Coronary Artery Vasospasm with Acute Myocardial Infarction in a Male Patient: Case Report

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Abstract: We present a 48-year-old male patient with a history of classic precordialgia, ST-segment elevation in inferior leads (II, III and aVF), with troponin and CK-MB (creatine kinase-MB) elevated on the day of admission that received conventional anti-ischemic treatment. Hemodynamically stable, symptom-free, is referred to cardiac catheterization. In the selective catheterization of the left coronary artery, a moderate lesion was observed in the middle third of the anterior descending coronary artery; in the right coronary artery, a subocclusive proximal lesion with TIMI (thrombolysis in myocardial infarction) II. We performed a single projection and opted for angioplasty. Before, it was decided to re-evaluate the left coronary artery, and it was observed in the contrast test that anterior descending artery was occluded. Intra-coronary nitroglycerin and new contrast injection were performed, which showed totally open descending artery with TIMI III, without obstructive lesions as initially suggested, and improvement of pain. It was a severe coronary vasospasm. When a new contrast injection was performed in the right coronary, with a therapeutic catheter, the disappearance of the subocclusive lesion and the presence of a normal coronary flow were observed, although there was a moderate plaque in its proximal third, which motivated the maintenance of angioplasty with stent placement in a proximal third of right coronary artery. During the passage of the intracoronary guidewire it was once again evident that diffuse coronary artery vasoreactivity was present; the procedure was successfully completed.

Key words: Angina pectoris, variant; acute myocardial infarction.

1. Introduction

Vasospastic angina, first described in 1959, is characterized by ST-segment elevation and episodes of chest pain, usually unrelated to physical effort [1]. Previously known as Prinzmetal’s angina or variant of angina, is a clinical entity marked by episodes of angina at rest that respond promptly to the use of short duration nitrates and is attributed to coronary vasospasm.

The prevalence of vasospastic angina is not well studied. It appears to be more frequent in individuals from Japan compared to Caucasian populations [2]. The diagnosis is most often made in individuals less than 50 years of age than in older people. In addition, it is believed that in women it is more common to present variant angina, although some prognostic studies show a male preponderance [3-5].

Although coronary spasm usually occurs in a focal area, it can also be multifocal and affect more than one coronary vessel at the same time. Disruption of blood flow to a specific area can lead to severe myocardial ischemia. Most spasms are believed to occur in areas of stenosis, but spasms adjacent to atheromatous plaques have been observed in some patients.

Vasospastic angina is caused by focal or diffuse spasm of a coronary artery of the epicardium, which results in a high degree of obstruction. Many patients have transient myocardial obstruction as the cause for angina, and the persistence of the obstruction can lead to myocardial infarction. At the heart of the pathogenesis of vasospastic angina is hypercontractility of the vascular smooth muscle due to vasoconstricting mitogens, leukotrienes or serotonin,
which can occur in healthy or diseased vessels, without
a direct relation to the demand for oxygen by the
myocardium. Spasms may occur, in this sense, in
angiographically normal coronary vessels, but more
commonly in sites of atherosclerotic plaques of
variable severity.

Coronary spasm frequently affects only one segment
of an artery. However, it is a generalized process that
results in diffuse narrowing of the coronary artery. In
approximately 9% of cases, multiarterial spasm is
described. Normal or discretely abnormal coronary
arteriograms have been reported in approximately 40%
of patients with vasospastic angina.

Patients with variant angina have typical angina at
rest associated with transient ST-segment elevation on
ECG (electrocardiographic), especially at night or in
the early hours of the morning. In 30% of patients, it
can be associated with exercise. Although coronary
spasms tend to resolve spontaneously, when prolonged,
它可以 lead to myocardial infarction, arrhythmias, and
sudden death.

Definitive diagnosis is made when coronary
vasoconstriction is demonstrated at angiography and
reversible with nitroglycerin or calcium channel
blocker. According to the COVADIS (Coronary
Vasomotion Disorders International Study Group),
establishing the diagnosis of vasospastic angina should
include nitrate responsive angina, transient ischemic
ECG changes and angiographic evidence of coronary
artery spasms [6].

