The Finding of the L. E. (Lupus Erythematous) Cells in Smears of Untreated, Freshly Drawn Peripheral Blood

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The L.E. CELL, found so consistently in disseminated lupus erythematous, is regarded as an in vitro phenomenon. Hargraves, who first found the L.E. cells in bone marrow preparations, soon realized that these findings were conditioned by the heparinization of the marrow material. He was unable to find L.E. cells in the direct marrow or peripheral blood smears.

Lee, Michael, and Vural emphasized the fact that they had never observed L.E. cells in smears from freshly drawn blood or marrow. They also quoted previous authors as mentioning their failure to find L.E. cells in marrow or peripheral blood smears. In a personal communication, Hargraves mentioned numerous workers who examined "hundreds of thousands of cells" in smears and were not able to find L.E. cells in the direct smears from untreated peripheral blood.

The case presented herewith is remarkable because of the identification of L.E. cells in the smears from freshly drawn peripheral blood smears. These cells were found at a time when the patient was in the terminal stage of the disease a few days before death. Only a small number of definite L.E. cells were found (see figs. 1 and 2). There was also a number of additional cells of phagocytic character with inclusions of nuclear material. The nuclear material did not show the characteristic nucleolysis and, therefore, these cells could not truly be called L.E. cells. According to Hargraves, lysis of the nuclear structure is characteristic of systemic lupus erythematosis.

Case Report

A 29 year old white woman entered Mount Sinai Hospital on May 23, 1952, in the fifth month of pregnancy, for evaluation of disturbed renal function, which was considered to be of pre-eclamptic nature. She had persistent albuminuria of one month's duration and anemia. There were no other symptoms. Physical examination was completely normal but for bilateral edema of the ankles. The blood pressure was 120/80, the pulse 84, and regular. Except for a red blood cell count of 2,160,000 per cu.mm. and a sedimentation rate of 26 mm. per hour, the laboratory workup was within normal limits. The patient was given three blood transfusions and had a slight febrile reaction to the first. She was discharged on May 29, 1952, without a definite diagnosis as to the cause of the albuminuria and anemia.

She was readmitted to the hospital on June 17, 1952, with complaints of dyspnea, orthopnea and edema of the ankles. Physical examination was essentially normal except for a systolic murmur at the apex which had not been heard on the previous admission. The uterus was enlarged with the fundus elevated about 22 cm. The blood pressure on this admission was 156/80, pulse 140, and respirations 36. The patient gave birth to a stillborn boy on June 23. Postmortem of the stillbirth showed brain hemorrhage. No evidence of lupus erythematous was found. The patient again had febrile reactions to blood transfusions. The patient

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ran a continuously febrile course, and on July 6, 1952, pleuropericarditis was diagnosed. With thoracentesis and antibiotic therapy, she became afebrile with disappearance of the chest findings and was discharged on July 19, 1952. During this hospital admission she had a normochromic anemia, leukocytosis, blood urea nitrogen of about 35 mg. per cent, hypoproteinemia with normal A/G ratio, a persistently elevated sedimentation rate, and urea clearance increasing from 29 per cent on admission to 181 per cent on discharge.

The patient was again admitted to the hospital on July 25, 1952 with a history of vomiting, weakness, and fever. Physical examination revealed her blood pressure to be 114/80 and pulse 116. A pleuropericardial effusion was again present. Disseminated lupus erythematosus was now considered and the L.E. test, using heparinized venous blood according to the technic of Weisberger, Meacham, and Heinle,⁸ was positive. ACTH was started on August 5, 1952, followed by oral cortisone begun on August 27. The patient now developed arthralgias and swelling of the fingers, and further questioning revealed the presence of similar symptoms two years prior to the present illness. She gradually improved and was discharged on 37.5 mg. of cortisone per day on September 27. Leukopenia became evident twenty-five days after the start of ACTH. Although the patient showed a persistent hypo-

![Fig. 1.—X 1400.](image1)

![Fig. 2.—X 1400.](image2)

proteinemia, reversal of the A/G ratio was not found until ten days after the start of the ACTH therapy.

The patient was readmitted to the hospital on December 8, 1952 with a blood pressure of 190/120, a temperature of 100, arthralgias of the fingers, large matted axillary nodes, and flatness in both lung bases. She had a slow downhill course with the pulse rate as high as 170 and rarely going below 100. Despite therapy with ACTH, antibiotics, blood, and parenteral fluids, the patient expired at 10:35 p.m. on December 26, 1952. It was during this admission, three days prior to death, that L.E. cells were found for the first time in the peripheral blood smear. Postmortem examination confirmed the clinical diagnosis of generalized lupus erythematosus.

**Comments**

A possible explanation for our finding of L.E. cells in the peripheral smear is that these cells were searched for in the terminal stage of the disease, when one would expect the L.E. factor to be at its peak concentration. It is likely, therefore, that the time factor may be of prime importance in the finding of these
cells. Our findings do not yet completely prove that the L.E. cell is an in vivo phenomenon, since the peripheral blood smear can be considered as a post vital artefact. Furthermore, it is possible that even the slight trauma used in the preparation of a blood smear might be sufficient to produce L.E. cells under conditions in which an increased concentration of the L.E. factor is present.

SUMMARY

A case of lupus erythematosus is reported in which L.E. cells were found in the smears of freshly drawn peripheral blood.

REFERENCES

4 HARGRAVES, M. M.: Personal communication, 1953.
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