IMAGES IN CARDIOLOGY

Sequential Activation of Vulnerable Plaques Endorsing the Inflammatory Hypothesis of Atherosclerosis

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Abstract

A case of sequential activation of vulnerable plaques in two different coronary vessels over the course of 2 days is being presented probably related to inflammation inciting these acute coronary events. ***Rhythmos*** 2017;12(4): 69-70.

Key Words: acute coronary syndrome; atherosclerosis; vulnerable plaque; inflammation

Abbreviations: ACS = acute coronary syndrome; LAD = left anterior descending; PCI = percutaneous coronary intervention

A 66-year-old gentleman was referred for coronary angiography due to recent crescendo angina and a positive exercise tolerance test. He complained of anginal symptoms over the past one year but symptomatology worsened dramatically over the past few weeks. Two weeks earlier he underwent an exercise test which was positive for symptomatic inferolateral ischemia (Fig. 1). Coronary risk factors included smoking, diabetes mellitus managed with oral hypoglycemic agents and hypercholesterolemia treated with atorvastatin. Coronary angiography revealed a critical lesion at the proximal segment of the left circumflex (Fig. 2, panel A, arrow), disease at the proximal and mid segment of the obtuse marginal branch and borderline lesions of the left anterior descending (LAD) (Fig. 2, panels B/D, arrows) after a large ramus intermedians (RI) branch. Percutaneous coronary intervention (PCI) with direct stenting of the left circumflex was performed successfully during the same session. Result is seen in Fig. 2, panel C, arrow).

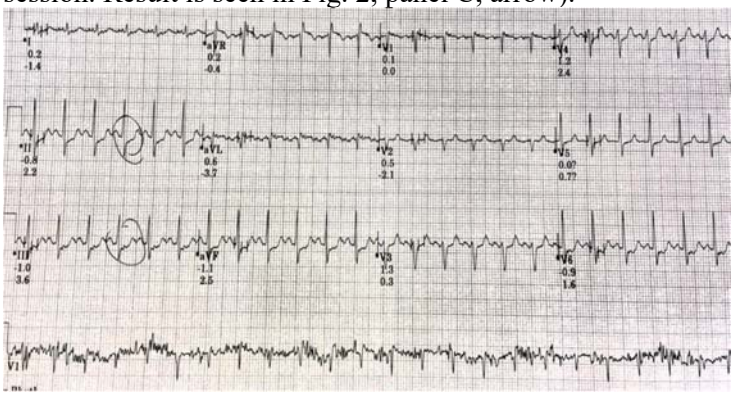

Figure 1. Depression of the ST segment was noted at peak exercise in leads II, III, aVF and V6 indicative of inferolateral wall ischemia.

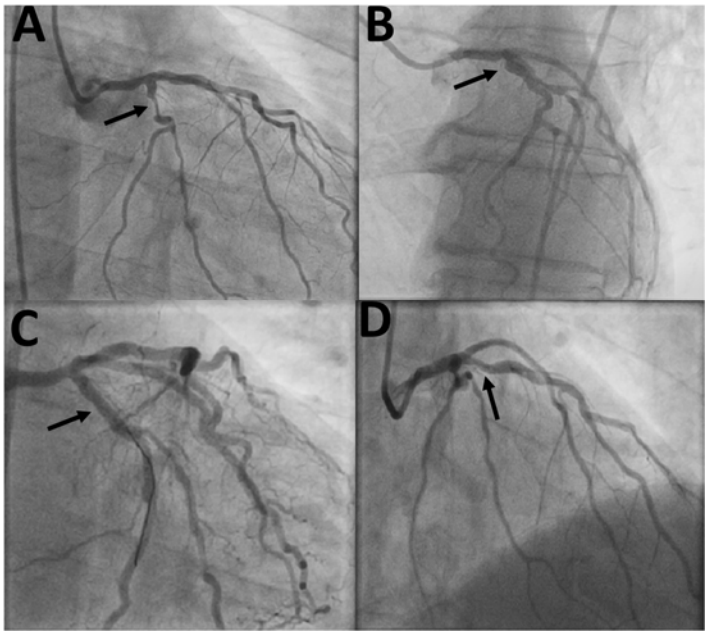

Figure 2. Right anterior oblique (RAO) views are displayed in panels A, C and D and a left anterior oblique view in panel B. See text for discussion.

