which was suggested by Hong et al. (1), published in *Anatolian J Cardiol* 2014; 14: 40-7. They had suggested that the presence of MB on a coronary artery was one of the clinically independent risk factors for atherosclerosis, such as age, diabetes, and dyslipidemia. Duygu et al. (2) previously stated that MB initiated and facilitated the development and progression of atherosclerosis in the *Anatolian Journal of Cardiology* in 2007 (2). Systolic compression, which is a diagnostic marker for the presence of MB on the coronary angiogram, is generally supposed to account for the hemodynamic and endothelial changes that promote the atherosclerosis. In fact, the hemodynamic abnormalities are induced by MB during the diastolic period, in which the coronary artery flow and the myocardial perfusion are at their maximum (3). Since the MB behaves as an anatomic obstacle that surrounds and limits the coronary artery from its outside, a diastolic flow gradient develops at the proximal part of the bridged arterial segment. So, the diastolic gradient exerts a "seeding effect" that urges the cellular and lipid component of blood to pass into the sub-endothelial layers of the coronary artery. Phagocytic cells, cholesterol, and lipoprotein particles are the main components of an atherosclerotic plaque and also determine the vulnerability of the plaque (4). Moreover, the diastolic gradient at the proximal segment induces an increased shear stress and endothelial dysfunction, which are represented by reduced nitric oxide synthesis, antithrombotic functions, and vasodilation. These are the initial and earliest abnormalities observed in the development of atherosclerotic plaque (AP).

In the preliminary results of our study, we observed that the serum levels of total cholesterol, LDL- and VLDL-cholesterol, and triglyceride were significantly higher in patients with MB and AP (n=7) and AP (n=9) compared to patients with only MB (n=18) (unpublished data). Patients with MB who had a normal lipid profile were free from atherosclerosis, while all patients with MB and coexisting hypercholesterolemia had atherosclerotic plaque in the proximal arterial segment of the MB. It reminded us that the ancient guilty; cholesterol; was again responsible for the atherosclerosis at the proximal coronary segment of the MB. Coexistence of MB and AP in the presence of hyperlipidemia indicates that cholesterol may be a prerequisite for the development of an AP proximal to the MB. MB was surprisingly detected with a high prevalence and was found to be highly associated with atherosclerosis and sudden cardiac deaths in young and young adult subjects (5). Nevertheless, a debate about the criteria of statin therapy, target cholesterol levels, and whether it is an equivalent of atherosclerotic coronary artery disease, as well as diabetes, will develop in the management of those patients with MB. We suggest that MSCT coronary angiography, which has a great capability in the detection of MB and AP, even at the initial stages, may guide the indication of statin therapy by documenting the presence of AP in association with MB.

Cholesterol; is accused for the atherosclerosis proximal to myocardial bridging?

To the Editor,

We excitedly read the article about the close relationship of myocardial bridging (MB) and atherosclerosis at its proximal segment,

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Author's Reply

The authors of this mentioned article did not send any reply for this Letter to the Editor, despite our insistent requests.