Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/jicc

Case Report

Spontaneous coronary artery spasm during coronary angiography: An uncommon manifestation of variant angina

Thomas Hovasse, Pankaj Jariwala, Thierry Lefèvre*

Institut Cardiovasculaire Paris Sud, Massy, France

ARTICLE INFO

Article history:
Received 9 February 2014
Accepted 14 April 2014
Available online 15 May 2014

Keywords:
Prinzmetal angina
Variant angina
Coronary syndrome X
Coronary artery spasm

ABSTRACT

Spontaneous coronary artery spasm is an uncommon cause of angina pectoris and myocardial ischemia. Diagnosis usually depends on demonstration of coronary spasm following invasive provocative testing. We report a case of spontaneous coronary spasm leading to occlusion of the left anterior descending coronary artery and provide a brief overview of the literature.

1. Introduction

There have been numerous reports of angina or myocardial infarction in patients with otherwise normal coronary arteries. This has been attributed to syndrome X, Coronary artery emboli, or coronary vasospasm. Spasm of an epicardial coronary artery or coronary spasm, was documented angiographically following pharmacological provocation in the early 1970s.[1] However, unexpected single or multivessel spontaneous coronary artery spasm during cardiac catheterization is uncommon and has been reported only in few case series or reports.[2,3] Spontaneous coronary artery spasm during cardiac catheterization, which responds only to intracoronary nitrates is considered to be an uncommon manifestation of variant angina.[1] We discuss a case and possible pathophysiology of unexpected spontaneous coronary artery spasm which was provoked by coronary angiography or left ventriculography.

2. Case report

A 45-year-old Caucasian male was admitted with a two-month history of exertional retrosternal chest pain (Canadian Cardiac Society class II). Cardiac risk factors included dyslipidemia and a past history of smoking. There was no history of illicit substance abuse. Physical examination was unremarkable. Baseline electrocardiogram showed normal sinus rhythm without evidence of myocardial ischemia or

* *Corresponding author*. Indo–US Superspeciality Hospitals, Anand Bagh, Shyam Karan Road, Ameerpet, Hyderabad 500016, Andhra Pradesh, India. Tel.: +91 9393178738 (mobile).
E-mail address: pankaj_jariwala@hotmail.com (P. Jariwala).

http://dx.doi.org/10.1016/j.jicc.2014.04.006

infarction [Fig. 1a]. A myocardial perfusion study with Tc-99m MIBI revealed a severe stress-induced anteroseptal perfusion defect, with complete reperfusion on a subsequent resting Tc-99m MIBI study.

Coronary angiography demonstrated a non-significant lesion of the proximal left anterior descending coronary artery (LAD) [Fig. 1b]. After measurement of intracardiac pressure, left ventriculography was performed with 30 ml of nonionic contrast. Left ventriculography showed anterolateral and apical hypokinesia and an ejection fraction of 50% using the Dodge method [Fig. 1d]. Following left ventriculography, the ECG showed ST-segment elevation in the precordial leads [Figs. 1c and 2a], and the patient complained of mild retrosternal chest pain of a similar nature to previous episodes. No anaphylactic reaction to the contrast material was noticed.

A repeat left coronary angiography was performed, which illustrated an occlusion of proximal LAD (Thrombolysis in Myocardial Infarction flow grade 0) [Fig. 2b]. Spontaneous coronary artery vasospasm was thought to be the etiology of the LAD occlusion, thus intracoronary nitroglycerin 200 mcg was administered. Immediately, the chest pain and ST-segment elevation resolved and the LAD occlusion resolved to reveal focal LAD spasm at the site of the moderate plaque in the proximal LAD. Further angiography documented TIMI 3 blood flow [Fig. 3a and b]. The left circumflex and right coronary arteries remained normal angiographically. The patient was discharged after the following day after documentation of a normal transthoracic echocardiogram. At one-month, the patient was free of angina on maintenance calcium channel blockers and nitrates.

