High Shear Thrombosis in Vasospastic Angina

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Abstract: In patients with vasospastic angina (VSA), transient local hemodynamic changes due to vasospasm can result in endothelial damage and thrombus formation. One hundred nine spasm segments (93 patients) were compared with 55 non-spasm segments (39 patients). All target lesions were analyzed by OCT. Thrombus was seen more frequently at spasm segments compared to non-spasm segments (28.4% vs. 7.3%, p = 0.026) and the size of thrombus was larger at spasm segments compared to non-spasm segments (0.26 ± 0.50 mm^2 vs. 0.04 ± 0.01mm^2,p = 0.023). Among the spasm segments, thrombus was located most frequently at spasm sites (77.4%) followed by upstream of spasm segments (22.6%). Plaque erosion was more prevalent at spasm segments compared to non-spasm segments (25.7% vs. 5.4%, p = 0.001). Thrombus and plaque erosion were more common at spasm segments compared to non-spasm segments. These findings suggest the potential benefit and treatment role of antiplatelet therapy in VSA.

1. Introduction

Coronary artery spasm plays an important role in the pathogenesis of not only variant angina but also various types of ischemic heart disease, including acute coronary syndrome.[1] Although fibrous cap disruption complicated by thrombosis is considered to be the most important mechanism for the development of acute coronary syndrome (ACS),[2,3] spasm of the coronary artery can also compress atherosclerotic plaques, causing vascular injuries and thrombus formation.[4,5] With postmortem examination, Maseri, *et al.* revealed fresh thrombi at spasm sites and suggested that blood stagnation caused by spasm may result in thrombus deposition at the site of the damaged intima.[6] In an animal experiment using electron microscopy, partial arterial constriction (40-60% reduction in luminal diameter that is insufficient to reduce the rate of distal coronary flow) resulted in endothelial denudation, platelet deposition and microthrombi formation in areas proximal to the point of maximum constriction.[7] Therefore, focal spasm can induce local hemodynamic changes which may result in endothelial damage and thrombus formation. Recently, we reported that optical coherence tomography (OCT) - defined erosion with thrombus occurred in more than a fourth of patients with vasospastic angina.[8] However, these changes have not been studied comparing spasm and non-spasm segments. Therefore, we aimed to evaluate the in-vivo morphological characteristics and thrombus formation at coronary spasm segments compared to non-spasm segments in patients presenting with suspected vasospastic angina (VSA) using OCT.

2. Method and results

2.1. Study population

93 patients with spasm segments and 39 patients without spasm segments were included for analysis. OCT images of spasm segments and non-spasm segments were analyzed at 0.2mm intervals blinded to clinical presentations. OCT images were analyzed 5mm proximally and distally from

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the maximum spasm site in VSA and from the minimal atherosclerotic lesions in non-spasm segments respectively. This was confirmed with the corresponding coronary angiography using side branches as markers.

2.2. OCT findings

Thrombus was more frequently seen at spasm segments compared to non-spasm segments (28.4% vs. 7.3%, p = 0.026) with the majority being white thrombus for both groups. The size of thrombus was larger at spasm segments compared to non-spasm segments (0.26 ± 0.50mm^2 vs. 0.04 ± 0.01mm^2, p = 0.023). Among the spasm segments, thrombus was located most frequently at spasm sites (77.4%) followed by upstream of spasm segments (22.6%). There was no thrombus seen downstream of spasm segments. Thrombus was found at 4 non-spasm segments in non-VSA patients. Fibrous cap disruption was found in both groups, at 3 spasm segments and 1 non-spasm segment. Lumen irregularity was more common at spasm segments (76.1% vs. 30.9%, p <0.001). There were significant differences in plaque characteristics between spasm and non-spasm segments. 44.0% of the spasm segments were composed of fibrous plaque, whereas 27.3% of the non-spasm segments had calcific plaque. There was no calcified plaque seen in spasm segments. Plaque erosion, however, was more common at spasm segments compared to non-spasm segments (25.7% vs. 5.4%, p = 0.008) (Fig. 1).

3. Conclusions

Our results show that thrombus and plaque erosion are common findings at spasm segments in patients with VSA compared to non-spasm segments in non-VSA patients.

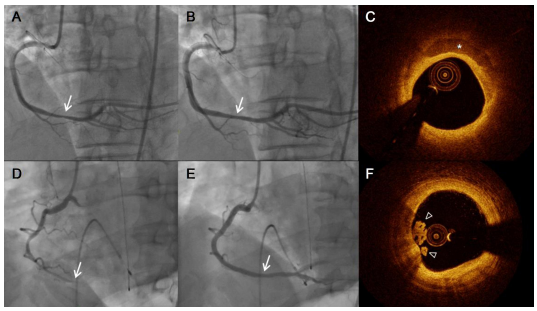

Fig. 1. Coronary angiography and OCT images of a representative case of non-vasospastic angina (A, B and C) and vasospastic angina (D, E and F)

Transient high shear stress and stenosis induced by vasospasm may play an important role in the formation of thrombus and plaque erosion in patients with VSA. These findings suggest the potential benefit and treatment role of antiplatelet therapy in VSA.

4. References

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