ACUTE MYOCARDIAL INFARCTION AND PULMONARY EMBOLISM CONCOMITANTLY ASSOCIATED WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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Abstract

A 20-year-old woman with systemic lupus erythematosus suffered from acute myocardial infarction and pulmonary embolism. The patient was medically treated with good results. Systemic lupus erythematosus concomitantly associated with acute myocardial infarction and pulmonary embolism has never been reported.

Key words: Systemic lupus erythematosus, Myocardial infarction, Pulmonary embolism

Acute myocardial infarction (AMI) in the course of systemic lupus erythematosus (SLE) is rare.[1,2] The most likely cause of this event is an atherosclerotic process, accelerated by steroid administration.[3] An alternative cause is coronary arteritis,[4] as supported by the favorable response to therapies which include high doses of corticosteroid.

Renal involvement is a serious feature of SLE. The most common pathologic lesion is immune complex-mediated glomerulonephritis.[5] Endothelial cell damage produced by immune complex or other mediators of inflammation may be responsible for the activation of the coagulation system in SLE patients, which may increase the risk of cardiovascular disease.[5]

Pulmonary abnormalities are also common in SLE. These include lupus pneumonitis, interstitial fibrosis, chronic pleuritis, alveolar hyaline membrane, capillary and arteriolar thrombus.[6-7] However, pulmonary embolism and hemorrhage as complications of SLE are rare.[8]

The following case illustrates the concomitant occurrence of AMI and pulmonary embolism in a young woman with active SLE and lupus nephritis who was successfully treated by anticoagulant and pulse steroid therapy.

CASE REPORT

A 20-year-old woman was admitted because of lower abdominal pain for one week and fever of one day's duration.

A diagnosis of SLE had been made seven years ago when she had malar eurption and arthritis. Two years later, she had nephrotic syndrome and lupus nephritis was found by renal biopsy. Steroid treatment (prednisolone 20mg bid) was started, and additional immunosuppression with imuran was used half year ago. She was a nonsmoker, menstruated normally and had never received contraceptives orally.

Physical examination on admission revealed a blood pressure of 120/70 mmHg, pulse 119

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per minute, respiration 18 per minute, and temperature of 37.9°C. A chronic lupus skin lesion over the malar areas was present. The lungs were clear. The apex of the heart was in the fifth intercostal space in the midclavicular line. The rhythm was regular. The first and second heart sounds were normal. There were no heave, thrill, or murmurs. The liver and spleen were not felt. The lower abdomen was tender, with no knocking flank pain. The remainder of the physical examination was normal.

Laboratory studies disclosed the following values: hemoglobin, 7.9 g/dl; hematocrit, 23.8 percent; white blood cell count, 11500/cu mm; platelet count, 27.2 x 104 /cu mm; erythrocyte sedimentation rate, >120 mm/hr; CRP, 1.2 mg/dl; creatine kinase, 24 IU/l; blood urea nitrogen, 12 mg/dl; serum creatinin level, 0.5 mg/dl; total cholesterol level, 282 mg/dl. The serum electrolytes, glucose, liver function tests, uric acid and protein findings were within normal limits. Antinuclear antibody was positive at a titer 1:>1280 (normal, 1:20 or less); anti-ds DNA antibody, 121 IU/ml (normal, < 30 IU/ml); anti-cardiolipin, normal; serum C3, 68.3 mg/dl (normal, 90-180 mg/dl); serum C4, 10 mg/dl (normal, 10-40 mg/dl). Urinalysis showed proteinuria and leukocytosis. Urine culture grew enterococcus. Chest radiograph and electrocardiogram were normal.

