Myocardial infarction in an 11-year-old child with systemic lupus erythematosus

To the Editor,

SLE is a chronic autoimmune disease that can affect almost every organ (1). Risk of cardiovascular diseases such as pericarditis, myocarditis, valvular heart disease, and myocardial infarction is increased in SLE, but the latter is observed rarely in childhood. An 11-year-old girl who had been followed-up at our pediatric nephrology clinic for SLE was admitted to our emergency room with chest pain followed by cardiac arrest. We detected 2–3 mm ST elevations in the DII, DIII, aVF, V5, and V6 leads of electrocardiography. Creatine kinase MB fraction (CKMB) was 7.75 ng/mL (range, 0.6–6.3) and troponin I level was 0.88 ng/mL (range, 0–0.04). Transthoracic echocardiography revealed areas of dyskinesia in the left ventricular apical region, paradoxical movement in the interventricular septum, and minimal aortic insufficiency. Coronary angiography revealed total occlusion of the

left anterior descending (LAD) and distal circumflex coronary arteries. The right coronary artery was normal. First, we applied intracoronary tirofiban HCl at a dose of 0.4 mcg/kg for bolus 5 min to the occluded lesions. Following this, we crossed the totally occluded lesion using a floppy guidewire and succeeded in restoring flow without percutaneous transluminal coronary angioplasty (PTCA). We crossed a similar totally occluded midsegment lesion in LAD using a floppy guidewire and performed PTCA using a 1.5x15 mm balloon catheter. We finished angiography after restoring distal flow. We initiated a 75 mg acetylsalicylic acid, 75 mg clopidogrel bisulfate, 0.2 mg/kg/day metoprolol, 1 mg/kg/day prednisolone, 0.1 mg/kg/day enalapril maleate, and 1 mg/kg enoxaparin sodium treatment, and warfarin sodium was added at a dose of 0.2/mg/kg to the treatment protocol a few days later. The results of further tests for thrombosis were normal. Thrombocyte function tests were normal. All cardiac enzymes returned to normal levels at the second-week follow-up. Laboratory tests ruled out antiphospholipid syndrome (APS). Anticardiolipin IgM and IgG levels were normal. At follow-up visit 1 week after discharge, the patient's physical examination and cardiac enzymes were still normal. Medications were not changed. Coronary thrombosis risk factors in SLE patients are hypercoagulability, nephrotic syndrome, APS, and anticoagulant factor deficiencies (2, 3). Hypercoagulability and collagen vascular diseases should be considered in young children with acute coronary syndrome. Coronary artery vasculitis and aneurysms are less common causes of myocardial infarction in SLE patients. Coronary arteritis observed in SLE is one of the components of systemic vasculitis (4). Current studies have shown that thrombi may recur; therefore, we recommend long-term anticoagulant treatment in APS (5). In our case, although antiphospholid antibodies were negative, we performed oral anticoagulant treatment because of the risk of recurrent thrombosis. When acute MI is suspected in children with SLE, prompt diagnosis with supportive laboratory findings is crucial. If required, coronary angiography and PTCA should be performed and long-term medications should be planned. Further studies are required to detect etiological factors and promptly initiate appropriate treatment.

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