To the Editor:

In the August 1, 1991 issue of Blood, San Miguel et al. published a report of 41 acute leukemia cases secondary to a myelodysplastic syndrome (MDS). Two were classified as M6 French-American-British (FAB) subtype on morphologic aspect, but the only erythroid immunologic marker tested, ie, glycophorin A, was negative. This characteristic, owing to the fact that glycophorin A is a relatively mature erythroid marker, may be responsible for an underestimation of this type of MDS transformation.

We report two cases of M6 acute myeloid leukemia (AML) after MDS in which we tested glycophorin A (Immunotech, Marseille, France) and more immature erythroid markers such as CD71 (Ortho Diagnostic Systems, Raritan, NJ), CD34 (Sera-Lab, Biosys, France), and HLA-DR (Ortho). Immunologic phenotyping was performed on separated cells from bone marrow aspirate by an indirect immunofluorescent method and analyzed by flow cytometry (FACScan; Becton Dickinson, Mountain View, CA).

The expression of glycophorin A, CD34, and HLA-DR on a few cells from our patients is presented in Table 1. In both cases, CD71 is more expressed than any other marker.

This phenotype corresponds to the last level of normal erythroblastic precursors differentiation (PIV) described by Terstappen et al., ie, decreased CD34 and increased CD71 expressions. Glycophorin A is expressed on more mature erythroblasts present in bone marrow aspirations. For this reason, in acute leukemia secondary to MDS, a more systematic search for CD34 and CD71 expression could be interesting, particularly in undifferentiated forms. This behavior should correct a possible underestimation of M6 acute MDS transformation.

MARC MAYNADIE
RENE-OLIVER CASASNOVAS
FRANCOIS BAILLY
BRUNO COUDERT
PAULE-MARIE CARL1

REFERENCES

RESPONSE

We appreciate the letter and comments of Maynadie et al. We do agree about the need for using other immunologic markers for the detection of erythroid blast cells because glycophorin A is a relatively mature marker. We have previously reported on the expression of CD71 in M6 leukemias. Nevertheless, this is not a specific erythroid marker such that its positivity in AML is not diagnostic for erythroleukemia. Our present policy is to include in the panel of monoclonal antibodies for such diagnosis not only glycophorin A and CD71, but also CD36 to cover the broadest spectrum possible within erythroid differentiation in view of the absence of specific early erythroid markers.

J.F. SAN MIGUEL
J.M. HERNANDEZ R.
GONZALEZ-SARMIENTO
M. GONZALEZ
I. SANCHEZ
A. ORFAO
M.C. CANIZO
A. LOPEZ BORRASCA

REFERENCE

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Immunophenotype in erythroleukemia secondary to myelodysplastic syndrome [letter; comment]

M Maynadie, F Bailly, RO Casasnovas, B Coudert and PM Carli