The patient was thought to have a lupus flare and urinary tract infection, and was given prednisolone and antibiotics daily. Two days later, however, she suddenly developed persistent severe substernal chest pain, with radiation to the left shoulder, associated with nausea and cold sweating. The electrocardiogram (Fig. 1) revealed sinus tachycardia with signs of an evolving Q wave anterolateral myocardial infarction, as well as right axis deviation and an increase in R wave amplitude with T wave inversion in the right precordial leads suggesting acute pulmonary embolism. An echocardiography demonstrated anterolateral wall hypokinesis with normal left ventricular systolic function. Creatine kinase (CK)

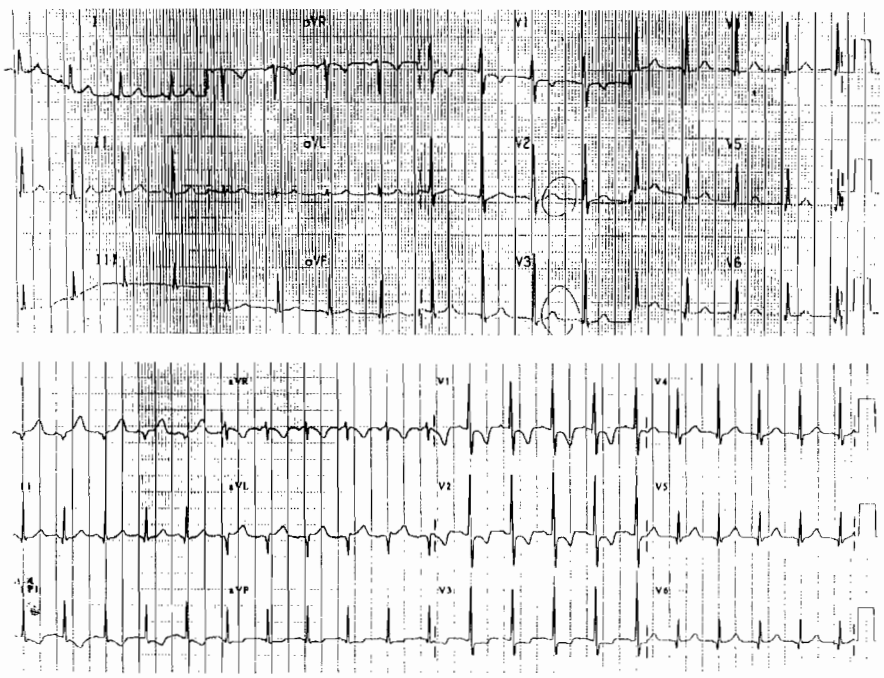

Fig. 1. Normal electrocardiogram on admission (top), and after acute myocardial infarction (bottom) showing sinus tachycardia, Q wave in I, aVL with ST elevation and reciprocal ST depression, as well as right axis deviation and increase in R wave amplitude in right precordial leads suggesting acute pulmonary embolism.

and CK MB form were, 1196 and 117 IU/liter; at 4 hours, 975 and 95 IU/liter; at 6 hours, 935 and 55 IU/liter; at 8 hours, 336 and 23 IU/liter; and at 24 hours, 185 and 25 IU/liter, respectively. Technetium 99m stannous pyrophosphate imaging was positive. AMI was confirmed. However, three hours later while awaiting cardiac catheterization, a follow-up echocardiogram showed complete resolution of the segmental wall motion abnormalities.

At cardiac catheterization, the intracardiac pressures were found to be normal. The coronary angiography showed no evidence of organic coronary artery disease. The left ventriculography showed a normal ejection fraction. The pulmonary angiogram (Fig. 2), however, demonstrated filling defects on the left and right lower pulmonary arteries.

Fig. 2. Selective balloon pulmonary angiography showing pruning of pulmonary vasculature with filling defects in left (top) and right (bottom) lower pulmonary arteries suggesting in-situ pulmonary thrombosis/embolism.

Conservative treatment was given. The patient was treated by pulse steroid therapy (methylprednisolone 1 gm intravenously qd for 3 days) for the disease flare-up. Aspirin, nitrate and heparin were instituted for management of AMI and pulmonary embolism. Clinical improvement was observed. The patient was subsequently discharged on a regimen of oral prenisolone, imuran, lopid, aspirin and coumadin daily, and still remains well.