One day after hospital discharge, the patient developed two episodes of rest angina, with clinical characteristics differing from the initial ones in terms of precordial chest pain location and nature. He sought medical attention and was re-admitted after the second episode. Upon admission, new ST-T changes were recorded with biphasic T waves in leads V2-4 (Fig. 3) and mild elevation of cardiac troponin T. A repeat coronary angiogram was obtained showing a patent stent of the left circumflex (Fig. 4, panel A, arrowhead) with critical stenosis of the LAD lesion (Fig. 4, panel A, arrow). After coronary injection of 300 μg of nitroglycerin, the lesion was partially improved, while a second lesion became apparent distal to the first one (Fig. 4, panels B/C/D, arrows); both lesions were distinctly hazy. Due to close vicinity of the first proximal lesion with the large RI branch and risk of possible compromise of this branch should a PCI be performed, initial dilation of the second lesion was decided. After direct stenting of the lesion and additional intracoronary injection of nitroglycerin, more angled views of the LAD were taken and analyzed, with some of them showing borderline degree of stenosis, and one (right anterior oblique with caudal angulation) indicating a significant (~80%) stenosis. Due to patient's presenting symptoms of acute coronary syndrome and ECG changes pointing to the LAD as the culprit vessel, it was finally decided to proceed with further proximal stenting and thus complete revascularization of the LAD lesions. There were thoughts of using intravascular ultrasound to guide placement of the

proximal LAD stent to avoid compromise of the RI branch. However, in order to keep it simple, direct stenting was cautiously undertaken and successfully performed without running into problems by landing the proximal end of the stent precisely in juxta-position to the RI origin. Final result is displayed in Figure 4, panels E and F (arrows).

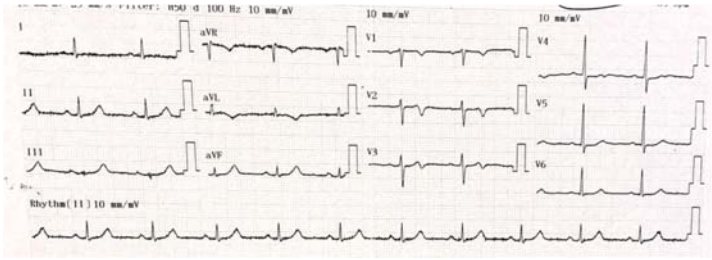

Figure 3. Biphasic T waves were noted in leads V2-4 and negative T waves in lead aVL, suggestive of ischemia at a distance from the initially stented vessel (left circumflex) going along with the new angiographic findings of critical disease in the left anterior descending (LAD) coronary artery (see Fig. 4).

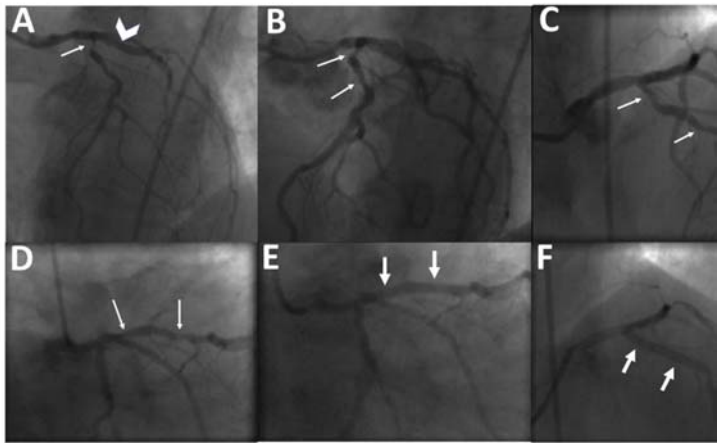

Figure 4. LAO views are displayed in panels A and B, and RAO views in panels C, D, E and F. See text for discussion.

