Systemic Lupus Erythematosus
Report of a Case with Acute Myocardial Infarction as the Presenting Syndrome*

JAMES R. WEBSTER, JR., CAPTAIN, MC, AUS**
AND HENRY F. FANCY, LT. COLONEL, MC, USA
Fort Leavenworth, Kansas

Systemic lupus erythematosus usually begins in an insidious manner; however, the initial symptoms may be violent and sudden. These more dramatic modes of onset include: abdominal crises, gastrointestinal hemorrhage, fulminating pneumonia, acute encephalopathy, renal failure, and severe thrombocytopenia or hemolytic anemia. We have had the opportunity to observe a case of systemic lupus erythematosus which presented as an acute myocardial infarction and it is herein reported.

Case Report
A 33-year-old white man was admitted to Munson Army Hospital with the chief complaint of crushing substernal pain which had been intermittently present for the preceding 24 hours. The pain radiated to both shoulders and there was some associated dyspnea and diaphoresis. There had been no cough or hemoptysis, and he had noted no gastrointestinal symptom.

Past history revealed that he had been hospitalized the preceding month because of a deep vein thrombophlebitis of the right leg. At this time, the hematocrit was 35 per cent and the urinalysis was completely normal. He was treated with the usual measures including anticoagulants and the process subsided rapidly. His only complication had been a transient penicillin reaction, manifested by a maculopapular skin rash. This cleared spontaneously when the drug was discontinued. He had previously been troubled by multiple contact allergies and eczematoid eruptions, as well as seasonal hay fever symptoms.

Physical examination at the time of admission revealed an acutely ill, sweating, white man who was slightly cyanotic. The blood pressure was 140/80 mm. Hg, the pulse was 105 per minute and regular, and the temperature was 98.2°F. The heart sounds were diminished.

The remainder of the findings were within normal limits. There was no residual of the previous thrombophlebitis.

Laboratory findings at the time of admission: the hematocrit was 33 per cent with a white blood count of 8,800 cells per mm.³ and a normal differential count. The serum transaminase was 56 sigma Frankel units, the blood urea nitrogen was 22 mg. per 100 ml. and the total protein was 7.25 gm. per 100 ml. with globulin of 3.7 gm. per 100 ml. The electrocardiogram (Fig. 1) revealed a pattern of anterior myocardial damage. The urine contained 2 gm. of protein per 100 ml., as well as red blood cells, white blood cells, and coarsely granular casts. The chest x-ray film was unremarkable.

Hospital Course: on the second hospital day his condition rapidly worsened. His pain had been controlled by opiates, but he became febrile, hypotensive, and developed to-and-fro pericardial friction. There was also an acute onset of congestive heart failure with cardiac enlargement, tachycardia, bilateral rales, hepatomegaly, and peripheral edema. The circulation time was prolonged, the venous pressure was elevated, and the serum transaminase was 205 sigma Frankel units, with a white blood count of 17,300 cells per mm.³ He was treated with oxygen, vasopressors, and measures to control the cardiac decompression, but showed only slight improvement.

On his third hospital day, he was clinically jaundiced and laboratory studies indicated an acute hemolytic process with a hematocrit of 14 per cent and a total bilirubin of 8.4 mg. per 100 ml. He was given transfusions of packed red blood cells in spite of a positive Coombs test. He developed an atrial tachycardia with A-V block, alternating with runs of auricular fibrillation, and a basal diastolic heart murmur was detected. The electrocardiographic changes (Fig. 1B) were compatible with evolution of an anterior myocardial infarct. On this day, a lupus erythematosus preparation showed typical L. E. cells, as well as rosettes and tart cells. Massive doses of steroids and chloroquine were added to his therapeutic regimen.

For the next several days, he appeared preterminal with toxic delirium, moderate conges-
tive heart failure, evidence of some continuing hemolysis, etc. During this time, the serum transaminase gradually returned to normal levels. On his eighth hospital day, he showed dramatic improvement and this was followed by gradual recovery from the acute stages of his illness, both clinically and from a laboratory standpoint.

