Austin Journal of Cardiovascular Disease and Atherosclerosis

Open Access

Austin Publishing Group

Case Report

Malignant Vasospasm of Non Culprit Vessel Following Coronary Intervention- An Unusual Occurrence

H.S Natraj Setty*, Shivanand S Patil, T.R Raghu, Jayashree Kharge, J.R Vijaykumar, B.K Geetha, Babu Reddy, C.N Manjunath

Sri Jayadeva Institute of Cardiovascular Sciences and Research, Bangalore, Karnataka, India

***Corresponding author:** H.S Natraj Setty, Sri Jayadeva Institute of Cardiovascular Sciences and Research, Bangalore, Karnataka, India

Received: September 13, 2017; **Accepted:** September 28, 2017; **Published:** October 05, 2017

Abstract

Spontaneous multivessel coronary artery vasospasm is a rare but important cause of morbidity and mortality. Coronary artery spasm is a vasoconstriction of coronary arteries that causes total or subtotal occlusion. It plays a prominent role in acute coronary syndrome. Coronary angiography and provocative testing usually are required to establish a definitive diagnosis. The mechanism of coronary artery spasm is poorly understood. We report a case of 75-year-old male, who presented with severe vasospastic angina that eventually progressed to ST-elevation myocardial infarction following percutaneous catheter interventions.

Keywords: Vasospasm; Coronary Intervention; Acute Coronary Syndrome

Case Presentation

A 75-year-old male was admitted with on and off the anginal pain of one-week duration. His ECG was showing ST-segment elevation with inverted T waves in inferior and lateral leads suggestive of evolved inferolateral wall myocardial infarction (Figure 1). The patient was pain-free at presentation. He was electively taken for coronary angiogram and revascularization. Coronary angiogram showed total thrombotic occlusion of the distal left circumflex artery (Figure 2A). The left anterior descending, right coronary and left main coronary arteries were normal. Left Coronary artery was cannulated with 6F, 3.5 curves extra back up guiding catheter. The lesion was crossed with 0.014" floppy guide wire and predilated with 2x10mm semi compliant balloon. A 3x28mm Drug Eluting Stent (DES) was deployed at distal circumflex at 18 atmospheric pressure. A 3x10 non-compliant balloon (NC sprinter) was used for post dilatation (Figure 2B). Post procedure, there was TIMI III flow in vessel and patient was asymptomatic with stable vitals. But after 4 hours of the procedure, the patient developed severe angina. The ECG was unremarkable at that time. He was subjected for a check angiogram to define the patency of stent. The coronary angiogram was unremarkable with TIMI III flow through the stented vessel (Figure 3A). He was started on coronary vasodilators keeping the possibility of micro vascular angina. He was also receiving intravenous GPIIb/IIIa inhibitor after the stenting since the lesion was thrombotic. After starting nitrates the pain subsided by 50%, again to reappear after 3 hours, but this time with ST elevation in anterior precordial leads. He was again taken for check angiogram. This time also the left circumflex had TIMI III flow with patent stent, but the left anterior descending artery had a diffuse spasm in its mid to distal segment (Figure 3B). Started with Intracoronary calcium channel blocker, sodium nitroprusside 100mcg and Nicorandil 5mg, Patient significantly improved after 30min and free of angina, patient discharged with antianginal medications prompt recovery on follow up.

Discussion

Coronary artery vasospasm is defined as transient total or near-total occlusion of a coronary vessel, occurring in either a normal or diseased arterial segment, which is reversible with isosorbide dinitrate [1]. It is a prominent cause of morbidity, both in patients with proven coronary artery disease and in those with "a variant form of angina pectoris" as originally described by Prinzmetal et al. [2]. If CAS lasts long enough, it can lead to angina and even myocardial infarction and sudden cardiac death [3]. However, brief episodes of CAS that go unnoticed can result in silent myocardial ischemia, or cause life-threatening arrhythmias [4]. The incidence of spontaneous CAS during CAG remains unknown but is widely considered to be rare. Atherosclerotic disease affecting large coronary arteries alters the vasomotor tone and reactivity of the affected vessels and intimate associations of spasm with sites of organic stenosis are well known. Mac Alpinreported that 88%of spasm cases causing ischemia were localized to the site of organic stenosis [5]. The mechanisms of CAS remain uncertain but are considered to be multifactorial. Many researchers believe that coronary spasm that develops during catheterization is partly spontaneous and partly catheter-induced. Electrocardiographic findings may appear normal at the beginning of CAS or when the CAS is mild. Total or subtotal spasm of a