Patient's post-procedural course was uneventful and he was discharged home in two days and has been asymptomatic for the ensuing three weeks.

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Although the exact inciting factors of the vulnerable plaque rupture are unknown, inflammation is considered to play a pivotal role in such an event. Indeed, acute coronary syndromes (ACS) have long been considered to be triggered by a generalized inflammatory process for which limited management options have been available. Concurrent activation of multiple lesions has been reported in up to one third of patients with ACS. [1-5] Although we did not use intravascular imaging techniques (ultrasound or optical coherence tomography) to identify vulnerable plaques in our patient and/or C-reactive protein levels to determine an inflammatory process activation,[6] the clinical, ECG, and angiographic features were consistent with such a scenario. Coronary vasospasm might have also played a role, however, the lesions of the LAD had worse angiographic appearance compared to their prior depiction even after reversal of the vasospastic component after the intracoronary injection of nitroglycerin.

Multiple plaque rupture has been associated with systemic inflammation, and a poor prognosis.[2] Fortunately, our patient did not sustain any life-threatening complications of recurring ischemia due to activation of coronary plaques at a second vessel and was fortunate to make it back to the hospital and undergo successful PCI. Preliminary studies have emerged that show that anti-inflammatory therapies may benefit patients with ACS. [7] However, until these data are corroborated by further studies and suggested therapies become more practical, our current anti-inflammatory approach is practically limited to use of high-intensity statin therapy to avail of the pleiotropic and plaque-stabilizing effects of such therapy,[8-10] combined with potent antiplatelet treatment and aggressive risk factor modifying strategies.[11]

REFERENCES

1. Hong MK, Mintz GS, Lee CW, et al. Comparison of coronary plaque rupture between stable angina and acute myocardial infarction: a three-vessel intravascular ultrasound study in 235 patients. *Circulation* 2004;110:928-933.
2. Tanaka A, Shimada K, Sano T, et al. Multiple plaque rupture and C-reactive protein in acute myocardial infarction. *J Am Coll Cardiol* 2005;45:1594-1599.
3. Kubo T, Imanishi T, Kashiwagi M, et al. Multiple coronary lesion instability in patients with acute myocardial infarction as determined by optical coherence tomography. *Am J Cardiol* 2010;105:318-322.
4. Dato I, Niccoli G, Cataneo L, Crea F. Multiple coronary plaque ruptures in a patient with a recent ST-elevation acute myocardial infarction causing recurrent coronary instability. *J Cardiovasc Med (Hagerstown)* 2013;14:681-682.
5. Maehara A, Mintz GS, Bui AB, et al. Morphologic and angiographic features of coronary plaque rupture detected by intravascular ultrasound. *J Am Coll Cardiol* 2002;40:904-910.
6. Tanaka A, Imanishi T, Kitabata H, et al. Distribution and frequency of thin-capped fibroatheromas and ruptured plaques in the entire culprit coronary artery in patients with acute coronary syndrome as determined by optical coherence tomography. *Am J Cardiol* 2008;102:975-979.
7. Ridker PM, Everett BM, Thuren T, et al. Antiinflammatory Therapy with Canakinumab for Atherosclerotic Disease. *N Engl J Med* 2017;377:1119-1131.
8. Schwartz GG, Olsson AG. The case for intensive statin therapy after acute coronary syndromes. *Am J Cardiol* 2005;96:45f-53f.
9. Athyros VG, Katsiki N, Karagiannis A, Mikhailidis DP. Short-, mid-, and long-term benefits of peri-procedural high-intensity statin administration in patients undergoing percutaneous coronary intervention. *Curr Med Res Opin* 2015;31:191-195.
10. Koskinas KC, Zaugg S, Yamaji K, et al. Changes of coronary plaque composition correlate with C-reactive protein levels in patients with ST-elevation myocardial infarction following high-intensity statin therapy. *Atherosclerosis* 2016;247:154-160.
11. Takata K, Imaizumi S, Zhang B, Miura S, Saku K. Stabilization of high-risk plaques. *Cardiovasc Diagn Ther* 2016;6:304-321.