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Chronic Diseases and Translational Medicine 3 (2017) 260−262

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Acute myocardial infarction caused by myocardial bridging alone confirmed by using intravascular ultrasonography

Available online 3 July 2017

Keywords: Acute myocardial infarction; Myocardial bridging; Coronary angiography; Intravascular ultrasonography

Dear Editor:

Although the underlying pathogenesis of acute myocardial infarction (AMI) caused by myocardial bridging (MB) is not fully understood, high shear stress that induces endothelial dysfunction or damage that leads to thrombus formation has been mainly considered. We report a case of AMI due to MB confirmed in a series of angiographic examinations, including intravascular ultrasonography (IVUS). Our data may provide credible evidence that supports this hypothesis.

A 28-year-old man presented with recurrent chest pain for 3 days. The symptoms aggravated and persisted on June 10, 2014. Electrocardiography (ECG) revealed ST elevation over the V_1-V_4 leads. His Troponin T level was elevated to 6.8 ng/ml (normal range, <0.04 ng/ml). A diagnosis of acute anterior ST-elevated myocardial infarction was made. Urgent coronary angiography revealed a proximal total occlusion of the left anterior descending (LAD) coronary artery (Fig. 1A). Immediate reperfusion of the artery was achieved with only a guide wire passed through the lesion (Fig. 1B). Hence, he was treated with thrombus aspiration (Fig. 1C) and intravenous perfusion of a Glycoprotein (GP) IIb/IIIa inhibitor for a heavy thrombus load accompanied by thrombolysis in myocardial infarction (TIMI) grade II flow. On June 16, 2014, he underwent coronary angiography again. Data showed a lighter thrombus load than that on June 10, and blood flow in the LAD coronary artery achieved TIMI grade III. A MB in the middle of the LAD coronary artery was found along with compression to 80% stenosis during systole (Fig. 1D). He was given dual antiplatelet drugs and statin. During the follow-up period, he was asymptomatic and the ECG finding indicated an old anterior myocardial infarction (Fig. 1E). To further avoid the possibility of fixed stenosis in the occluded lesion, he was admitted and underwent coronary angiography for the third time on October 9, 2014. As expected, data indicated a MB in the middle of the LAD coronary artery with compression to 80% stenosis during systole, without any fixed atherosclerotic stenosis or thrombus (Fig. 1F and G). A significant "milking effect" was observed during systole (Fig. 1F, arrow), which was released in diastole (Fig. 1G). Furthermore, the IVUS image confirmed that his AMI was caused by the MB alone (Fig. 1H and I). IVUS imaging revealed MB during diastole (Fig. 1H) and systole (Fig. 1I). A half-moon-like, echolucent area surrounding the bridge was observed during the whole cardiac cycle.

MI is defined as an intramural segment of a coronary artery that normally courses epicardially. It is a common congenital abnormality of coronary arteries, usually thought to be a benign anatomical variant.

In our case, we performed angiographic examination three times and completed the IVUS test to confirm the imaging of the LAD lumen with MB but no significant

Peer review under responsibility of Chinese Medical Association.

