Journal of Cardiology (2008) **52**, 300—304

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JOURNAL of CARDIOLOGY
Official Journal of the Japanese College of Cardiology
www.elsevier.com/locate/jjcc

CASE REPORT

Coronary vasospastic crisis leading to cardiogenic shock and recurrent ventricular fibrillation in a patient with long-standing asthma

Gulten Tacoy (MD)[a,*], Sinan Altan Kocaman (MD)[a], Serhat Balcioğlu (MD)[b], Aslı Tanindi (MD)[a], Murat Özdemir (MD)[a], Mustafa Cemri (MD)[a], Atiye Çengel (MD)[a]

[a] *Gazi University, Faculty of Medicine, Cardiology Department, Besevler 06500, Ankara, Turkey*
[b] *Kastamonu Devlet Hastanesi, Cardiology Department, Kastamonu, Turkey*

Received 1 April 2008; received in revised form 26 May 2008; accepted 28 May 2008
Available online 29 July 2008

KEYWORDS
Coronary vasopasm;
Asthma bronchiale;
Cardiogenic shock;
Ventricular fibrillation

Summary Acute myocardial infarction in patients with normal coronary arteries is a therapeutic dilemma. Coronary artery vasospasm and thrombosis are the most commonly encountered clinic problems and appear in localized coronary segments. The incidence of cardiovascular disease is increased in asthmatic patients. β_2-Adrenergic agonists use is associated with increased cardiovascular events. Although myocardial ischemia during asthma has been described in literature, acute myocardial infarction and ventricular fibrillation with normal coronary arteries in patients with asthma bronchiale is a rare entity. Our patient with long-standing asthma bronchiale presented with cardiogenic shock whose coronary angiography revealed multivessel disease and undergone primary percutaneous coronary intervention. Due to ongoing chest pain and hemodynamic instability; an early bypass surgery was planned. A control angiogram was performed before surgery. After intracoronary nitrate administration all narrowings in coronary vasculature disappeared. Symptom relief and clinical improvement was achieved with nitrate and calcium channel blockers later. After 2 months she was readmitted with cardiac arrest due to recurrent ventricular fibrillation and intracardiac defibrillator implantation was performed.
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Introduction

Acute myocardial infarction in patients with normal coronary arteries is a therapeutic dilemma. Hypercogulability, inflammation, coronary embolism,

* Corresponding author. Tel.: +90 3122025633; fax: +90 3122129012.
E-mail addresses: gtacoy@gmail.com (G. Tacoy), sinanaltan@gmail.com (S.A. Kocaman).

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doi:10.1016/j.jjcc.2008.06.003

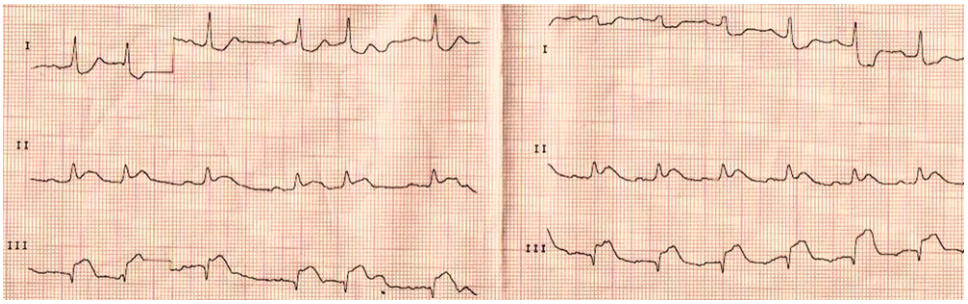


Figure 1 ECG findings on admission.

increased sympathic activation, and coronary artery spasm are the possible proposed pathways in this setting [1,2]. Coronary artery vasospasm and thrombosis are the most commonly encountered clinic problems and appear in localized coronary segments. During receiving beta agonist agents due to long-standing asthma multifocal and multivessel coronary vasospasm is relatively uncommon.

We describe a patient with long-standing bronchial asthma who had an acute myocardial infarction and cardiogenic shock whose coronary angiography resembled multivessel disease and undergone primary percutaneous coronary intervention although symptom relief and clinical improvement was achieved with nitrate and calcium channel blockers (CCBs) later. After 2 months she was readmitted with cardiac arrest due to recurrent ventricular fibrillation.