Episodes of Prinzmetal angina often occur in
clusters, with prolonged asymptomatic periods from
weeks to months. Seizures can be precipitated by
emotional stress, hyperventilation, exercise, or
exposure to cold. A circadian variation is frequently
present in episodes of angina, with most seizures
occurring early in the morning [7, 8]. Compared with
patients with chronic stable angina, patients with
variant angina are younger and, except for smoking,
have fewer coronary risk factors [7, 9].

In patients with a documented spontaneous episode,
the diagnosis can be made based on nitrate responsive
angina with associated transient ECG changes.
Occasionally, a spontaneous episode may occur during
diagnostic angiography so that all three elements can
be documented. However, when typical spontaneous
episodes cannot be frequently documented,
provocative tests are performed to make the diagnosis.
During the provocation test, the diagnosis of
vasospastic angina is confirmed if the stimulus induces
inductive chest pain, transient ECG changes and a
constrictive response > 90 percent.

The management of variant angina should initially
include the prevention or treatment of atherosclerosis,
either by medical intervention or lifestyle change, since
atherosclerosis is common in a patient with vasospastic
angina. The use of nitrates, calcium channel blockers is
the main forms of treatment for patients with variant
angina.

Sublingual or intravenous nitroglycerin often closes
episodes of variant angina rapidly and long acting
nitrates are helpful in preventing recurrence. Calcium
antagonists are extremely effective in preventing
arterioconary spasm of variant angina and should be
prescribed at the maximum tolerated doses. Similar
rates of efficacy were observed among the diverse
types of calcium antagonists. Prazosin, a selective
α-adrenoreceptor blocker, has also been shown to be
effective in some patients, while aspirin may increase
the severity of ischemic episodes. The response to
beta-blockers is variable. Revascularization may be
useful in patients with variant angina who also present
with discrete, ﬁxed and proximal obstructive lesions
[10-12].

This study aimed to present a case of variant angina
with a focus on diagnosis through coronary
angiography and treatment.

2. Case Report

A 48-year-old male patient presented to our hospital
complaining of precordialgia that irradiated to the
anterior cervical region and left upper limb. It started
without triggering factors, with an intensity of 7/10, with duration of 30-40 seconds, ceasing spontaneously, with 3 months of evolution and worsening in the last weeks. The pain also presented a longer duration (30 minutes), associated with profuse sweating, nausea and vomiting for which he sought care. This was a recurrent episode of chest pain for him in last weeks, but he never had before. The patient had no significant risk factors for coronary artery disease, nor previous comorbidities and use of medicine, and negative family history.

After a blood test and an ECG showing ST segment elevation in DII, DIII and aVF leads, conventional anti-ischemic treatment was instituted with improvement of symptoms and electrocardiographic pattern. Troponin levels were 1,070 IU/L and CKMB (creatine kinase myocardial band) 52 IU/L on the day of admission. The patient has been developed hemodynamically stable, free of symptom, being chosen to direct cardiac catheterization.

During cardiac catheterization, a moderate lesion was observed in the middle third of the anterior descending coronary artery in the selective catheterization of the left coronary artery, which is more evident in the cranial projection (Fig. 1a). In the selective catheterization of the right coronary, a subocclusive proximal lesion with slow TIMI (thrombolysis in myocardial infarction) II coronary flow was evident (Fig. 1b). Due to the apparent severity of the lesion, a single projection was made and opted for angioplasty at that time.

Before the treatment of the right coronary artery, it was decided to reassess the left coronary artery. A new selective catheterization of the left coronary artery, already observed in the contrast-test injection to confirm catheterization, was performed, when it was observed that the anterior descending coronary artery was occluded (Fig. 1c). At that moment, the patient began to report a typical pain irradiating to the upper limb with the registration of ST-segment elevation.

![Fig. 1  Coronary angiogram showing: (a) a moderate lesion in the middle third of the left anterior descending artery; (b) A subocclusive proximal lesion with slow TIMI II coronary flow of the right coronary artery; (c) the anterior descending coronary artery spasm; (d) total reperfusion of anterior descending artery with TIMI III flow without obstructive lesions; (e) the disappearance of a sub occlusive lesion and the presence of a normal coronary flow in right coronary artery; (f) angioplasty with stent placement in a proximal third of right coronary artery; (g) diffuse coronary artery spasm in right coronary artery induced by guidewire insertion.](image-url)
Intracoronary nitroglycerin and new contrast injection were performed, which showed total reperfusion of anterior descending artery with TIMI III flow (Fig. 1d), without obstructive lesions, as initially suggested, with pain improvement.