Production and Hosting by Elsevier on behalf of KeAi

http://dx.doi.org/10.1016/j.cdtm.2017.05.001

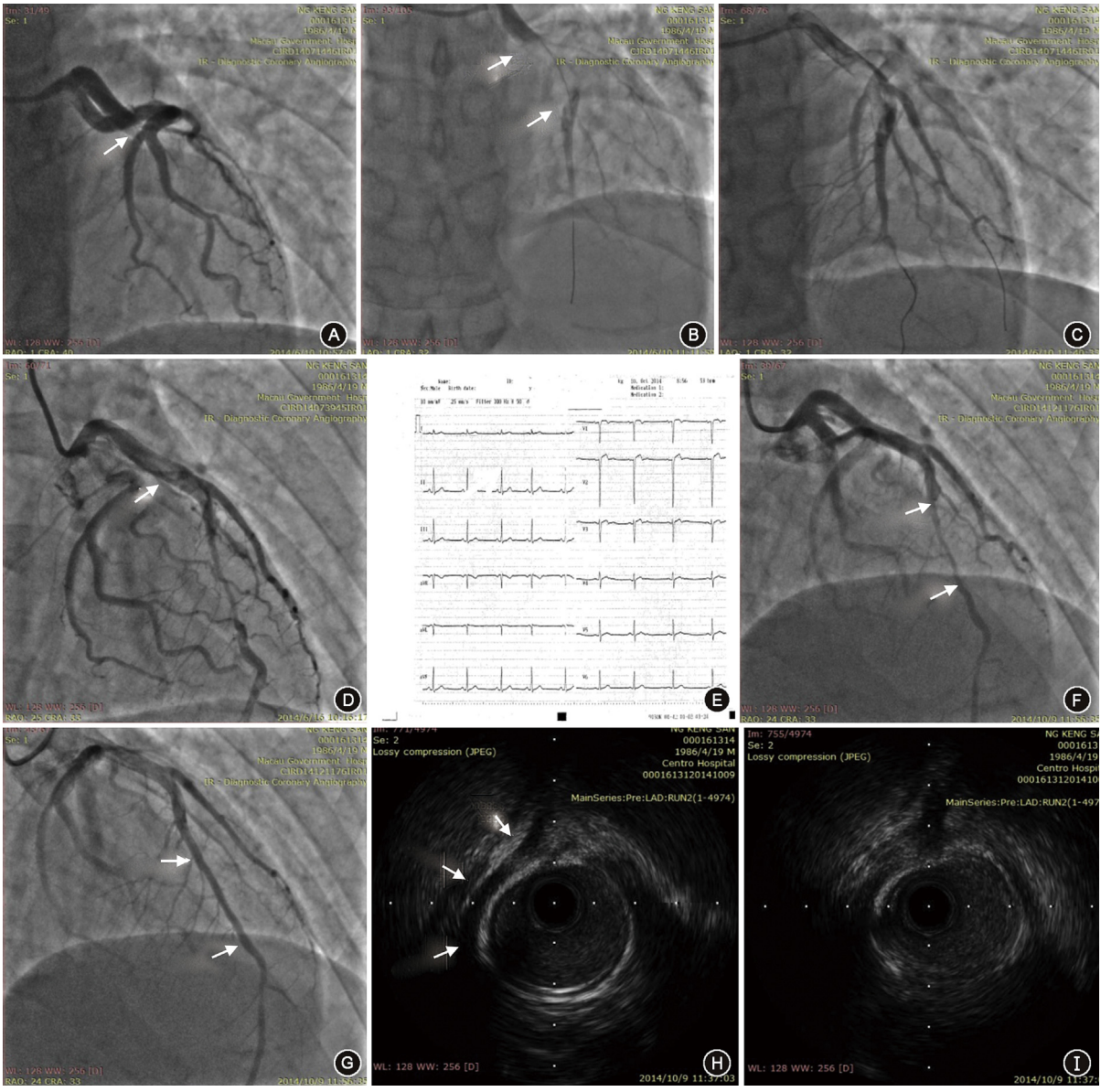

Fig. 1. A series of coronary angiography, electrocardiography (ECG), and intravascular ultrasonography examination images. **A.** Urgent coronary angiogram taken on June 10, 2014, showing the proximal total occlusion of the left anterior descending (LAD) coronary artery (arrow). **B.** Immediate reperfusion of the artery achieved with only a guide wire passed through the lesion (arrow). **C.** Coronary angiogram after treatment with thrombus aspiration. **D**. A myocardial bridge (MB) in the middle of the LAD coronary artery, with compression to 80% stenosis during systole (arrow), found on the repeated coronary angiography on June 16, 2014. **E.** ECG indicated an old anterior myocardial infarction during the follow-up period. **F and G.** On October 9, 2014, repeated coronary angiography detected MB in the middle of the LAD coronary artery, with compression to 80% stenosis during systole, without any fixed atherosclerotic stenosis or thrombus. A significant "milking effect" can be observed during systole (F, arrow), which is released in diastole (G, arrow). **H and I.** Intravascular ultrasonography (IVUS) image confirming that the AMI was caused by the MB alone. IVUS image of the MB during diastole (H, arrow) and systole (I). A half-moon-like, echolucent area surrounding the bridge can be observed during the whole cardiac cycle.

atherosclerosis. We obtained full imaging pictures of the coronary angiograms of the patient, which were seldom reported in previous cases. In our case, thrombus formation was found proximal to the MB on urgent coronary angiography, and the thrombus disappeared during the third coronary angiography after 4 months of dual antiplatelet treatment. Similarly, in 2012, our colleagues, Zhu et al., reported the case of an adolescent

athlete with MB who developed MI without any risk factors of coronary atherosclerotic heart disease.[1] We conclude that the main reason for the AMI in the present case was the thrombus formation proximal to the MB. In addition, coronary spasm in the segment, which included the MB of the LAD coronary artery, should be considered. However, further definite imaging evidence for coronary spasm is needed in this case.

Atherosclerosis evolution is significantly suppressed in the LAD coronary artery segment under the MB regardless of age, whereas the segment proximal to the bridge is vulnerable.[2–5] These effects, which are due to the existence of the MB, result from the altered hemodynamic force in the segment under the MB. Scanning electron microscopy revealed that the endothelial cells proximal to the MB were polygonal and flat in shape, but those under the MB became spindle shaped, engorged, and aligned in the direction of the blood flow.[5] These changes of the endothelial cell shape and alignment between the two segments indicate that under MB, the intimal surface of the LAD coronary artery is subjected to high shear stress, resulting in a reduced susceptibility to atherosclerotic change.[6]

Such hemodynamic change in the human coronary segments under the MB also decreases the expression levels of vasoactive proteins such as endothelial nitric oxide synthase, endothelin 1, and angiotensin-converting enzyme in the endothelial cells of the arterial intima relative to the segments proximal to the bridge.[7] High shear stress also reduces the ability of intimal smooth muscle cells to produce collagen fibers under the MB.[8,9] The alteration of the hemodynamic force in the coronary artery is caused by MB compression itself during systole. It is rephrased that such force is practically subjected to the MB thickness and length for regulation of the atherosclerosis suppression in the segment under the MB.[4]

Although the underlying pathogenesis of AMI caused by MB is not fully understood, high shear stress that induces endothelial dysfunction or damage that leads to thrombus formation has been mainly considered. Our present case with a series of angiographic examinations, including IVUS, may provide credible evidence that supports this hypothesis.

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23 November 2016

Edited by Jing-ling Bao