who had high platelet counts and subsequently developed acute leukemia. However, the data continue to suggest that leukemia will develop in a greater percentage of patients with low platelet counts than in those with normal or elevated platelet counts. We emphasize that patients with low platelet counts do not always develop leukemia, and as the letter of Lewy and Kansu indicates patients with high platelet counts are not guaranteed free from the risk of developing leukemia.

It would have been useful if Lewy and Kansu had included the median follow-up time in their patients, since only 20 of 37 patients had "prolonged" follow-up. It would also have been useful to document the proportion of patients with "prolonged" follow-up who had low, normal, or high platelet counts. In this manner, the relative risk of developing leukemia within each type of patient could have been determined. It should be noted that in our small series, the median duration of follow-up was 5 yr. with a minimum duration of follow-up of 1 yr.

It is difficult to make definite conclusions from a review of cases reported in the literature. In addition to the problem of patient information frequently being incomplete, there is a natural selection into the literature of patients with adverse outcomes (for example, normoblastic anemia with sideroblasts in the bone marrow. Am J Clin Pathol 35:338, 1961

**Autologous Antibodies to AHF and Phenytoin**

*To the Editor:*

Several months ago we described in your pages two patients in whom long-term administration of phenytoin preceded the appearance of circulating anticoagulants against antithromphilic factor (AHF; factor VIII). [Poon MC, Saito H, Ratnoff OD, Forman WB, Winsneski J: Techniques for demonstration of the specificity of circulating anticoagulants against antithromphilic factor (factor VIII), with studies of two cases possibly related to diphenylhydantoin therapy. Blood 49:477, 1977]. One of these patients had also been treated with penicillin, making interpretation of the role of phenytoin difficult.

We now wish to make note of a 50-yr-old female with a history of epilepsy who had been treated with phenytoin for 35 yr. About 1 mo after two orthopedic procedures she became aware of the presence of bruises, and 3 mo thereafter she was admitted to the hospital for study. In brief, the patient's plasma contained a circulating anticoagulant against AHF at a titer of 64 U/ml. (One unit of inhibitor was defined as twice the concentration of the inhibitor preparation that inactivated 50%, of the...
Use of Baker’s Yeast to Detect Complement Receptor

To the Editor:

We recently reported the presence of lymphocytes with both T cell markers and complement receptors in acute lymphoblastic leukemia. The detection of these doubly labeled cells was made possible by using an adaptation of the method of Mendes et al., in which neuraminidase-treated sheep red cells and complement-coated zymosan particles were used. After our initial studies we had difficulties in obtaining zymosan particles that were easily usable in this assay system.

In looking for a solution to this technical problem we found that baker’s yeast, from which zymosan is prepared, could be readily substituted for the zymosan particles and was a reliable indicator for complement receptor detection. The yeast particles are prepared as follows: 250 mg Fleischmann’s active dry yeast is boiled in 60 ml 0.85% saline solution for 1 hr. All yeast particles should then stain with trypan blue. The boiled particles are centrifuged at 400 g for 5 min, washed once in gelatin-veronal buffer (GVB), and resuspended in cold GVB at 5 x 10^8 particles/ml. These can be stored at –20°C in 5-ml fractions or coated with complement and then stored at –70°C. Prior to complement coating, the yeast particles are washed three times, aspirated through a 25-gauge needle twice, and resuspended to 5 x 10^8 yeast particles/ml in GVB. Equal volumes of whole mouse serum and yeast are rotated at 37°C for 15 min. The yeast complement particles (YC) are washed three times in cold GVB, resuspended to 10^9 YC/ml, and passed through a 25-gauge needle. Then 0.2-ml aliquots are stored at –70°C and thawed immediately prior to use. The YC can now be substituted for the zymosan complement particles in the double-rosette method previously reported, and the same number of complement receptor positive cells can be detected with either reagent.

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REFERENCES


Oxidative Hemolysis and Erythrocyte Phospholipids

To the Editor:

In recent years many investigators have studied alterations in red cell phospholipid composition and metabolism with a view toward correlating such alterations with the hemolytic process. In a number of studies a decrease in the phosphatidyl ethanolamine (PE) fraction of membrane lipids has been described. PE constitutes about 30% of the total of human red cell phospholipid. In view of the current interest in oxidative changes associated with membrane alterations in anemia, it would seem desirable to remember that on the basis of current understanding a decrease in PE might be a consequence of one or more
Autologous antibodies to AHF and phenytoin [letter]

OD Ratnoff and MS Rabaa