We wish to make several remarks about the paper by Cranendonk et al.: Evaluation of the use of the Hemalog D in acute lymphoblastic leukemia and disseminated non-Hodgkin’s lymphoma in children [Acta haemat. 71: 18–24 (1984)], which deals with automated cytochemistry performances in acute leukemias. The subject, indeed, is not a worthless one: automated leukocyte differential devices, in fact, are more and more diffused among hematology laboratories, so that the awareness of their qualities and defects deserves considerable interest.

In that paper, the authors report their results concerning Hemalog D differential counts in 79 children with acute lymphoblastic leukemia (ALL) and 18 children with disseminated non-Hodgkin’s lymphoma; only 25 of them, however, were studied since the onset of their disease. The authors generalize their conclusions, claiming that the Hemalog D seems inadequate in diagnosis and follow-up of leukemic patients. Over the last 4 years we have been able to analyze a large number of leukemic blood samples with the Hemalog D [1, 3]. We would like to report very briefly our experience, which is indeed quite different.

As far as reliability and accuracy are concerned, the Hemalog D appears entirely capable of detecting and signaling pathological samples: with one exception, in 43 cases of acute leukemia adequate flags (increase in LUCs or HPX, abnormal Remainder, LR or LPX warnings) were constantly provided, allowing an immediate microscope check. The only exception was represented by a patient with acute promyelocytic leukemia, in which no alarm signals were reported in the printout: the peroxidase display plot, however, was clearly abnormal, and the association with pseudo-eosinophilia, marked leukocytosis, decreased hemoglobin and platelet count would have pointed out that sample as abnormal in any case.

The negative results obtained by Cranendonk et al. may be explained in part by considering several details of their study. (1) These authors were interested only in lymphoblastic leukemias; the Hemalog D, owing to the peroxidase staining it carries out automatically in each blood sample analyzed for routine differential, is obviously cleverer in recognizing and characterizing non-lymphoid leukemias. (2) The great majority of the patients studied at diagnosis (19 out of 25) had ALL-LI subtype: the small LI lymphoblasts, of course, are more difficult to identify, even at the microscope, because of their size and morphological features. Our results in adult ALL are very different [1, 3]: none out of 15 ALL patients (5 with LI, 8 with L2 and 2 with L3) had normal LUC value at diagnosis. The LUC value was higher in L2 and L3 patients, and the display pictures were clearly distinct in the three FAB subtypes. (3) The reference ranges adopted by the authors seem too wide, even for a
pediatric population, especially with respect to LUC and HPX values. According to the majority of investigators [5], in our laboratory normal LUC value is considered below 2.5%, and normal HPX value 2.0%. In the two cases Cranendonk et al. describe as examples of normal Hemalog D printouts in leukemic patients the LUCs were 4.7 and 3.0%, respectively; both patients, moreover, showed leukopenia (3.1 and 2.9 × 10^9/1), which is a sufficient reason for microscopic check (and we do not know anything about their hemoglobin and platelet count). Thus, such cases would probably not have escaped from a routine screening in most laboratories. To sum up, the Hemalog D has proven totally reliable, in our hands as well as in most reported studies [4, 6, 7], in pointing out abnormal samples from leukemic patients. Numerical results by the Hemalog D, on the other hand, are no longer useful as far as diagnosis and classification of acute leukemias are concerned. Peroxidase display plots, however, provide a visual representation of the leukemic cell distribution, which is a source of useful information for skilled observers. Such a ‘morphological approach’ to the cell distribution pattern provides valuable clues in many fields, such as classification of leukemias according to FAB subtypes [1, 3, 6], prediction of induction therapy outcome [1, 7], early warning of relapse [1–3, 6] and detection of blastic transformation in chronic myelocytic leukemia [1,4].

References


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