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Editorial

Editorial: Anomalous aortic origin of coronary arteries: What we know, what we need to know

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Primary percutaneous coronary intervention (pPCI) with stent implantation is now an established reperfusion strategy for patients with ST-segment elevation myocardial infarction (STEMI) [1]. Recent guidelines for management of patients with STEMI strongly recommend to perform pPCI among STEMI patients with symptom duration <12 h [2]. It is also recommended that first medical contact-to-balloon time should be less than 90 min for pPCI [2]. In Japan, a recent prospective observational study with 2030 consecutive STEMI patients reported that pPCI was conducted in 97.2% of the patients, median door-to-balloon time was 42 min, and pPCI was performed within 90 min after the first medical contact in all cases [3]. In the clinical catheterization laboratory, diagnostic coronary angiogram is first performed to identify the occluded vessel segment. The prolonged diagnostic catheterization procedure could delay the reperfusion therapy, which may lead to worse clinical outcome for STEMI patients. Because the anomalous origin of coronary arteries could be one of the causes of prolonged diagnostic catheterization, the cardiologist performing coronary angiography should know the patterns of coronary anomalies.

In this issue of the Journal, Yamamoto et al. report a case of STEMI in the territory of an anomalous left circumflex coronary artery (LCX) running through the retro-aortic region [4]. This is an important message as there is some possibility of atherosclerotic development and rupture of plaque in an anomalous LCX. Anomalous origin of the LCX from the right sinus of Valsalva was first described in 1933 and is the most common congenital coronary variant, with prevalence of coronary angiography at 0.18–0.67% [5–7]. However, it was considered as a benign anomaly because no clinical complication was reported related to this anomalous origin of the LCX [8]. Recently, it has been reported that some patients had an increase in coronary artery disease in these aberrant vessels due to the acute angulation of its origin from the aorta and its posterior retro-aortic course [9,10]. According to this study, significant obstructive coronary disease in the retro-aortic segment was found in the majority of anomalous LCX (73%); however, additional atherosclerotic change was not observed in another two coronary arteries [9]. For this reason, it was still controversial whether the mechanism of acute vessel occlusion is the continuous compression of angulated segment of the anomalous LCX due to cardiac motion or plaque rupture with subsequent thrombosis. This case report clearly demonstrated that the mechanism of vessel occlusion was the disruption of atherosclerotic plaque and subsequent formation of mural thrombus by using intravascular ultrasound. The retro-aortic posterior course could be a contributing factor in the development of atherosclerosis in the anomalous LCX because of increased sheer stress at the proximal part of this artery due to its ostial angulation. Similar to previous studies, the LCX in this case report originated from a common ostium in the right coronary sinus and took a posterior course to the great vessels before supplying the posterolateral surface of the left ventricle. In accordance with the current report, previous studies revealed that the majority of stenosis in the anomalous LCX appears to be confined to the proximal to mid body of the vessel [10].

Although coronary angiography is a routine procedure in the cardiac catheterization laboratory, it is limited by its two-dimensional imaging aspect and a low sensitivity for identifying lesions in the presence of positive remodeling and diffuse disease. Moreover, coronary atherosclerotic plaques cannot be characterized. Intravascular ultrasound (IVUS) is a catheter-based imaging modality, which provides high resolution cross-sectional images of the coronary arterial walls. Because of its unique feature, IVUS can clearly visualize ruptured plaque. The assessment of ruptured plaques gives us a lot of information regarding the progression of atherosclerotic plaque and plaque vulnerability. A previous IVUS study showed that at least 1 plaque rupture was found somewhere other than on the culprit lesion in patients with acute coronary syndrome [11]. Another 3-vessel IVUS study reported that plaque ruptures occurred mainly in proximal segments of the left anterior descending artery, the proximal and distal segments of the right coronary artery, and the entire LCX [12]. Therefore, the use of IVUS before intervention in cases of anomalous origin of the LCX may clarify the precise mechanism of plaque formation in this region.

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