Coronary Balloon Angioplasty in a Severe Takotsubo Syndrome

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ABSTRACT

We reported a patient with Takotsubo syndrome, with severe symptoms, prolonged angina with hemodynamic compromise, in the context of severe coronary artery spasm, without response to full medical treatment, which was successfully managed with coronary balloon angioplasty. A 49-year old woman was admitted with chest pain, ECG changes and elevated myocardial necrosis markers suggestive for acute coronary syndrome. First coronary angiography revealed normal epicardial arteries and typical left ventricular apical ballooning, strongly suggestive for Takotsubo syndrome. Forty-eight hours later, with standard medical treatment, patient developed again severe angina with hemodynamic consequences. Second angiography showed total occlusive spasm of one coronary artery, without response to full medical treatment. Coronary balloon angioplasty was performed with final good result. Two months later, angiography revealed normal coronary arteries and normal ventricular shape. The patient is currently asymptomatic.

As far as we know, no other examples of similar cases were published in medical literature. Therefore, interventional treatment can be taken into consideration for some particular types of patients with Takotsubo syndrome, non-responsive to medical treatment; despite of balloon angioplasty or stenting of coronary vasospasm is not a standard of care.

Keywords: Takotsubo syndrome, balloon angioplasty, coronary artery spasm

CASE REPORT

A 49-year old woman, with no significant medical history, presented to the Emergency Department accusing severe chest pain and dyspnoea for 20 minutes. Symptoms had started two days before, intermittently. Patient was very anxious and stressed (she was divorced and she had lost her job recently). She wasn’t smoker or illicit drugs’ consumers, and had no medical treatment at home. The first electrocardiogram showed sinus rhythm, with Q waves in the anterior leads, and large negative T waves from leads V2 to V5 (Figure 1). Echocardiography revealed preserved basal left ventricular function, with apical ballooning, no intraventricular pressure gradient and no valvular...
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...disease. The global left ventricular ejection fraction was 35%.

Patient received standard medication for acute coronary syndrome: aspirin 250 mg, clopidogrel 600 mg, enoxaparin 60 mg subcutaneous, and perfusion of nitroglycerine. After 10 minutes, all symptoms disappeared. Coronary angiography and ventriculography were performed after 30 minutes and revealed normal coronary arteries, with a particular ventricular shape with systolic ballooning of the apex and hypercontraction of the basal segments (Figure 2A, B, and C).

For two days, the patient was free of symptoms on standard medication (aspirin, clopidogrel, diltiazem, statin, and nitrates). Her blood pressure was low, so, she didn’t receive any ACE inhibitors. Seventy two hours after the first angiogram, she accused again severe chest pain, with bradycardia and large T waves in the anterior leads and minor ST segment elevation in the lateral leads. Coronary angiography was repeated, and revealed normal right coronary artery (RCA), left main (LM) and proximal left anterior descending artery (LAD) with mild vessel spasm and Thrombolysis in Myocardial Infarction (TIMI) 3 flow, and proximal circumflex coronary artery (LCX) with severe spasm, with limitation of contrast media progression and TIMI 1-2 flow (Figure 3A). Left ventriculography showed the same aspect.

Patient received three intracoronary boluses of 200, 400 and 600 mcg of nitroglycerine, with no effect. Then, she received three intravenous boluses of verapamil and two intravenous boluses of adenosine. However, patient continued to complain of severe angina, with ST segment elevation on the monitor. She also received analgesics (morphine) for pain and for cutting her adrenergic response. But, meanwhile, a large obtuse marginal (OM1) became totally occluded (TIMI 0 flow) and also distal LAD flow became TIMI 2, with severe spasm (Figure 3B). At this point, the patient had severe chest pain, with bradycardia and low blood pressure, and one total occluded coronary artery, with severe persistent spasm on the others, without response to all given vasodilators.

