

Chest pain with ST-T changes on electrocardiogram and localized stenosis on coronary angiography could be a coronary vasospasm

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ABSTRACT

Coronary vasospasm is characterized by chest pain at rest with ST-T changes on electrocardiogram and coronary angiography showing virtually normal coronaries. The definitive diagnosis requires the stimulation of coronary vasospasm using provocative agents, which can be life threatening. We present a case where localized stenosis of proximal left anterior descending artery was observed on the coronary angiography, which disappeared on subsequent views, and hence, coronary stenting was deferred and patient responded well to medical management alone.

Key words: Coronary vasospasm, Prinzmetal angina, variant angina

INTRODUCTION

Coronary vasospasm is characterized by chest pain at rest with ST-T changes on electrocardiogram (ECG) and coronary angiography showing virtually normal coronaries. Here, we present a case where localized stenosis of proximal left anterior descending artery (LAD) was seen on the coronaries angiography (CAG), which disappeared on subsequent views, and hence, coronary stenting was deferred and patient responded well to medical management alone.

CASE REPORT

A 38 year old man was admitted to a hospital with complaints of left sided chest pain at rest radiating to left

shoulder for 15-20 minutes. On examination, his heart rate (HR) was 70/minute, blood pressure (BP) 110/70 mmHg and S1 and S2 normal on examination. Electrocardiogram (ECG) showed ST depression > 2 mm in lead II, III, V4, V5, and V6. Patient gave history of similar pain at rest for 10-15 minutes, a day before admission. He was started on ecosprin 325 mg and clopidogrel 300 mg, and IV nitroglycerine (NTG). ECG changes along with chest pain disappeared soon after starting NTG. However, the patient had similar complaints of chest pain next day early morning while he was in hospital with ST depression in lead II, III, V4, V5, and V6, and those reverted with increasing the dose of NTG. Since the hospital lacked a coronary angiography facility, he was referred to our hospital for further management.

On admission in our hospital he was on NTG, HR: 80/min, BP: 120/70 mmHg and ECG was not showing any ST-T changes. Next day, he was posted for CAG. CAG was done through right femoral Artery which initially showed 90% stenosis in proximal LAD in right antero-oblique (RAO) caudal view. The decision for stenting was taken and on subsequent views, stenosis disappeared in antero-posterior (AP) cranial, lateral and RAO caudal view. After

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repeat dye injection, AP cranial view showed diffuse spasm from proximal to distal LAD and hence coronary artery spasm (CAS) was diagnosed. An intracoronary dose of NTG 100 µg and 2 mg of nicorandil was injected followed by repeat fluoroscopy with dye injection, which revealed resolution of the spasm and stenosis. Patient was diagnosed to have coronary artery spasm and was started on oral diltiazem 30 mg 8 hourly, and nicorandil 5 mg 12 hourly. He was discharged after one more day of observation.

DISCUSSION

Variant angina, first described by Myron Prinzmetal, is a form of angina which is characterized by chest pain at rest with ST-T segment elevation, and is caused by coronary vasospasm.^[1,2] However, coronary vasospasm is not synonymous with Prinzmetal's Variant angina (PVA), since the coronary vasospasm also includes angina with either ST depression and/or, exertional angina.^[3] The clinical features of coronary vasospasm include sudden syncope along with chest pain. The pain usually occurs in clusters, and two to three attacks in an interval of 30-60 minutes more commonly during midnight to 8 am are seen.^[2] The Japanese have the highest incidence of Prinzmetal's angina and it is uncommonly reported in the third world population.^[2] CAS is more common among males, and male prevalence increases with age.^[4] The other risk factors for CAS are smoking, alcohol intake, stress and lipid abnormalities.^[4]

The pathophysiology of variant angina is still under investigation; however, coronary artery endothelial dysfunction and increased vascular smooth muscle contractility are believed to be possible underlying mechanisms causing coronary vasospasm.^[5] The most commonly accepted hypothesis of this increase in coronary vasomotor tone is the decreased nitric oxide production by coronary arterial endothelium, and an imbalance in endothelium derived relaxing and contracting factors.^[2,5] There is also an increase in the amount of Phospholipase C, which shifts calcium from the intracellular compartment to the extracellular compartment; and hence, results in contraction of smooth muscle.^[6] The CAG is normal or near normal in up to 70% of patients with coronary vasospasm.^[7] There may be diffuse spasm of coronaries in others, with the right coronary artery as the most common site followed by LAD; and sometimes, it can be seen as triple vessel disease due to diffuse spasm of all three vessels.^[8] The definitive diagnosis of coronary vasospasm includes provocative stimulation of coronary arteries using hyperventilation test or drugs like acetylcholine and ergonovine.^[4] However, these provocative tests can cause serious complications.^[2] The management of coronary

vasospasm is mostly medical, and, includes calcium channel blockers and coronary vasodilator like nicorandil.^[2,4] In very resistant cases, culprit coronaries can be stented.^[9]

In our case, we found unprovoked stenosis in LAD, which was initially localized and hence could not be differentiated from thrombosis, and thus we decided to stent this blockage. As we were preparing to stent, the spasm resolved by itself, and thus the diagnosis of coronary vasospasm was made. The resolution of recurrent diffuse spasm after intracoronary injection of NTG and nicorandil confirmed the diagnosis of coronary vasospasm in our case. Hence concluding, one should be aware of the possibility of coronary vasospasm in a situation of coronary stenosis with absence of visible thrombus, as it could prevent an unnecessary stenting, and provide an equally effective outcome with the medical treatment alone.

CONCLUSION

Coronary vasospasm should always be considered in the setting of chest pain at rest with ST-T changes and localized stenosis in the absence of obvious thrombosis. This may prevent unnecessary coronary stenting of a condition which otherwise requires medical management.

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