PRELIMINARY AND SHORT REPORTS

DEMONSTRATION OF THE L. E. CELL IN THE ABSENCE OF ANTICOAGULANT*

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Since Hargraves first published his report on the "L. E. Cell" in Jan., 1948 (1), interest in this phenomenon has been mounting steadily. In his earlier work, Hargraves used heparin as an anticoagulant in preparing his material, but later (2) demonstrated that the phenomenon occurred with the use of any of the common anticoagulants. Sundberg and Lick (3) first reported finding the L. E. cell in the peripheral blood. Haserick and Bortz (4) have more recently induced the cell by mixing plasma from the peripheral blood of a patient with acute disseminated lupus erythematosus with normal marrow cells. We (5) have recently shown that the L. E. cell can be induced by mixing the peripheral blood from cases of acute disseminated lupus erythematosus with the peripheral blood from normal individuals.

In all of this work anticoagulants of various kinds were used. The phenomenon has not previously been demonstrated in direct smears of blood or bone marrow. Hargraves (2) has pointed out that, even on touch preparations made by directly smearing out the particles of bone marrow material, L. E. cells were observed only in the material outside of the touch preparation, where it had come in contact with the heparinized plasma. The constant presence of anticoagulants in preparations in which L. E. cells were demonstrated led to the query, can the L. E. cell be demonstrated in the absence of an anticoagulant?

An answer to this question might be obtained by mixing the serum from a patient with acute disseminated lupus erythematosus with defibrinated blood buffy layer from normal healthy people. Fortunately, we had at hand two cases of acute disseminated lupus erythematosus whose sternal marrow had previously been shown to contain numerous L. E. cells and whose heparinized peripheral blood plasma had been used to induce L. E. cell formation in normal sternal marrow and peripheral blood (5).

TECHNIC

Twenty cubic centimeters of venous blood was withdrawn from the patient with acute disseminated lupus erythematosus and allowed to stand until a retractile clot had formed. The same quantity of venous blood was taken from each of twelve healthy people. The blood specimens from normals were defibrinated by stirring each in a test tube with a glass stirring rod, and the clots discarded. The defibrinated blood was centrifuged for five minutes at 2000 RPM. One half cubic centimeter of the buffy layer formed in each tube was mixed with one half cubic centimeter of acute disseminated lupus erythematosus serum and permitted to stand for twenty minutes at room temperature. After standing, the mixture was placed in a Wintrobe hematocrit tube and centrifuged for five minutes at 2000 RPM. Serum layers in each tube were discarded, smears of the buffy layers were made, and stained with Wright and Giemsa stains.

To control the above procedure, blood serum from a healthy person was mixed with the buffy layers of centrifuged specimens of each of the defibrinated blood specimens used. The buffy layer from each of the specimens from the normal people was examined before mixing with acute disseminated lupus erythematosus serum for presence of L. E. cells.

RESULTS

L. E. cells were present in all smears of buffy layers to which serum from either of the patients with acute disseminated lupus erythematosus had been added. (Figs. 1, 1a, and 4).

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In addition, the nucleolysis and agglutination usually noted in smears containing L. E. cells were present. (Figs. 2 and 3).

No evidence of L. E. cell formation was found in the control smears.

**FIG. 1.** Two L. E. Cells with adjacent “clumping” produced by mixing serum from the clotted peripheral blood of a patient with acute disseminated lupus erythematosus and defibrinated normal peripheral blood. The Buffy Layer obtained from (a) 440X, (b) 1620X.

**COMMENT**

This demonstration apparently rules out anticoagulants as a causative factor in the L. E. cell phenomenon.

Removal of elements contained in the clot narrows the search for causative factors.

**SUMMARY**

A technic for demonstrating L. E. cells without the use of anticoagulants is outlined and results described.
FIG. 2. Characteristic "clumping" resulting from agglutination and nucleolysis.

FIG. 3. High power magnification of agglutination and nucleolysis.

FIG. 4. Characteristic L. E. cell induced in the buffy layer obtained from normal defibrinated peripheral blood by serum from clotted peripheral blood from a case of acute disseminated lupus erythematosus.
Note: Since this article was written Dr. Harry Agress, Instructor in Clinical Medicine, School of Medicine Washington University, has made an important contribution, as yet unpublished, to L. E. Phenomenon research. He has granted us permission to announce his results in advance of his own publication as they are further proof that L. E. cells are produced in the absence of anti-coagulants. He has devised a special technic and has clearly and convincingly demonstrated L. E. cells and rosettes in abundance in fixed, stained sections consisting of clotted sternal marrow elements untouched by anti coagulants and obtained from a patient in a moribund condition with acute disseminated L. E. In addition Dr. Agress has demonstrated what are thought to be extracellular L. E. bodies in the same preparations. These bodies are reported to be identical morphologically and to date have shown the same staining properties as those bodies found lying in L. E. cells and the rosettes.

REFERENCES