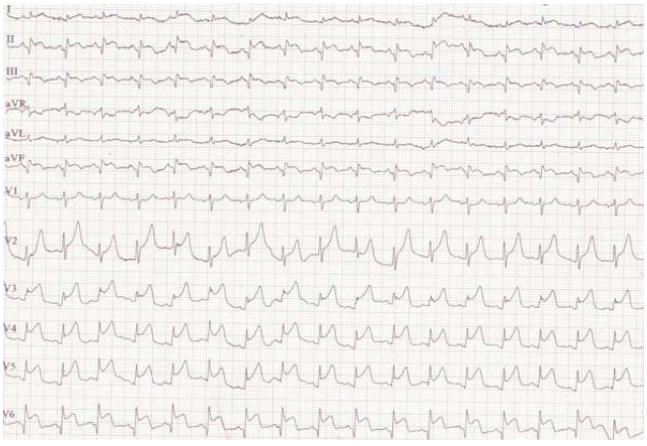

Figure 1: Electrocardiogram showing inferolateral MI.

Citation: NatrajSetty HS, Patil SS, Raghu TR, Kharge J, Kumar JRV, Geetha BK, et al. Malignant Vasospasm of Non Culprit Vessel Following Coronary Intervention- An Unusual Occurrence. Austin J Cardiovasc Dis Atherosclerosis. 2017; 4(1): 1033.

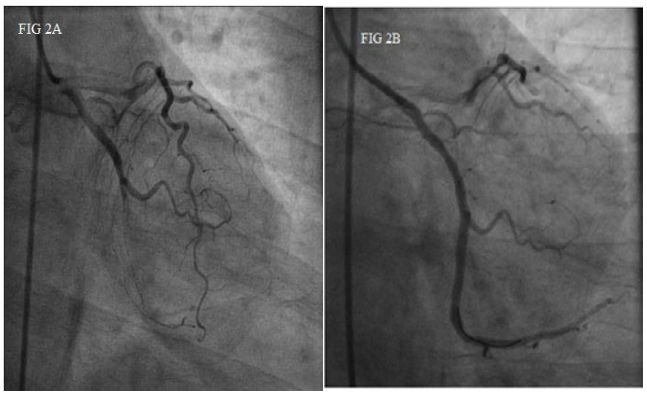

Figure 2: A: RAO caudal revealing mid LCX 100% occlusion. **B:** RAO caudal view showing patent LCX stent.

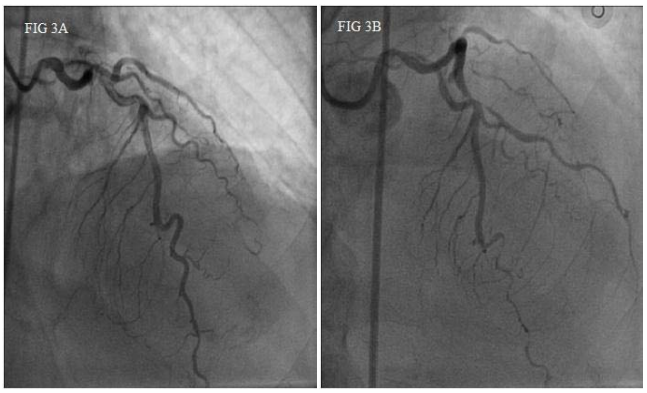

Figure 3: A: RAO cranial showing normal LAD. **B:** Diffuse Spasm of the distal LAD.