Case report

A 56-year-old woman was admitted to the emergency room complaining of severe chest pain. She gave a history of hypertension and long-standing bronchial asthma. One week before this presentation she was admitted to another hospital with a history of severe chest pain and severe dyspnea. Her chest pain was occurred during exercise and rest and radiated to back, neck and could wake her from sleep for 1 year. Initial evaluation in that hospital demonstrated ST-T changes on ECG. Therefore she was transferred to coronary intensive care unit where cardiac arrest developed and she was entubated. After extubation her hemodynamic impairment continued and she was referred to our hospital on 5th day. Her treatment consisted of Quinapril/hydrochlorothiazde 1× 20/12.5 mg, Trimetazidine 3× 20 mg, Isosorbid-5-mononitrate 1× 50 mg, Tiotropium Br 1× 18 μg, Formoterol fumarat 2× 12 μg, Theophylline 2× 200 mg, Montelukast sodium 1× 5 mg/day.

In our emergency room, she had severe dyspnea. Her physical examination revealed a blood pressure 100/60 mmHg, pulse 80 per minute without additional pathologic findings. ST segment elevation in inferior and right ventricular derivations were determined on admission. ST segment elevation increased with severe chest pain and her blood pressure decreased to 70/40 mmHg with 2:1 AV block on her ECG (Fig. 1). Troponin T level was 2.47 and other laboratory parameters were normal. She had no history of diabetes mellitus, smoking, dyslipidemia and family history for cardiovascular disease. Primary percutaneous intervention was preferred as a treatment approach. On coronary angiogram, there was a severe stenosis of the LAD in its midportion with severe narrowing of the diagonal branch and two sequential severe narrowing in mid-CX (Fig. 1). Her blood pressure was 60/40 mmHg during coronary angiographic examination. Nitroglycerine was not administrated because of severe hypotension. After injection radiocontrast material to left main coronary artery ventricular fibrillation developed and converted to sinus rhythm with 360 J defibrillation. Then right coronary artery was visualised and a severe narrowing in mid-RCA was found (Figs. 2 and 3). Although TIMI II flow was observed in the whole coronary vasculature, due to ECG findings and the dominancy of RCA, the culprit lesion was assumed to be mid-RCA and intervention was planned accordingly. Predilatation was performed with 2.5/20 mm balloon catheter. A bare metal stent (3.0/18 mm) was placed in the mid-RCA and her blood pressure increased to 130/80 mmHg. In follow-up at catheterization laboratory her blood pressure decreased with chest pain and dopamine infusion was started. An intraaortic balloon pump was inserted. She was transferred to coronary intensive care unit. In her control ECG ST elevation in inferior leads decreased (Fig. 4). Two-dimensional echocardiogram demonstrated posterior hypokinesia, minimal pericardial effusion, left ventricular EF 58%. Aspirin (100 mg), Clopidogrel (1× 75 mg/day

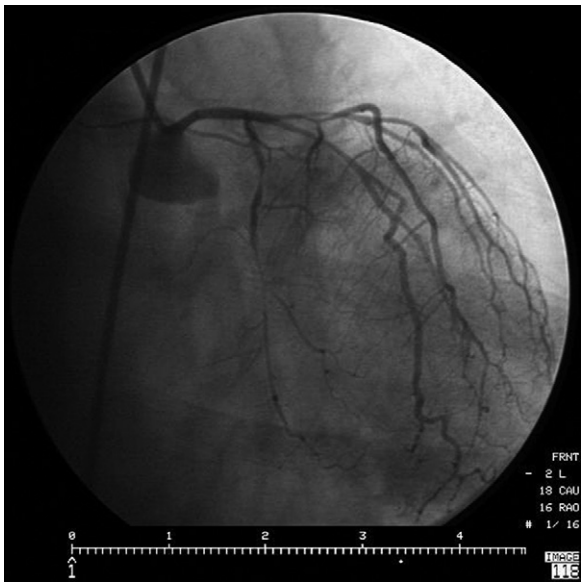


Figure 2 Coronary angiographic evaluation of left coronary artery.

