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Case Report

A rare case of acute myocardial infarction with a non-specific symptom in a young female with systemic lupus erythematosus

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ABSTRACT

A 31-year-old female with an 18-year history of systemic lupus erythematosus (SLE) complained of epigastralgia and consulted the emergency outpatient department at our hospital. Her physical examination revealed tenderness at the scrobiculus cordis, which was a non-specific symptom of coronary heart disease (CHD). We ultimately gave a diagnosis of acute myocardial infarction based on coronary angiography and performed percutaneous coronary intervention. Although pre-interventional intravascular ultrasound demonstrated distinct atherosclerotic lesions in the coronary arteries, there were no atherosclerotic lesions in other systemic arteries. Although CHD in young SLE patients is a significant cause of morbidity and premature death, it tends to be misdiagnosed because their symptoms may be non-specific. In addition, this case highlights the fact that even SLE patients with no systemic atherosclerosis are at risk for the development of CHD.

<**Learning objective:** Coronary heart disease (CHD) in young systemic lupus erythematosus (SLE) patients is a significant cause of morbidity and premature death, but it tends to be misdiagnosed because their symptoms may be non-specific. Moreover, SLE patients are at risk for the development of CHD.>

Introduction

Systemic lupus erythematosus (SLE) is a chronic inflammatory disease of unknown cause that can affect the skin, joints, lungs, nervous system, serous membranes, and/or other organs of the body. Cardiac involvement includes pericarditis, Libman–Sacks endocarditis, myocarditis, and, most critically, coronary heart disease (CHD). Among patients with SLE, CHD is most commonly due to atherosclerosis, since they have an increased prevalence of atherosclerosis secondary to systemic inflammation and the adverse effects of long-term glucocorticoid treatment. In autopsy studies, substantial atherosclerosis was found to be present in up to half of young patients with SLE [1,2]. The present case highlights the need to recognize that patients with SLE are at high risk for CHD, even if they are young.

Case report

A 31-year-old woman was admitted with epigastralgia. She had no smoking history and was not obese (body mass index 18.7 kg/m^2). She had symptoms of SLE at age 13 years and this was complicated with lupus nephritis within a year. She also presented with hypertension and dyslipidemia at about the same time that she developed lupus nephritis, which were treated with losartan potassium 25 mg/day and pitavastatin 2 mg/day, respectively. Although SLE was treated with 6 mg of prednisolone, 150 mg of mizoribine, and 3 mg of tacrolimus hydrate taken orally daily, she showed finger-joint swelling before one week prior to admission. Due to the exacerbation of SLE, the daily dose of prednisolone was increased from 6 mg to 15 mg, and her joint swelling improved. She also exhibited epigastralgia 4 days before admission. Her intermittent pain became continuous pain and she consulted the emergency outpatient department at our hospital. On admission, her level of consciousness was clear and her blood pressure was normal (120/80 mmHg), whereas she had sinus tachycardia (100 beats per minute) and hyperventilation (20 times/minute). A physical examination revealed tenderness at the scrobiculus

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cordis, although the results of cardiac and pulmonary auscultation were normal. Chest radiography showed a cardiothoracic ratio of 56% in the supine position, and there was no pleural effusion or pulmonary congestion. There were no abnormal findings on imaging, including abdominal radiography and computed tomography (CT). In a blood examination, anticardiolipin antibody was negative and prothrombin time and activated partial thromboplastin time were normal. Cardiac enzymes were elevated [white blood cell count (WBC) 134,00/μL; differential WBC count: neutrophil 11,400/μL, lymphocyte 1179/μL; aspartate aminotransferase 76 U/L, lactate dehydrogenase 288 U/L, creatine kinase (CK) 518 U/L, CK-myocardial band 40.5 ng/mL, troponin T positive]. Serum levels of total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, and triglyceride were 104, 35, 50 and 52 mg/dL, respectively, and casual blood glucose and hemoglobin A1c (US National Glycohemoglobin Standardization Program) were 93 mg/dL and 5.4%, respectively. Other significant laboratory findings included normal renal function with a creatinine level of 0.7 mg/dL and a normal anti-double stranded DNA antibody level of 7.7 IU/mL (normal, 0–12 IU/mL). Urine protein and occult blood reaction were both negative (spot urine protein/spot urine creatinine 0.09 g/g creatinine). Hypocomplementemia [C3 57 mg/dL (normal, 73–138 mg/dL), CH50 23 U/mL (32–58)], and mild elevation of C-reactive protein [0.4 mg/dL (0–0.3)] were present. Electrocardiography showed ST-segment elevations in V1-4 and T wave inversion in I, aVL, and V2-6 (Fig. 1), and echocardiography showed akinesia of the anteroseptal wall and the apex of the left ventricle (Fig. 1A,B); the left ventricular ejection fraction (LVEF) was determined to be 44% using Simpson's method. Based on these findings, we gave a diagnosis of acute myocardial infarction (AMI). After the oral administration of crushed aspirin 200 mg and clopidogrel sulfate 300 mg, she underwent emergent coronary angiography, which revealed severe stenosis (99%) with thrombolysis in myocardial infarction (TIMI) grade 2 at the proximal left anterior descending coronary artery (LAD) (Fig. 2C,D). The right coronary artery was intact angiographically, and the posterior descending artery was collateral to the LAD (Fig. 2E). The attenuated coronary plaque that corresponded to the above lesion was documented by intravascular ultrasound assessment (Fig. 3). We performed percutaneous coronary intervention (PCI) [stent implantation (MULTI-LINK 3.0 × 30 mm)] and the coronary flow in the LAD improved to TIMI grade 3. Although the CK levels increased to 605 U/L, heart failure did not develop. Beginning the day after PCI, aspirin and clopidogrel sulfate were decreased to 100 mg/day and 75 mg/day, respectively, and continued thereafter. Since her systolic blood pressure was low (80–90 mmHg), we discontinued the oral administration of losartan potassium. In its place, we initiated the daily oral administration of bisoprolol fumarate at 0.625 mg/day for the treatment and prophylaxis of cardiac remodeling. As for steroid therapy, the daily oral dose of prednisolone at 15 mg was continued, and rheumatoid symptoms suggesting the exacerbation