major coronary artery results in ST-segment elevation in the leads corresponding to the distribution of that coronary artery. However, CAS may cause ST-segment depression, indicating less severe, subendocardial myocardial ischemia than does ST-segment elevation. Of note, CAS is more frequently associated with ST-segment depression rather than ST-segment elevation [6]. Smoking, age and elevated high-sensitivity C-reactive protein (hs-CRP) are significant risk factors for CAS [7]. CAS can be precipitated by physical, mental stress, magnesium deficiency, alcohol consumption, the cold press or test, hyperventilation, the Valsalva manoeuvre, remnant lipoproteins, and the administration of pharmacological agents such as cocaine, sympathomimetic agents (epinephrine, nor epinephrine.), beta-blocking agents (propranolol), parasympathomimetic agents (methacholine, pilocarpine.), and ergot alkaloids (ergonovine, ergotamine.), particularly in the morning when spontaneous CAS is most likely to occur. Activated platelets may trigger CAS by releasing vaso constrictor substances; including thromboxane and serotonin, both of which are found to be associated with CAS [8-9]. We report an unusual case of vasospasm in non culprit LAD without organic stenosis. The patient had severe refractory vasospastic angina with coronary angiographic documentation of diffuse spasm of the non culprit LAD with eventual progression to ST-segment elevation myocardial infarction. The patient continued to be symptomatic despite treatment with different forms and doses of nitrates, calcium channel blockers, and GPIIb/IIIa inhibitor for few hours and Sodium Nitroprusside, Nicorandil later he improved symptomatically.

Conclusion

Coronary artery vasospasm can cause a transient, abrupt and marked decrease in the diameter of an epicardial coronary artery. Aetiological mechanisms implicated were all probably related to an exaggerated contractile response of the vascular smooth muscle in the affected coronary vessels. Cases involving spasm of non stented vessel are seldom reported. This is due to the hyper reactive coronary vessel and this should be suspected in appropriate case scenario.

References

1. Ozaki Y, Keane D and Serruys PW, "Progression and regression of coronary stenosis in the long-term follow- up of vasospastic angina," Circulation. 1995; 92: 2446-2456.
2. Prinzmetal M, Kennamer R, Merliss R, Wada T and Bor N, "Angina pectoris I. A variant form of angina pectoris. Preliminary report," The American Journal of medicine. 1959; 27: 375–388.
3. Maseri A, L'Abbate A, Baroldi G, et al. Coronary vasospasm as a possible cause of myocardial infarction. A conclusion derived from the study of "preinfarction" angina. N Engl J Med. 1978; 299: 1271-1277.
4. Yasue H, Kugiyama K. Coronary spasm: clinical features and pathogenesis. InternMed. 1997; 36: 760-765.
5. MacAlpin R. Correlation of the location of coronary arterial spasm with the lead distribution of ST segment elevation during variant angina. Am Heart J. 1980; 99: 555-564.
6. Cheng CW, Yang NI, Lin KJ, Hung MJ, Cherng WJ. Role of coronary spasm for positive noninvasive stress test result in angina pectoris patients without hemodynamically significant coronary artery disease. Am J Med Sci. 2008; 335: 354-362.
7. Hung MJ, Hsu KH, Hu WS, Chang NC, Hung MY. C-reactive protein for predicting prognosis and its gender-specific associations with diabetes mellitus and hypertension in the development of coronary artery spasm. PLoS One. 2013; 8: 77655.
8. Fernandez D, Rosenthal JE, Cohen LS, Hammond G, Wolfson S. Alcohol-induced Prinz metal variant angina. Am J Cardiol. 1973; 32: 238-239.
9. Oi K, Shimokawa H, Hiroki J, et al. Remnant lipoproteins from patients with sudden cardiac death enhance coronary vasospastic activity through upregulation of Rho-kinase. Arterioscler ThrombVasc Biol. 2004; 24: 918-922.

Austin J Cardiovasc Dis Atherosclerosis - Volume 4 Issue 1 - 2017
ISSN: 2472-3568 | www.austinpublishinggroup.com

Citation: NatrajSetty HS, Patil SS, Raghu TR, Kharge J, Kumar JRV, Geetha BK, et al. Malignant Vasospasm of Non Culprit Vessel Following Coronary Intervention- An Unusual Occurrence. Austin J Cardiovasc Dis Atherosclerosis. 2017; 4(1): 1033.