Coexistence of myeloblasts and B-CLL cells in peripheral blood

Coexistence de myéloblastes et de cellules B-LLC dans le sang périphérique

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B-chronic lymphoid leukemia (B-CLL) and acute myeloid leukemia (AML) are two separate diseases and they are clonally unrelated. [1] The coexistence of these two leukemias has been rarely described in the literature.[2] Most of these cases have been reported to occur after treatment of CLL with cytotoxic drugs suggesting that AML may be a secondary leukemia also knows as therapy-related AML. [2]

This letter reports a case of a concomitant detection of myeloblasts and B-CLL cells in a patient and highlights the usefulness of cytomorphology and CD45/SSC dot plot in the recognition and discrimination of the 2 neoplasm cell populations.

An 87-year-old male with medical history of diabetes and high blood pressure presented to the department of hematology with complaints of fatigue, pallor and weakness for the investigation of a chronic lymphocytosis.

His blood tests showed hemoglobin=5 g/dL; white blood cell= 18.62 10^9/L; platelet count 70 10^9/L and slight lymphocytosis ranging from 6 to 15 10^9/L persisting for about 8 months.

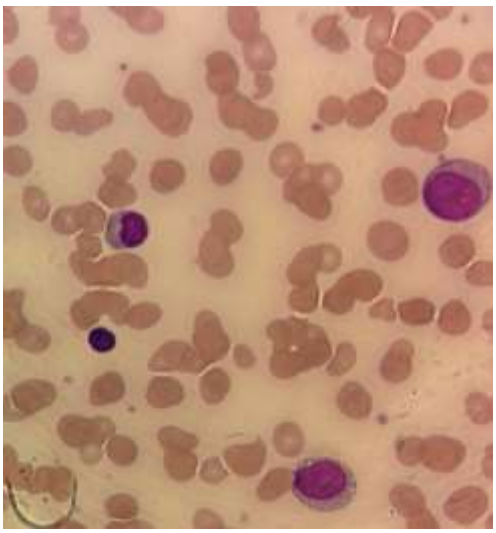

Figure 1. The peripheral bloods smear showing two circulating blast cells (on the right) with two small mature lymphocytes (on the left)

The peripheral blood (PB) smear showed small lymphocytes with scant cytoplasm and well condensed chromatin, and large blasts with high nuclear cytoplasmic ratio and fine nuclear chromatin, containing prominent nucleoli, however smudge cells were not seen (Figure 1).

The flow cytometric analysis of peripheral white blood cells was performed; and CD45 vs SSC Dot plot revealed two separate populations: large blasts with dim CD45 expression located in the so-called "Bermude Area" [3] and small lymphocytes with high and heterogeneous CD45 expression (Figure 2).

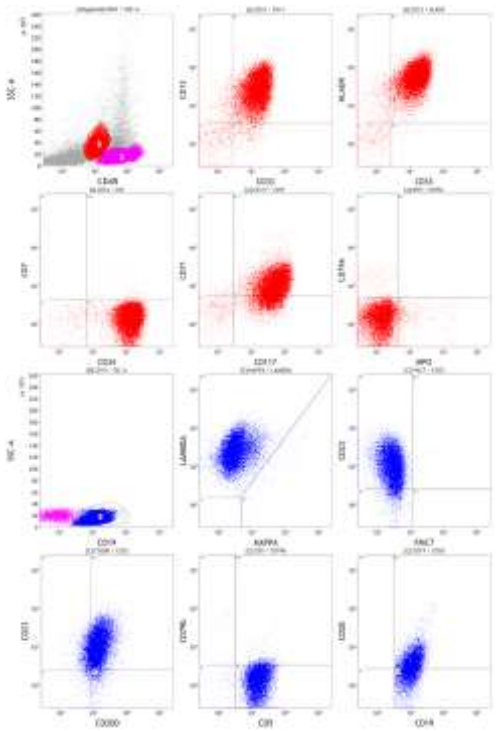

Figure 2:
Peripheral nucleated cells evaluated by flow cytometry. Myeloblasts (red color) was characterized by the co-expression of CD33, CD13, CD117 and HLA-DR, whereas B-lymphocytes (blue color) were identified by the heterogenous expression of CD45 and were characterized by typicall CLL immunophenotype: CD19+, CD5+, CD23+, CD20 + (dim), CD79a -, CD200+ and lambda light chain restriction (dim).

The European Research Initiative on CLL (ERIC) & European Society for Clinical Cell Analysis (ESCCA) working group proposed that the application of CD45 must be left to the individual laboratory preference (i.e. "not recommended") because this marker is used

for identification of leukocyte subsets and provides a backbone to many gating strategies but is not essential to identify CLL.[4]

In our laboratory, the reason of adding CD45 to the lymphoproliferative disease antibody panel is that some acute lymphoblastic leukemia, so-called "ALL/L1 subtype" in the FAB classification, [5] can have small blastic cells with clomped chromatin. These cells are counted by a hematology counter as lymphocytes and can mislead to a diagnosis of CLL in the elderly, while the CD45 vs SSC dot plot allows us to separate the blasts from the mature lymphocytes.

In this case report, the blastic population expresses immaturity markers (CD34 and HLADR), and myeloid markers (CD117, CD13 and CD33) (figure 2), however, the myeloperoxidase (MPO) testing was negative by both cytochemistry and flow cytometry. Whereas the lymphocyte population was considered to be CLL cells by the expression CD19, CD20, CD5, CD23, CD200 and lambda light chain restriction and lack of expression of CD79a and FMC7 and CD34.

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