



RESEARCH ARTICLE

ATYPICAL PRESENTATION AND CORONARY ANGIOGRAM OF A CASE OF VASOSPASTIC ANGINA

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ABSTRACT

Vasospastic angina is a rare disease which can present as an acute coronary syndrome emergency. This disease should be thought of in any atypical ECG not following the classic arterial territory involvement in a patient with acute chest pain. Also if there is evidence of long tubular narrowing with normal anatomy of other coronary arteries, then the probability of vasospasm should be kept in mind. Here we present a case of Vasospastic angina at our hospital which has atypical angiographic presentation.

Key words:

Vasospastic Angina,
Prinzmetal Angina,
Angioplasty,
Nitroglycerine.

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INTRODUCTION

Prinzmetal angina or Vasospastic angina is characterized by recurrent angina symptoms at rest and transient ST-segment changes. Prinzmetal angina can be a life-threatening condition with complications ranging from malignant arrhythmia and high degree AV-block to death. Therefore, prompt recognition of Prinzmetal angina is essential to avoid complications and provide appropriate management to improve the outcome for the patient.

Case report

A 48 yr old male non smoker, non alcoholic with no past history of hypertension, diabetes, ischemic heart disease or family history of coronary artery disease presented to casualty department with acute onset chest pain since 2 days, associated with dyspnoea and also low grade fever. In the casualty department he was diagnosed to have ACS/acute pericarditis by the index doctor. Admission ECG demonstrated ST-segment

elevation in all the anterior precordial leads and also inferior leads with a ST segment depression in the AVr lead. (Fig 1) A complete blood picture was done to rule out the diagnosis of acute pericarditis. Cardiac troponins were within the normal range. He was treated with anti-platelets and antianginals at the casualty department. His chest pain reduced within few minutes and the subsequent ECG did not show any of the features of an evolving myocardial infarction. So, he was electively posted for coronary angiography the following day to rule out the possibility of ACS. Pre-procedural ECG showed only a partial resolution of ST segment changes (Fig 2). On angiography, Left Anterior Descending artery, a type 3 vessel showed a long (> 30mm), tubular 80-85% isolated stenosis in the mid segment (Fig 3). The remaining coronary vessels appeared normal. Left Circumflex in (Fig. 4) and RCA in (Fig.5). Hence we prepared to stent the mid LAD segment. Due to the discrepancy between the ECG and angiographic findings, we did a Nitroglycerine 200 microgram injection before wiring the lesion to check the vasomotor response. After the NTG test we were surprised to note that the stenotic segment significantly dilated (Fig. 6). This clinical picture is consistent with vasospasm, suggestive of classical Prinzmetal disease.

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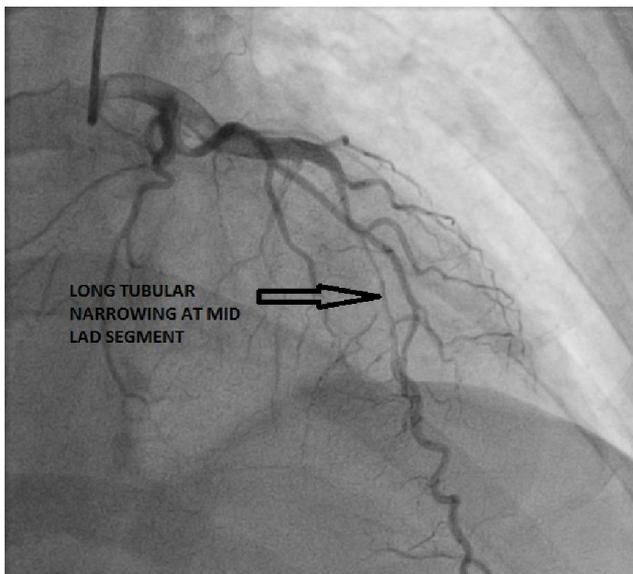


Fig. 3. Coronary angiography RAO cranial view showing a long tubular Mid LAD narrowing