DISCUSSION

Cardiovascular disease has been reported to be a major cause of both morbidity and premature mortality in SLE patients. The incidence ranges from 6.1 to 8.9 percent in several series,[9] consisting mainly of Libman-Sacks endocarditis, pericarditis, myocarditis, valvulitis and coronary artery disease.[10] Pathogenesis of cardiovascular disease in SLE is multifactorial, including inflammation-induced and antiphospholipid antibody-related vascular injuries, thrombosis from the underlying disease and traditional cardiovascular risk factors, corti-costerioid treatment and renal disease with resulting hypertension which accelerates athero-sclerosis.[11]

AMI in the course of SLE is very rare.[1-2] It is assumed that the infarction develops as a result of premature atherosclerosis, coronary arteritis, coronary spasm, and/or intracoronary thrombosis with angiographically normal coronaries related to hypercoagulability.[12] This case illustrates AMI associated with an angiographically normal coronary artery in a young patient with SLE, and therefore an organic coronary artery disease secondary to atherosclerosis is not likely. Though patient had no history of original chest pain before or after the infarction, the possibility of coronary spasm induced AMI cannot be completely ruled out. It is also likely that coronary arteritis and/or in situ intracoronary thrombosis may be the cause of AMI in this patient. Coronary arteritis is considered to be due to an

immunologic reaction of the coronary artery during the process of SLE. Transient occlusion may have occurred from an intense coronary inflammatory reaction with in situ thrombus formation not detected angiographically resulting in necrosis and AMI, which subsequently resolved.[13] This mechanism is suggested by the results of serologic studies (increased antinuclear antibodies, increased anti-DNA, decreased C3), by the immediate resolution of segmental wall motion abnormalities on echocardiography, by the normal coronary angiogram, and by the favorable response to therapy which included pulse steroid treatment. The infarction was therefore apparently caused by acute coronary obstruction from an in situ thrombus or embolus, in the absence of significant coronary disease.

AMI is an unusual complication of SLE, and the concomitant presence of pulmonary embolism has not previously been documented. The incidence of pulmonary abnormalities associated with SLE is about 48 %, comprised mainly of lupus pneumonitis, interstitial fibrosis, chronic pleuritis, alveolar hyaline membrane, intersitial edema, capillary and arteriolar thrombi.[14] Pulmonary embolism associated with SLE has been very rarely reported.[8] In this patient, the electrocardiogram suggested acute pulmonary embolism, and the subsequent pulmonary angiography demonstrated filling defects over left and right lower pulmonary arteries also suggesting arteriolar formation of an in situ thrombus or embolus. Moreover, her chest distress and dyspnea were relieved after one week's treatment with heparin in spite of an angiographically normal coronary artery. The pathogenic mechanisms of pulmonary embolism in SLE can be multifactorial, including the deposition of immune complexes, microangiitis, coagulopathy and nephrotic syndrome.[8] The thrombotic tendency associated with SLE may be responsible for the patient's pulmonary embolism. The hyperlipidemia and activation of the coagulation system that accompanied her nephrotic syndrome, anemia, urinary tract infection, prolonged use of corticosteroid, and increased thrombosis in the setting of antiphospholipid antibodies must have been major additional factors in the development of pulmonary embolism as well as the concomitant AMI in this patient.

The present case demonstrates an unusual development of AMI and pulmonary embolism concomitantly associated with SLE. It also suggests the effectiveness of anticoagulant and pulse steroid therapy to prevent the recurrence of thrombosis and SLE flare, respectively, in such a patient. SLE is a multisystem disease. The cause of death in SLE is usually related to renal insufficiency or infection, and AMI and pulmonary embolism generally have not been considered to be among its manifestations. This case emphasizes that they may indeed be serious and sometimes fatal consequences of SLE. Indeed, vascular events have emerged as the third leading cause of death in SLE[15].

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紅斑性狼瘡引發急性心肌梗塞併肺栓塞

李應紹

摘要

一廿二歲紅斑性狼瘡女性病人突發急性心肌梗塞及肺栓塞，經藥物治療後痊癒。文獻上尚未有報導紅斑性狼瘡同時引發急性心肌梗塞及肺栓塞的案例。

關鍵詞：紅斑性狼瘡，心肌梗塞，肺栓塞

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