ative atrial fibrillation, with great interest. In this study, the groups are well balanced and standardized in many aspects, but there is no data revealing the duration of the on-pump procedures, which is very important and forms a basic variable in coronary artery bypass grafting. On the other hand, one should underline the two major causes of atrial fibrillation as cardiopulmonary bypass and oxidative stress/inflammatory response triggered by cross clamping (2–4). Many cellular and non-cellular elements are activated during cardiopulmonary bypass, particularly the triggering pro-inflammatory mechanisms (5). Thus, we strongly believe that the groups in this study should have been standardized considering the cardiopulmonary bypass and cross-clamp times if the SYNTAX score is a predictor of postoperative atrial fibrillation. We would deeply appreciate if the authors share their opinion or any data related to the matter.

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Author`s Reply

To the Editor,

We are pleased with the authors' interest in our article titled "SYNTAX score predicts postoperative atrial fibrillation in patients undergoing on-pump isolated coronary artery bypass grafting surgery" that is published in Anatolian J Cardiol October 18. Epub ahead of print (1), and we would like to thank them for their contribution. As the authors have mentioned, the prolongation of ischemic time increases the risk of postoperative atrial fibrillation (PoAF). Mathew et al. (2) have reported that the pump and cross-clamp times during coronary bypass surgery predict PoAF. However, the cross-clamp and bypass times were not included in our patient data, and we believe that the patient population was too small to add these variables in the analysis; there would be too many variables for a small group and this fact could disrupt the results. With the inclusion of these data, our hypothesis can be further tested in a bigger patient population.

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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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