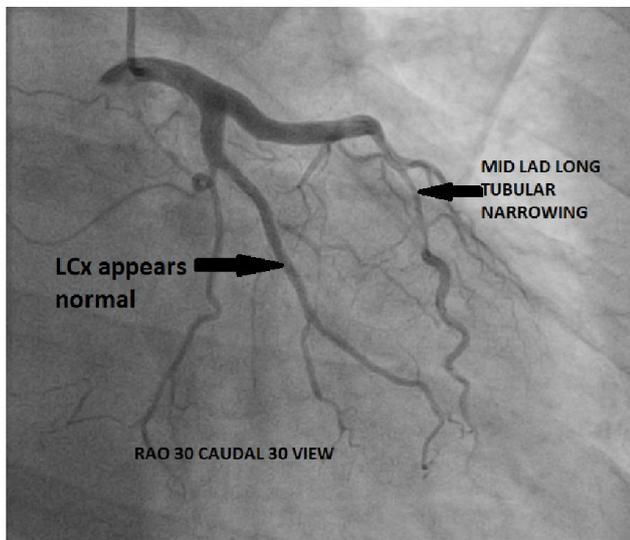


Fig. 4. Angiogram of Left Circumflex which is normal



Fig. 5. Angiogram of Right Coronary Artery which is normal

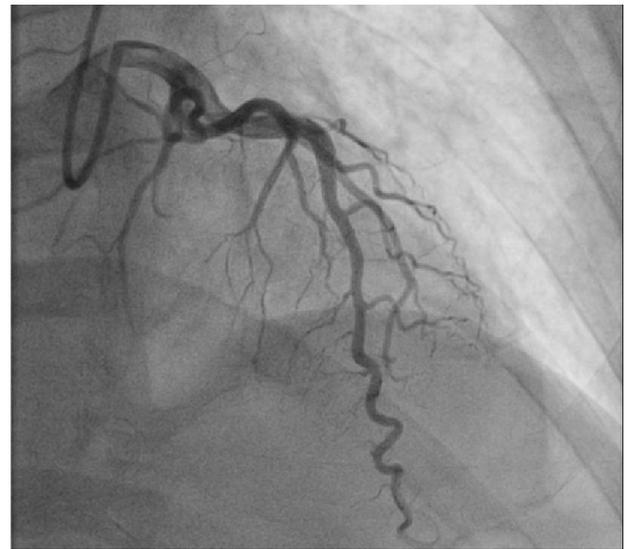


Fig. 6. After intracoronary Nitroglycerine 200 micro.gms the vasospasm in the Mid LAD segment showed significant dilatation

This is a rare angiographic presentation where only the mid LAD was affected.

DISCUSSION

The first case of Prinzmetal angina was described in 1959 by Prinzmetal *et al.* (Prinzmetal *et al.*, 1959) Since then, several trigger factors have been reported to be associated with Prinzmetal angina these include, illicit drugs such as cocaine, amphetamine or marijuana, but also bitter-orange, alcohol, butane, chemotherapy drugs, over-the-counter medication and different antibiotics. However, vasospastic angina can also occur without any triggering factor. (Stern *et al.*, 2009) The classical symptom being recurrent angina at rest with spontaneous remission. A circadian pattern has been noted and Prinzmetal angina preferentially occurs during the morning hours. Conflicting results exist on the pathophysiology. Proposed mechanisms responsible for this disease entity are hyperactivity of the sympathetic nervous system and vagal withdrawal or reduced nitric oxide synthase and endothelial dysfunction. While Egashira *et al.* (Egashira *et al.*, 1996) demonstrated enhanced Phospholipase C enzyme activity resulting in vasospastic angina without impairment of nitric oxide synthase. A genetic predisposition has also been discussed. (Murase *et al.*, 2004)

Complications comprise myocardial infarction, malignant arrhythmia and even sudden cardiac arrest or death. A complete AV block can also result in Stokes-Adam-attacks and patients should then be treated with a pacemaker. Therapeutic management consists of calcium-channel blockers and long-acting nitrates due to their vasodilatory effects. Furthermore Fluvastatin has demonstrated a positive effect on endothelial function and can therefore be recommended. (Yasue *et al.*, 2008) In patients with life-threatening arrhythmia an ICD should be considered. (Al-Sayegh *et al.*, 2007) The importance of this case lies in the unusual ECG presentation of vasospastic angina i.e. ST segment elevation of all the precordial and limb leads in the ECG. Even more peculiar was the focal nature of

the vasospasm which was seen only in mid LAD artery in the angiography.

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There is no Competing interest or conflict of interest for both the authors

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