CASE REPORT

Coronary Artery Spasm (CAS) Simulating Inferior ST Elevation Myocardial Infarction (STEMI)

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SUMMARY

Coronary arteries vasospasm (CAS) is commonly seen in invasive cardiology laboratory during diagnostic catheterization or coronary intervention. Though the incidence of Printzmetal angina is uncommon, coronary vasospasm resulting in acute myocardial infarct is rare, especially if there is no significant atherosclerotic plaque within the coronary vasculature.

KEY WORDS:
Coronary artery vasospasm; Acute myocardial infarction

INTRODUCTION

We report a case of acute coronary syndrome (ACS) due to coronary artery spasm. In this hospital and the country, the most common cause of ACS is atherosclerosis. Though uncommon, CAS may explain why some patients presenting with ACS and has a normal coronary angiogram.

CASE REPORT

A 66 year old patient, with history of coronary artery disease diagnosed in 2004, presented to the district hospital with sudden onset of chest pain on 7 December 2008. Electrocardiography (ECG) showed ST segment elevation in the inferior leads suggestive of acute inferior ST segment myocardial infarction (STEMI) (figure 1). However, this was missed and as a result, he was not treated with thrombolytic therapy. Instead, he was given anti-platelet therapy with IV heparin. He was stable during the treatment. Of note, the ECG on 8 December 2008 showed resolution of the ST segment elevation in the inferior leads with no Q waves seen. The ECG on 10 December 2008 showed recurrent ST segment elevation in the inferior leads. He was transferred to this hospital on 11 December 2008 for further management.

He had previous percutaneous coronary intervention (PCI) to proximal Obtuse Marginal 1 (OM1) which was stented with BioDivYsio 2.75x8mm stent (Biocompatibles Cardiovascular Inc., California, USA) on 9 November 2004. When he arrived in this hospital, he was stable with no chest pain. ECG showed sinus rhythm with no ST segment changes.

His full blood count and renal profile and random glucose were all within normal limits. Of note, the creatine kinase was not elevated. After discussion with the patient, he agreed to undergo coronary angiogram.

Coronary angiogram was performed on 12 December 2008 via right femoral access. The diagnostic coronary angiogram showed diffuse disease in mid - distal left circumflex (LCX), focal disease 70% stenosis in mid obtuse marginal (OM1) after the stent and diffuse disease in proximal - mid right coronary artery (RCA) (Diagram 2a). The left descending artery (LAD) was normal, except for focal stenosis at distal LAD.

We decided to proceed to percutaneous coronary intervention to the RCA as we felt this was the culprit vessel. We used a Judkins Right guiding catheter (JR4) (Cordis Corp) to engaged the RCA and during the process, the ECG monitor showed ST segment elevation which resolved spontaneously. There was no damping of the blood pressure. We decided to re-engaged the RCA and injected 100 micrograms of glyceryltrinitrate (GTN) into the RCA. A repeat angiogram showed a normal RCA (diagram 2b). As the ST elevation resolved, his angina at the same time also resolved.

We re-engaged the left coronary artery (LCA) with a Judkins Left (JL4) (Cordis Corp) diagnostic catheter and injected 100 micrograms of glyceryltrinitrate (GTN) into the LCA. A repeat angiogram showed a normal LCA.

Patient was treated medically with anti-platelet, nitrate, calcium channel blocker and statin. Beta-blocker was not started as it was thought to be associated with vasospasm. He was discharged well on 13 December 2008. A 6 months follow-up in clinic, he remained asymptomatic.

DISCUSSION

Coronary artery vasospasm is the most common cause of non-obstructive lesions in acute coronary syndrome, the incidence of which is approximately 40-50% [1]. Prognosis of CAS greatly depends on coexisting atherosclerotic coronary artery disease (CAD). Survival at 5 years can be as high as 99% at 1 year and 94% at 5 years if there is no significant atherosclerotic CAD. On the other hand, survival falls to 87% at 1 year and 77% at 5 years in patients with multi- vessel CAD [1]. It is important to emphasize that most patients with CAS do not have ST-segment elevation myocardial infarction. Likewise, it is important to emphasize that absence of ST elevation during angina does not exclude CAS. CAS has been closely-linked with vulnerable plaques, thrombosis, percutaneous coronary intervention (PCI), and stenting [2]. Often, CAS has been overlooked during coronary angiography, and unnecessary stents deployment performed for the vasospastic vessels. In our case report, the patient had

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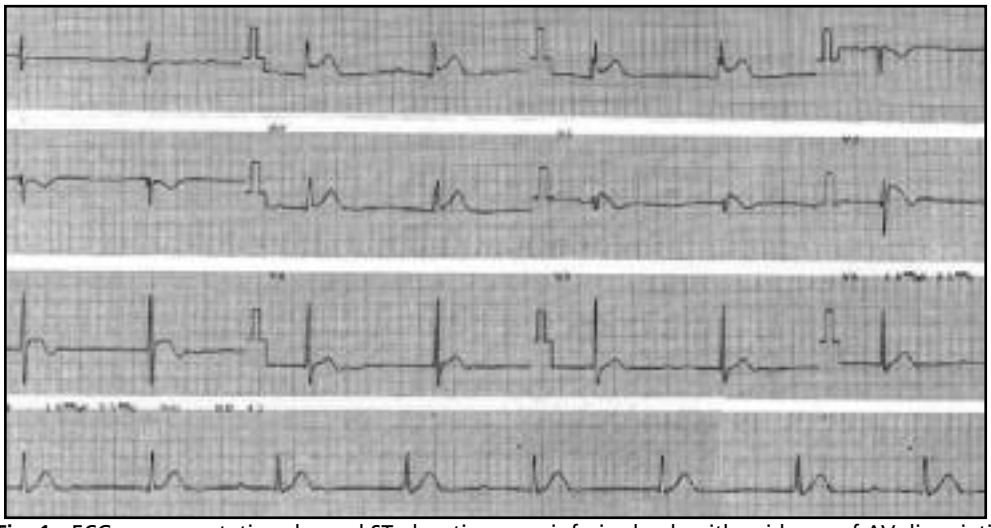

Fig. 1: ECG on presentation showed ST elevation over inferior leads with evidence of AV dissociation.