It was therefore diagnosed as a severe coronary vasospasm. When a new contrast injection was performed on the right coronary artery with a therapeutic catheter, the presence of a sub occlusive lesion disappeared with a normal coronary flow (Fig. 1e), although there was a moderate plaque in its third proximal, which led to the maintenance of angioplasty with stent placement in a proximal third of right coronary artery (Fig. 1f). During the passage of the intracoronary guidewire, diffuse coronary vasoreactivity (spasm) was evident (Fig. 1g). The procedure was successfully completed with an ideal result. On discharge, the patient was free of angina symptoms and had no side effects after treatment. It was initiated a dual antiplatelet therapy with aspirin and clopidogrel, associated with nitrates to control vasospastic angina and relief of sudden symptoms.

3. Discussion

Spasm often occurs in arteries without stenosis, but many patients with Prinzmetal’s angina have spasms adjacent to atheromatous plaques [13]. In the case described, the patient had a moderate plaque in the right coronary, which led to the implementation of an interventional therapeutic strategy with stent implantation in the proximal third of the coronary artery with optimal angiographic result.

Differently, in the anterior descending coronary was chosen to maintain clinical treatment, since after infusion of nitroglycerin the total resolution of the spasm was observed and there was no evidence of atherosclerotic plaque. It is believed that women are more commonly affected, but some studies show that is five times more prevalent in men, between 40 and 60 years, and has as only risk factor smoking [14]. It is uncommon, accounting for 2% of unstable angina investigated by coronary angiography [15].

The exact cause of the spasm is not well defined. Endothelial dysfunction is caused by decreased levels of nitric oxide and increased vasoconstrictors, as well as oxidative stress and inflammation. The autonomic nervous system also seems to be involved, since spasm is prevented by acetylcholine and alpha-adrenergic blockers. In some patients, it is a manifestation of vasospastic disorder and is associated with migraine, Raynaud’s phenomenon or asthma induced by aspirin [16, 17].

Complications may occur mainly during the first three months of evolution, such as incapacitating pain, acute myocardial infarction, as the patient presented with elevated cardiac markers (troponin and CKMB), atrioventricular block, extra systoles and ventricular tachycardia, and rarely ventricular fibrillation and sudden death [18]. Myocardial infarction and potentially fatal arrhythmias can occur in approximately 25 percent of patients not treated with vasospastic angina. Therapy that reduces the frequency of symptomatic episodes seems to decrease the frequency of life-threatening events, but the evidence is not robust [19].

Patients with vasospastic angina usually describe anginal symptoms, including pain or retrosternal pressure, with radiation to the neck, jaw, left shoulder, or arm, with each episode having many of the features of angina seen in patients with obstructive coronary artery disease. This may be particularly true if there is significant coexisting atherosclerosis [20]. Our patient, for instance, developed a classic chest pain with ST segment elevation and elevated cardiac markers, which implies to rule out acute coronary syndrome as a relevant differential diagnose of variant angina.

Notably, symptoms associated with vasospastic angina usually occur at rest and may exhibit a circadian pattern, with most episodes occurring in the early hours of the morning [21]. Each episode of resting pain usually lasts for 5 to 15 minutes and presents ischemic changes of the ST segment in an ECG during an
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episode of chest discomfort, which returns to the baseline in the resolution of symptoms. In severe cases, associated arrhythmias may occur, ranging from cardiac block to ventricular tachycardia [22]. Patients without obstructive coronary disease, on the other hand, have a good long-term prognosis.

For most patients who present with typical chest pain, including those whose episodes occur predominantly at rest (and in whom vasospastic angina is a diagnostic possibility), the diagnostic evaluation should focus on fixed obstructive coronary artery disease. Vasospasm of the coronary artery is an important cause of chest pain, but not the exclusive cause. Coronary atherosclerotic disease, for example, is much more common, and its presence confers a worse prognosis. In this sense, this initial approach is reasonable given the significantly greater probability of fixed disease compared to vasospastic angina. This assessment usually involves stress testing in patients with normal troponin values. Therefore, adequate evaluation and interventions for both diseases should be considered in most of these patients.

