



SPONTANEOUS CORONARY ARTERY SPASM IN LEFT ANTERIOR DESCENDING ARTERY - A CASE REPORT

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Abstract :

Coronary artery spasm has been well documented during angiography. Coronary artery spasm should now be considered a well proven clinical entity which may or may not be associated with clinical symptoms and coronary atherosclerosis and may be either spontaneous or iatrogenically induced. The most important clinical entity associated with coronary spasm is that of Prinzmetal's angina. Patients who have variant angina are more likely to develop coronary spasm during angiography. Coronary artery spasm is also likely to occur during coronary angiography as a result of catheter irritation. Coronary artery spasm is commonly reported with right coronary artery. We report a case of spontaneous occurrence of coronary artery spasm in left anterior descending artery.

Keyword : Coronary Artery Spasm Left Anterior Descending Artery

INTRODUCTION:

Coronary artery spasm (CAS) is a dynamic and reversible occlusion of an epicardial coronary artery

caused by focal or diffuse constriction of the smooth muscle cells within the arterial wall. Coronary artery spasm quite common during coronary angiogram and is more commonly reported in right coronary artery. It can cause profound morbidity including myocardial infarction and mortality as significant proportion of patients continued to have symptoms despite optimal medical management. It is a variant form of angina initially described by Prinzmetal and colleagues (Prinzmetal or variant angina) in 1959. We herein report a case of CAS and management of such patients remained a debate with the absence of hard scientific evidence and guidelines.

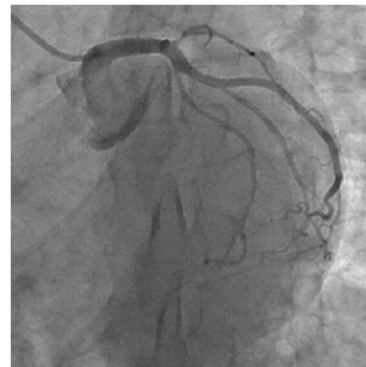
CASE REPORT:

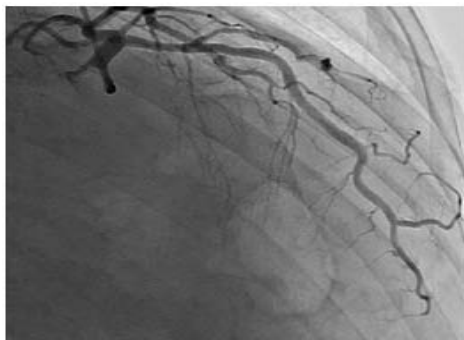
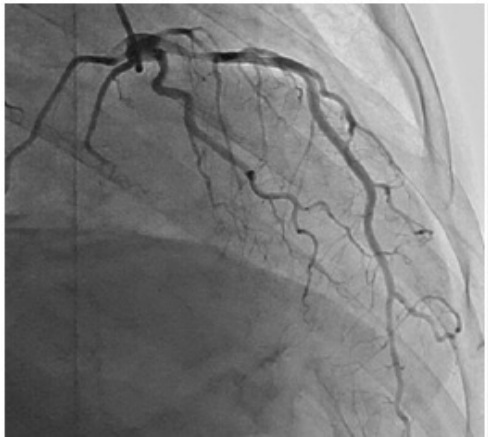
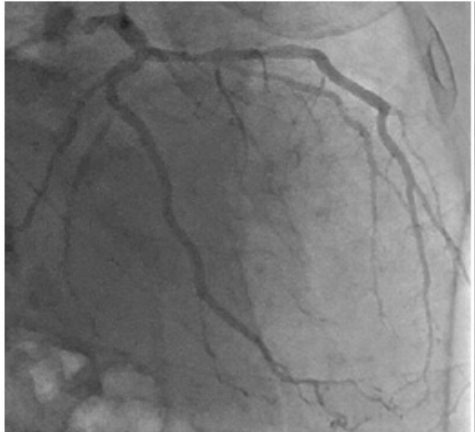
A 55 yr old male, chronic smoker and alcoholic, hypertensive for 2 yrs, was admitted with typical anginal pain and class II dyspnoea for 4 hr duration in the early hours of the day in our coronary care unit. He was anginal free before admission and his vitals and systemic examination were normal. ECG (a) showed ST segment elevation in v1 -v6, lead I and avL, with reciprocal changes in inferior leads.

Hypokinesia of anterior wall segment with ejection fraction 45% was noted in 2D ECHO. So we decided to thrombolyse the patient with streptokinase. Post lysis period was uneventful and patient was free of angina and ECG (b) showed resolved ST segment elevation in anterior leads. Patient was continued with medical management and was discharged in stable condition. 3 months after discharge, patient was readmitted with symptoms of class II Exertional angina with intermittent episodes of rest angina and Class II dyspnoea for the past one month. ECG and 2D ECHO were normal. As patient is symptomatic, we took the patient for coronary angiogram. It was done through radial route after adequate local anaesthesia and intra radial cocktail {Heparin 1000U, Diltiazem 30µg and Nitroglycerine (NTG) - 200µg}. Left Coronary Artery was engaged using 5F Tiger catheter in LAO View. In the LAO caudal (**Figure 1**)& RAO caudal view (**Figure 2**), after adequate contrast injection, LMCA and LCX showed normal flow and caliber but LAD was not visualized after D1. Patient developed angina with ST segment elevation in the anterior leads and angina relieved after Intracoronary (IC) NTG. In subsequent RAO caudal (**Figure 3**)and RAO cranial View(**Figure 4**), LAD was visualized with mid LAD showed 50% stenosis after D1 with distal normal calibre & TIMI II flow. Mid LAD showed normal calibre vessels with distal TIMI III flow on subsequent contrast injection in LAO caudal(**Figure 5**) and RAO Cranial views(**Figure 6**). As catheter tip was well away from the long segment of LAD spasm during angiography, this suggest high likelihood of having a Prinzmetal angina rather than a catheter induced spasm. Beta blockers and aspirin were withdrawn and patient was discharged from the hospital with diltiazem, clopidogrel and statins. Provocative test was deferred and patient was asymptomatic at discharge. The patient is on regular follow up.