By his fourth hospital week, he was semi-ambulatory, the hematocrit was 43 per cent, the lupus erythematosus preparation was consistently negative, and his electrocardiogram was compatible with evolution and healing of an acute anterior myocardial infarct (Figs. 1C and 1D).

The urine showed occasional protein and persistent casts.

His subsequent course has been complicated by: a Cushingoid state, an intermittent low sodium syndrome, a pneumococcal pneumonia, fluid retention, myasthenia with a positive edrophonium test, but a disappointing response to cholinergic drugs, and a severe depressive reaction. The electrocardiograms have shown gradual improvement and recent tracings have revealed a stable pattern (Fig. 1E). At present, he is being treated with prednisone, digitalis, chlorothiazide, chloroquine, and spirinolactone. His
condition appears relatively stable with the proteinuria as the chief laboratory manifestation of his disease.

**DISCUSSION**

Originally, the heart was thought to be infrequently damaged by systemic lupus erythematosus. More recently, it has been found to be involved in a majority of cases, either clinically or at postmortem examination. The most common forms of lupus erythematosus heart disease are: pericarditis (30 to 80 per cent), verrucous endocarditis (20 to 50 per cent), or myocarditis (10 to 50 per cent).1,2 Congestive heart failure occurs in 10 to 25 per cent of cases,3 although extracardiac factors such as systemic hypertension, anemia, and fluid retention often contribute to the development of the cardiac decompensation. As might be expected, most cases show at least transient electrocardiographic abnormalities at some stage of the disease.4

Frank myocardial infarction is unusual in systemic lupus erythematosus, and the infarct is often attributed to coexisting coronary arteriosclerosis.5 However, in several necropsied cases, localized myocardial infarction has been ascribed purely to an inflammatory vasculitis with extensive fibro-nid necrosis and vascular occlusion.1

The rapid rise and fall of the transaminase and the serial electrocardiographic changes in this case are compatible with an acute myocardial infarct. Some features do suggest pericarditis with coexistent myocardial damage. Many of the clinical findings (i.e., the friction sound, congestive heart failure, arrhythmias, etc.) might be present in either instance. While it cannot at this time be categorically stated that this patient sustained an infarction, it is our opinion that he did. We believe that it was on the basis of a coronary artery vasculitis due to the lupus erythematosus. He certainly had no evidence of extracardiac arteriosclerosis, and was being maintained on anticoagulants at the time of admission. In any case, given the initial clinical, laboratory, and electrocardiographic findings which this patient manifested, the presumptive diagnosis had to be acute myocardial infarction. Long term follow-up may answer some of the questions which still remain.

This diagnosis should be considered in the differential diagnosis of the uncommon causes of acute cardiovascular catastrophies, since with this entity, recognition and appropriate medical therapy can produce the dramatic results which were observed in this most interesting case of lupus erythematosus heart disease.

**REFERENCES**


For reprints, please write Dr. Webster at 2530 Kenilworth Avenue, Wilmette, Illinois.

---

**DIRECT VISION INTRACARDIAC SURGERY AND INEXPENSIVE THERMOREGULATING HEART-LUNG MACHINE**

The heart-lung machine as one of the most essential tools of modern surgery was discussed by Drs. Juro Wada, Toyokazu Tushitor and Yoshio Suda, Sapporo City, Japan. Its cost, however, has been an obstacle in its worldwide popularity. Majority of artificial lungs are made of plastic material or stainless steel. The former is light, transparent, but has problems in its sterilization. The latter is easy to sterilize, but difficult to make or model. To solve the problems, the authors used copper for constructing the oxygenator, which is easy to model, and used it with nickel plating. In addition, to minimize the amount of and mechanical trauma to the blood in conventional Brown-type heat exchanger, a nickel plated copper coil was placed in the oxygenated blood reservoir of the oxygenator, through which temperature regulating water circulates. With this idea, they made a bubble oxygenator called "thermo-helix-oxygenator" and a rotating disc oxygenator called the "thermo-disc-oxygenator" they have been using clinically since 1960.

Presented at the 7th International Congress on Diseases of the Chest, New Delhi, India, February 20-24, 1963.