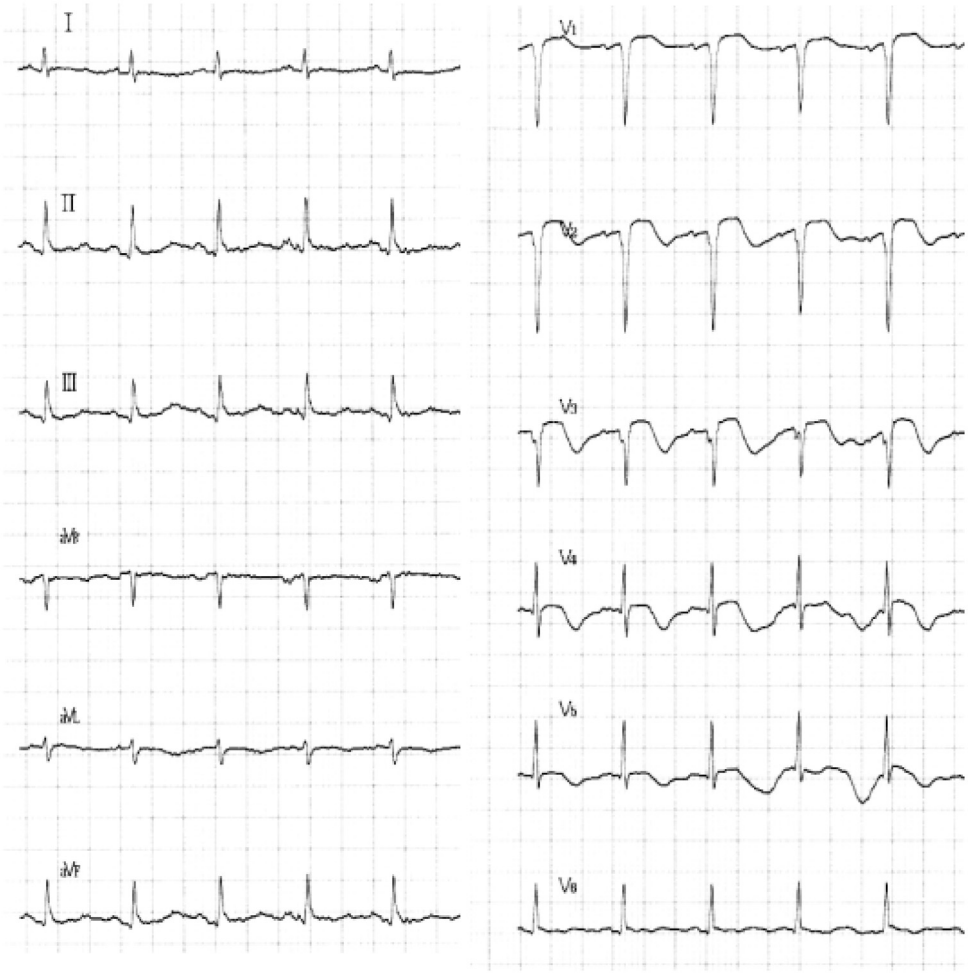

Fig. 1. Electrocardiography on admission.

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