3. Discussion

Since its first description as an etiology of cardiac chest pain in 1959 by Prinzmetal et al, coronary artery spasm (CS) has been recognized as an infrequent cause of angina pectoris and myocardial ischemia in patients with normal or near normal coronary angiography.[4]

However, demonstration of spontaneous CS during coronary angiography is relatively uncommon.[5] The diagnostic value of spontaneous spasm during coronary angiography is definitive evidence of Prinzmetal's angina or variant angina.[6] In its absence, provocation tests using physiological maneuvers (hyperventilation) or pharmacological agents[6,7] are considered to be part of the routine investigation for confirming the spastic nature of chest pain.

The incidence of spontaneous coronary vasospasm has not been established, however, during coronary angiography, the incidence has been reported to be between 0.26% and 0.93%.[8] Coronary spasm is not restricted to normal coronary artery segments. Indeed the majority (60%) occurs at sites of coronary plaque. Recently, Hong et al compared the plaque components at coronary sites with focal spasm after ergonovine provocation test in 30 variant angina patients with those at culprit coronary sites in unstable angina patients, using virtual histology–intravascular ultrasound (VH–IVUS).[9]

Coronary spasm can be defined as an exaggerated contractile response of epicardial coronary artery smooth muscle to various stimuli in the absence of significant obstructive coronary artery disease (CAD). Though multiple factors

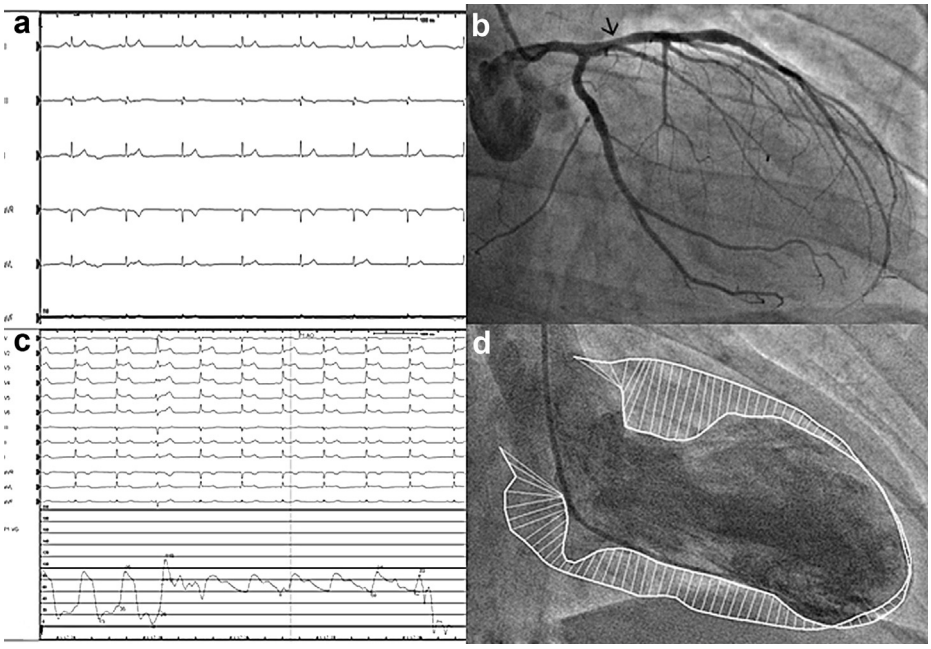

Fig. 1 – a–d: (a): Online ECG shows normal sinus rhythm and normal ST-segments before the start of angiography in a catheterization laboratory. (b): Left coronary injection showing mild lesion (plaque) in the proximal segment of left anterior descending artery (arrow) and normal circumflex artery. (c): Online ECG and pressure tracing (LV to aorta pullback) showing ST elevation in anterolateral leads at the end of left ventriculography and minimal fall in blood pressure. (d): Left ventriculography in systolic frame with border tracing for calculation of ejection fraction demonstrated akinesia of apical and anterolateral segments with EF of 50% being measured by Dodge method.