Multiple triggers have been associated with the development of vasospastic angina. Several drugs such as ephedrine and sumatriptan can cause typical chest pain due to coronary spasm. Coronary spasm is also associated with smoking, cocaine, amphetamine, marijuana and alcohol consumption, which can often explain myocardial infarction in young patients with few traditional cardiovascular risk factors. Environmental factors such as cold weather can cause spasms in the coronaries. Valsalva maneuver, hyperventilation, and coronary manipulation through cardiac catheterization also can produce hyperreactivity of the coronaries.

On the other hand, technical limitations of noninvasive and invasive coronary evaluations can lead to misleading results. For example, a coronary event caused by the rupture of a nonobstructive atherosclerotic plaque with thrombosis, followed by spontaneous reperfusion, may lead to a seemingly normal or innocuous coronary evaluation. In this situation, a diagnosis of vasospastic angina can be misapplied, and important interventions to treat and prevent other atherosclerotic events may be delayed. A complete reassessment of the clinical syndrome should accompany an angiographic result or other unexpected diagnosis.

The main challenge of the treatment is focused on decreasing episodes of angina and preventing complications like myocardial injury and arrhythmia. Initial medical treatment should include cessation of smoking and pharmacological therapy—especially the use of calcium channel blockers. Avoiding medications or drugs that can trigger coronary vasospasm (e.g., cocaine, marijuana, and ephedrine-based products) is also important. Nitroglycerin given by any via effective treats episodes of angina and myocardial ischemia within a few minutes, and long-acting nitrate preparations reduce the frequency of recurrent events.

Until atherosclerotic coronary disease (a much more frequent cause of chest pain) is excluded, standard therapies, including antiplatelet or antithrombotic agents, statins and beta blockers, may be given. Statin therapy appears to improve clinical outcomes in patients with acute myocardial infarction induced by coronary spasm in non-obstructive coronary arteries [23]. Once the diagnosis of coronary artery vasospasm is made, calcium channel block and long-acting nitrates can be used for long-term prophylaxis.

Calcium channel blockers, as nifedipine, amlodipine, verapamil, and diltiazem effectively prevent coronary vasospasm and variant angina, and they should be administered preferably to beta-blockers. Amlodipine may be preferable because of its long half-life [24].

Beta-blockers are beneficial in most patients with atherosclerotic coronary stenosis and angina pectoris of exertion and are sometimes useful in combination with the above medications to control the symptoms in these patients. However, non-selective beta-blockers may be harmful in some patients because beta-receptor blockade, which mediate vasodilation, allows
unopposed alpha receptor-mediated coronary vasoconstriction to occur and may worsen vasospastic angina in selected cases. Thus, non-selective beta blockers, such as propranolol, should be avoided. In addition, aspirin should be used with caution, because at high doses it is an inhibitor of prostacyclin production.

Other agents have been tested with variable success, including endothelin antagonists such as bosentan [25]. Initial experience with cilostazol has been positive but limited [26]; additional research is needed to validate its clinical use. Revascularization may be useful in patients with variant angina who also present with discrete, fixed and proximal obstructive lesions [10-12]. The prognosis of variant angina is usually excellent in patients who receive medical therapy, especially in patients with normal or near-normal coronary arteries [27].

Prinzmetal’s angina is an important differential diagnosis of acute coronary syndrome and should always be remembered, once early diagnosis favors a better prognosis when associated with pharmacological measures and changes in lifestyle.

4. Conclusions

A coronary vasospasm should be considered as resulting from extensive vasomotor and endothelial dysfunction of the coronary circulation. It remains highly challenging to diagnose but has important symptomatic and prognostic implications. Therefore, variant angina should be considered in patients with and without typical cardiovascular risk profiles, and medical management should include medications to control vasospasm. In our patient, the angiographic finding compatible with coronary artery spasm during the procedure was the confirmatory evidence that he had Prinzmetal’s angina. Aside from symptoms of chest pain, coronary spasm can cause infarction, left ventricular impairment, promote life threatening arrhythmias and ultimately sudden cardiac death. Hence, it should always be remembered as a relevant differential diagnosis of acute coronary syndrome and needs to be monitored over time.

References


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