Our decision was to perform angioplasty of proximal circumflex artery. A 6Fr JL4SH guiding catheter (Launcher, Medtronic) was used, and two 0.014” wires (Asahi Prowater, Abbott) were placed with difficulty in the distal OM1 and the distal LCX (Figure 3C). One soft inflation was performed with a 2.0x14 mm balloon Jocath Mercury (Abbott) in the proximal part of LCX, up to a maximal pressure of 6 atm, for 30 seconds. Consequently, distal flow in the OM1 became TIMI 3, and the chest pain decreased significantly.

Angiographic control of proximal circumflex coronary artery revealed mild residual spasm and no dissection. A normal anterograd flow was observed in the LAD and LCX. The result was considered optimal, with no further indication for stenting (Figure 3D).

The myocardial scintigraphy performed a week later was normal, without perfusion defects and with normal systolic function (Figure...
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4 D). Patient was discharged the next day on aspirin (75 mg/day), diltiazem (90 mg/day), atorvastatin (40 mg/day), and nitrates. ECG was in sinus rhythm, with persistent Q waves and negative T waves in the anterior leads.

Two months later patient needed a cardio exam to be hired and angiography was repeated. It showed normal coronary arteries and normal ventriculography (Figure 4A, B and C). The ECG was normal. Patient remained asymptomatic, on same medication. 

DISCUSSION

Takotsubo cardiomyopathy or syndrome is also known as “neurogenic myocardial stunning”, “stress cardiomyopathy”, “stress-induced cardiomyopathy”, “transient left ventricular apical ballooning”, “ampulla cardiomyopathy” or “broken heart syndrome”. “Takotsubo” is the Japanese name for traps that fisherman still use to catch octopus. In this syndrome, the left ventricle takes the shape of this particular trap due to a state of complete exhaustion of the heart muscle (myocardial stunning) in the mid-section and the apex of the heart. The first cases of this cardiomyopathy have been reported in Japan in 1990, and more recently in the US and other European countries. Typically, the syndrome appears after an acute emotional stress, patient describing chest pain (suggesting an acute coronary syndrome), with significant ECG changes (ST elevation or large T negative waves or diffuse nonspecific ST-T abnormalities), and biomarkers of heart damage (troponin, creatin kinase), but with complete resolution of the symptoms and the apical wall motion abnormality.

Mayo defined four clinical criteria for the diagnostic of Takotsubo syndrome: (1) specific shape of left ventricle, (2) absence of obstructive coronary artery disease or plaque rupture, (3) ST elevation or T wave inversion or biomarker elevation, (4) absence of myocarditis or pheochromocytoma (1-4).

One of the main causes of Takotsubo syndrome was suggested to be multivessels, diffuse coronary artery spasm (7).

The management of this syndrome is primarily symptomatic and includes monitoring and treatment of possible complications (acute left ventricle failure, pulmonary oedema, ventricular arrhythmias, etc.) (5-9).

The main differential diagnostic is Prinzmetal angina. Vasospasm is involved in both syn-

FIGURE 3. Angiography during severe chest pain (72 hours after admission). A. Severe spasm of the proximal LCX, with TIMI 1-2 flow; B. Spasm of the middle LAD, and total occlusion of the first marginal; C. Severe spasm of LAD and two wires were placed with difficulty in LCX; D. Final result after balloon angioplasty showing release of the coronary spasm.

FIGURE 4. Follow up. A. Normal follow-up left coronary angiogram; B. Normal follow-up right coronary angiogram; C. Normal follow-up ventriculography; D. Normal follow-up myocardial scintigraphy.
CONCLUSIONS

We reported a patient with Takotsubo syndrome, with severe symptoms, prolonged angina with hemodynamic compromise, in the context of severe coronary vessel spasm, without response to full medical treatment. An interesting and unique finding of this case is that vasospasm was relieved with coronary balloon angioplasty (a procedure that itself can induce further vasospasm). Balloon was inflated up to 6 atm, so no artery injury was produced. As far as we know, no other examples of similar cases were published in medical literature. Therefore, interventional treatment can be taken into consideration for some particular types of patients with Takotsubo syndrome, non responsive to medical treatment, despite of balloon angioplasty or stenting of coronary vasospasm is not a standard of care.

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