;16)(p11.2;p13.3) and del(17p) That Shows Both Acute Myeloid Leukaemia and Chronic Lymphocytic Leukaemia at the Same Time

E.ÇALLIKU¹, A.PEROLLA¹, V. SEMANAJ³, D.ROKO², T.DEDEJ-KURTI³, S.GRAZHDANI⁴, A.BABAMETO², A. IVANAJ¹

1. UHC "MOTHER TERESA", SERVICE OF HEMATOLOGY, TIRANA, ALBANIA

2. UHC "MOTHER TERESA" DEPARTMENT OF GENETICS, TIRANA, ALBANIA

3. NETWORK LABORATORY, TIRANA, ALBANIA

4. KORÇA' S HOSPITAL, ALBANIA

OBJECTIVES

- To demonstrate a rare case of t(8;16)(p11.2;p13.3) and del(17p) showing both Acute Myeloid Leukaemia and Chronic Lymphocytic Leukaemia at the same time.

CONCLUSIONS

This example shows how important it is to have a full diagnostic assessment of patients who have unusual haematologic symptoms. Having AML and CLL at the same time, especially when they have high-risk cytogenetic characteristics, makes diagnosis and treatment very difficult. It is important to keep studying the clonal linkages and molecular causes of these cancers in order to improve outcomes and customise treatment for these difficult cases

INTRODUCTION

it is quite uncommon for a person to have both acute myeloid leukaemia (AML) and chronic lymphocytic leukaemia (CLL) at the same time, especially when they first show up and haven't had any cytotoxic medication before. it is even less likely to have genetic problems at the same time, including t(8;16)(p11.2;p13.3) and del(17p), which are both linked to aggressive disease types and bad outcomes. Some studies say that these two cancers may be caused by the same leukemogenic pathways, clonal evolution, or a problem with the same hematopoietic stem cell.

METHODS

CASE REPORT:

A patient came in with indications of pancytopenia and blood that looked strange on the outside. a blood smear and bone marrow aspirate showed two types of cells: one that looked like AML blasts and another looking like tiny mature lymphocytes [fig.1]. Immunophenotyping found two different clonal populations: an AML clone (cd33+, cd64+, cd123+, HLA-DR +, cd11c+, cd117+, cd56+) [fig.3] and a residual CLL clone (cd19+, cd5+, cd23+, kappa-restricted, cd22 weak) [fig.4]. Cytogenetic analysis showed t(8;16)(p11.2;p13.3), and FISH analysis was positive for del(17p), which means that the TP53 pathway was broken [fig.2]

RESULTS

This is an unusual example of de novo AML and untreated CLL happening at the same time, with each having its own clonal origin. the cytogenetic profile of the AML clone was complicated and aggressive. The CLL clone stayed clinically indolent, but it had the high-risk del(17p) change.

The patient had induction chemotherapy for AML, but the outlook was still bad because of the high-risk genetics and disease burden.

Figure 1. Bone marrow smear showing blasts and mature lymphoid cells

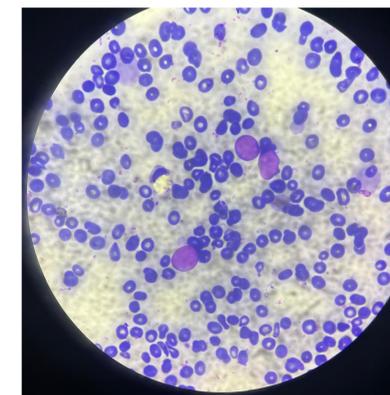


Figure 2 [a,b]: Karyotype and FISH

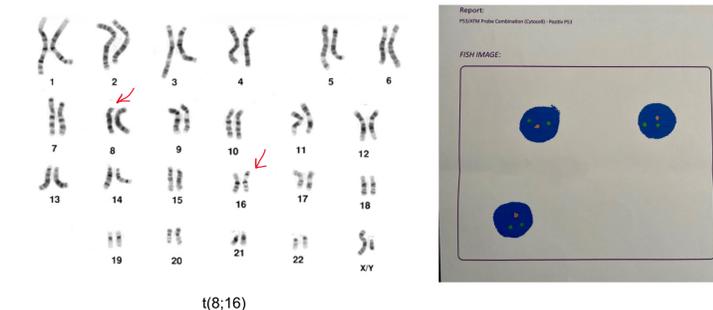


Figure 3:Flow cytometry showing AML clone

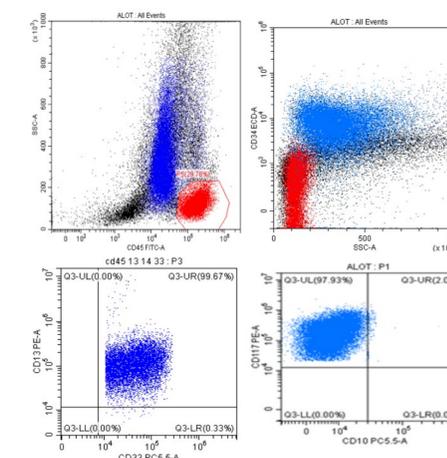
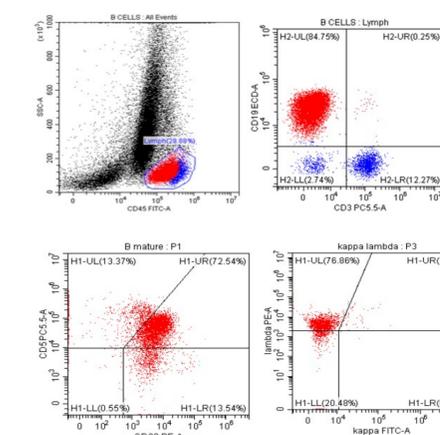


Figure 3:Flow cytometry showing CLL clone



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DISCLOSURES

No disclosures