Fig 1: ECG - At admission
Fig 2: Post thrombolysis
Fig 3: LAO Caudal





Discussion:

Coronary spasm can appear as an angiographic narrowing, provoked by mechanical stimulation, cigarette smoking, cocaine use, alcohol, and , acetylcholine, cold pressor testing, or hyperventilation ⁽¹⁾. Definitive diagnosis is demonstrated by relief of the narrowing either spontaneously or by nitrate administration initially described by Prinzmetal and colleagues (Prinzmetal or variant angina) in 1959, this form of angina was not provoked by the usual factors, such as exercise, emotional upset, cold, or ingestion of a meal⁽²⁾ Endothelial cell dysfunction has been proposed to explain coronary vasospasm.⁽⁵⁾ In response to increases in shear stress, platelet products, and other agonists, normal endothelial cells release endothelium-derived relaxing factor (nitric oxide), resulting in vasodilatation. When endothelium is damaged, as occurs with hypertension, elevated cholesterol, smoking, or use of cocaine, endothelial nitric oxide is reduced or lost. Thus, when platelets aggregate at such sites with release of vasoconstriction substances such as serotonin and thromboxane A₂, arterial smooth muscle cells contract, causing spasm. The prevalence is higher in Japanese and Korean populations than in the Western population, probably due to a combination of genetic and environmental factors⁽⁵⁾. The prevalence of myocardial infarction with normal coronary arteries is about 5%. It is more prevalent in women (10-25%) compared to men (6-10%). The reported incidence of coronary artery spasm is also higher in Asian than Caucasians.

Coronary artery spasm is one of the major potential initiating mechanisms of coronary thrombosis. It has been documented that the most likely cause of ST-segment elevation in a patient with normal coronary arteries is spasm. Thrombotic occlusion could occur with prolonged spasm and a myocardial infarction could develop. Prinzmetal's angina spasm is not confined to the right coronary artery, and the area of narrowing is usually at least 1 cm distal to the ostium. The narrowing is frequently eccentric and irregular, and it has a long (0.5-2.5 cm) segment. Our patient, presented with acute coronary syndrome and when subjected to coronary angiogram, coronary vasospasm was demonstrated in the coronary artery with catheter tip significantly away from the coronary ostium. The spasm and angina symptoms promptly reversed with nitroglycerine suggestive of Prinzmetal angina. This patient presented with an episodic pattern of symptom. The diagnosis can be made by demonstration of vasospasm by provocation test like acetylcholine challenge or intravenous methylergonovine. In most case reports, the diagnosis was based on the clinical and laboratory findings without provocation. The recently reported CASPAR study by Ong et al. took a large series of patients presenting with chest pain progressing to coronary angiography. 28% (138 of 488) had no demonstrable culprit lesion, and of these 86% underwent acetylcholine provocation testing. 49% (=42) demonstrated evidence of significant coronary spasm defined as a reduction in vessel calibre of at least 75% (1) Martínez-Sellés et al. demonstrated ergonovine induced spasm in 20% of 346 patients who had presented with chest pain and normal coronary arteries, and that inducible vasospasm was a positive predictor for future presentation with vasospasm (1) Around 25% of patients continue to have intractable angina despite optimal treatment. These episodes can be detrimental

and occasionally life-threatening when myocardial infarction or arrhythmias occur. They usually respond well to nitrates, and long-acting nitrates are useful in preventing attacks. Response to beta blockers in patients with Prinzmetal's angina is variable. Calcium channel blockers are effective in preventing the coronary artery spasm of variant angina and should ordinarily be prescribed in maximally tolerated doses.

Conclusion

Coronary artery spasm is a well documented cause of chest pain and can present as acute coronary syndrome. Coronary arteriography may demonstrate normal arteries or disease at a level inconsistent with symptoms. The stenosis may be dynamic, involving large or small and often multiple coronary arteries. It can cause acute myocardial infarction, including sudden death. Coronary spasm is more likely to occur in a patient with atherosclerosis. It becomes clinically challenging to differentiate a patient with iatrogenic spasm from that of a patient with Prinzmetal angina, since catheter induced spasm are likely to occur even in these patients. Though provocative test may be diagnostic, it is not commonly practised, and it is not feasible in a patient who had already had an acute coronary syndrome. We therefore suggest the use of intra-coronary nitrate to be mandatory in all patients with coronary stenosis or obstructions before considering angioplasty to avoid the potential pitfall of inappropriate coronary intervention.

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