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Author’s Reply

To the Editor,

We are pleased by the author’s (1) interest in our case report entitled “Recurrent spontaneous dissection affecting different coronary arteries of a young female” published in the February 2016 issue (16: 137-40) of Anatol J Cardiol.

The authors proposed that medical treatment may be an option for this case because of spontaneous healing potential of the coronary artery dissection and its recurrent nature. However, it should be accepted that there is no guideline-directed treatment and diagnostic algorithm for spontaneous coronary artery dissection. In large case series, conservative treatment is the preferred strategy for stable patients without ongoing ischemia and if the involved arteries are small or medium sized. Patients with ongoing chest pain, ST elevation, or hemodynamic instability should undergo PCI, particularly when the dissection affects major arteries supplying large areas of the myocardium (2-5). An emergency coronary artery bypass grafting (CABG) should be considered if the dissection extends from the left main into the left anterior descending artery (LAD) and circumflex arteries.

In our case, as shown in the first figure, there is a TIMI 0 flow in LAD after the first septal branch. We first performed PCI to relieve the ongoing ischemia and reduce the infarct size. In the second episode, the patient suffered acute pulmonary edema treated with initial medical treatment; however, repeat angiogram showed persistent flow-limiting lesion, possibly caused by the intramural hematoma. Because of the life-threatening nature of this condition and hemodynamic instability, we were forced to consider the patient for CABG. In the third episode, the reason behind choosing PCI was the patient’s severe ischemia that was unresponsive to medical treatment and compromised hemodynamics, with TIMI I–II flow in the right coronary artery.

Moreover, we accept the role of adjunctive intracoronary imaging, such as optical coherence tomography (OCT) and intravascular ultrasound (IVUS), particularly in diagnosing SCAD subtypes, intramural hematoma, and localizing side branch/true lumen for the intervention (6). However, because of lack of IVUS or OCT facilities in our laboratory at that time, we could not use these techniques.

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Effects of cardiopulmonary bypass on new-onset atrial fibrillation

To the Editor,

We read the 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), in which the authors described the effects of SYNTAX score on postoper-
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