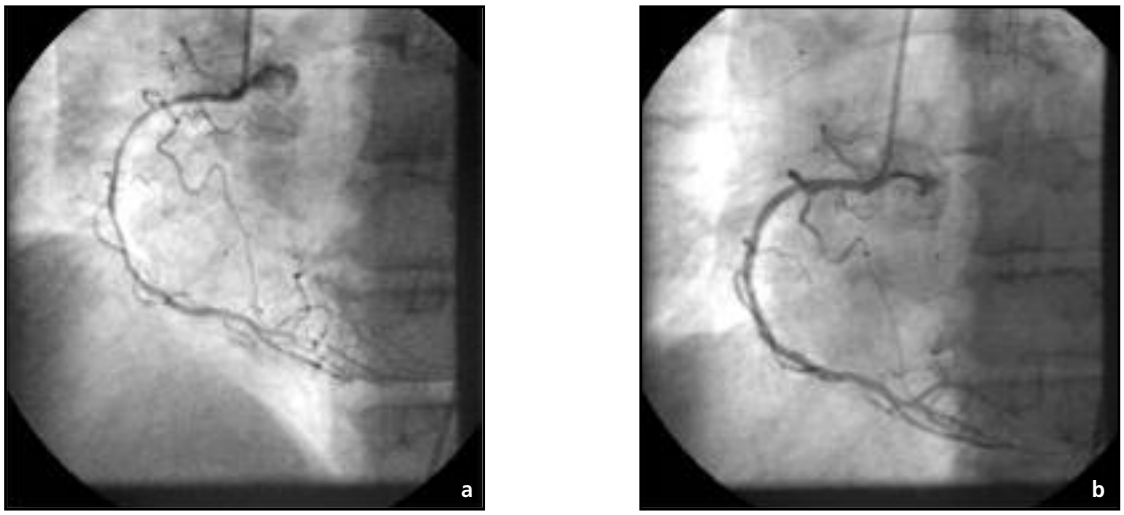

Fig. 2: Figure 2a showed apparent diffused CAD over RCA. Figure 2b showed normalized coronary artery after intracoronary glyceryltrinitrate administration without the need for stent deployment.

received coronary stenting to OM 1 in year 2004. Retrospectively, we felt that the initial OM 1 "lesion" may be non-obstructive, and thus may not require stenting.

The understanding of pathogenesis of CAS is still limited and uncertain. Hypothesis suggests that CAS occurred in patients with imbalance control in vascular smooth muscle tone. The mechanism for control of vascular tone involves activation of voltage-gated Ca2+ channel at the presynapse of sympathetic nerve. The final pathway is associated with the K-ATP channels in smooth muscle cells. It is likely that processes extrinsic to smooth muscle are also involved. Certain extrinsic factors are thought to have triggered CAS and this includes cocaine abuse, chest trauma, hyperventilation, and certain provocative agents such as acetylcholine, ergonovine, histamine, or serotonin. Other factors include hyperinsulinemia, magnesium deficiency, carcinoid crisis, pseudoephedrine, vitamin E and estrogen deficiency. In rare cases it appears to be a manifestation of generalized vasospastic disorder along with migraine headache and Raynaud phenomenon; it has also been reported with aspirin-induced asthma [2].

Literatures suggested few test to provoke vasospasm [3] includes acetylcholine provocation test. Definitive diagnosis remains diagnostic coronary angiogram. There is an interesting case report published in The Journal of Invasive Cardiology in 2007 by Vineeta Ahooja. This case report is on a patient with previous coronary artery bypass surgery and now is admitted for graft study. In this case report, all his diseased native arteries and graft vessels were normalised after given intracoronary nitrates [4]. This case report together with our current report would alarm the interventionist or cardiothorasic surgeon for possible unnecessary intervention in patient with CAS.

In conclusion, treatment of CAS includes nitrates and long acting calcium channel blockers. Beta-adrenergic blockers should be avoided in coronary vasospasm because of their propensity to increase the frequency and duration of attacks. It is important to consider CAS as cause of ischaemia, as it could be reversed with intracoronary nitrates, without the need for coronary stenting.

Conflict of Interest: None to declare.

REFERENCES

1. Onaka H, Hirota Y, Shimada S, Suzuki S, Kono T, Suzuki J *et al.* Prognostic significance of the pattern of multivessel spasm in patients with variant angina. Jpn Circ J. Jul 1999; 63(7): 509-13.
2. Gersh BJ, Braunwald E, Bonow RO: Chronic Coronary Artery Disease, Braunwald E, ed. Heart Disease: A Textbook of Cardiovascular Disease, 6th ed. Philadelphia, WB Saunders; 2001: 1324-8.
3. Kohei Wakabayashi, Hiroshi Suzuki, Yuki Honda *et al.* Provoked coronary spasm predicts adverse outcome in patients with acute myocardial infarction. JACC; 2008. Vol 52: 518-22.
4. Vineeta Ahooja, Deepak Thatai. Multivessel Coronary Vasospasm Mimicking Tripple Vessel Obstructive Coronary Artery Disease. J Invasive Cardiol; 2007. Vol 19(7): E178-81.