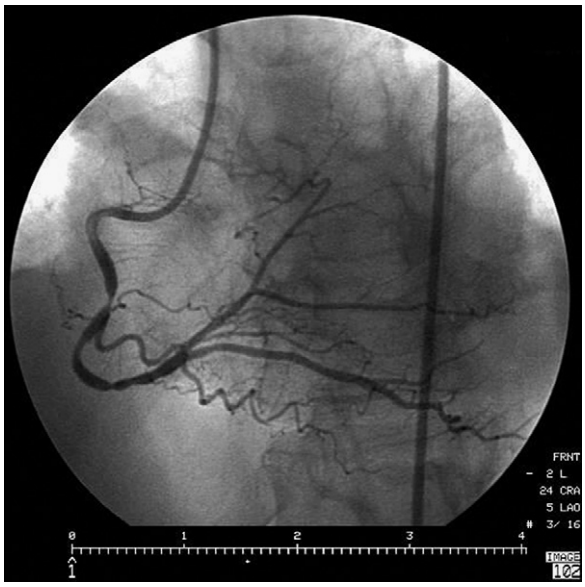


Figure 3 Coronary angiograhic evaluation of right coronary artery.

with a 300 mg loading dose), Atorvastatin (1× 10 mg), UF heparin, dopamine infusion, Ipratropiyum bromur (20 μg/day), Salbutamol sulfat (120 μg/day) were administrated.

On follow-up, her dyspnea aggravated and in physical examination bilateral wheezes and rales were found. Intravenous diuretic infusion was applied but her symptoms did not improve. On 3rd day the support of intraaortic balloon pump was ceased due to paleness of the extremity. Ventricular fibrillation developed on 5th day under the support of dopamine infusion and treated by defibrillation. At this stage due to ongoing chest pain and hemodynamic instability; an early bypass surgery for her left coronary system was planned. A control angiogram for her RCA was performed before surgery. The presence of new stenosis not evident on the first angiogram hinted the diagnosis of vasospasm. After 200 μg intracoronary nitrate administration there was complete disappearance of all lesions in patent and normally appearing coronary arteries (Figs. 5 and 6). Left ventriculogram demonstrated posterobasal and inferior hypokinesia. Although severe hypotension (70/40 mmHg) persisted for nearly 6 h after the intervention dopamine was withheld and long acting oral nitrate (2× 20 isosorbid-5-mononitrate), CCB (diltiazem 3× 90 mg) were given to patient. We gave the medications in maximum dosage which the patient tolerated. She received CCB after each meal and

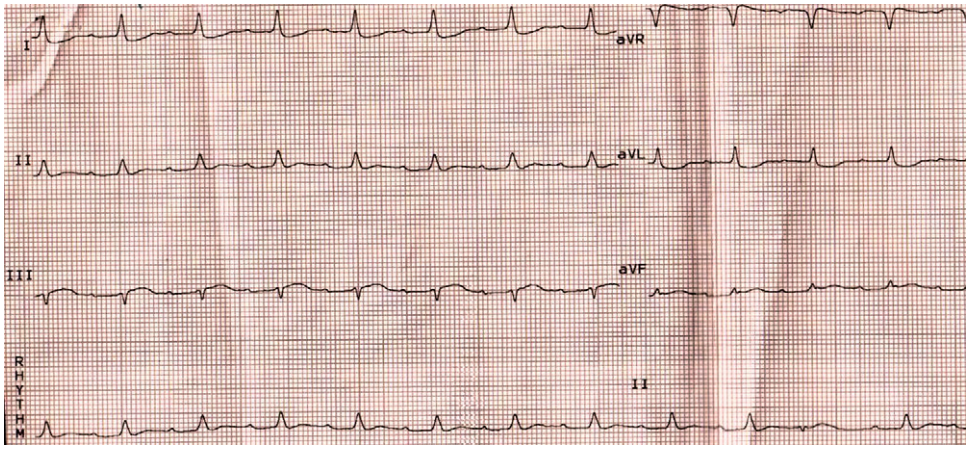
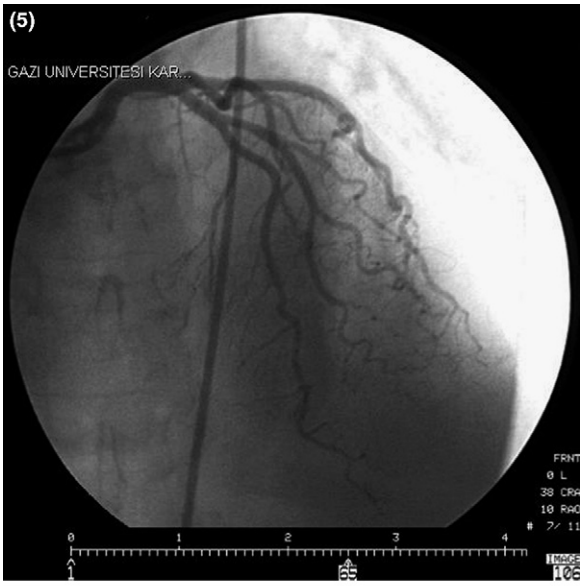
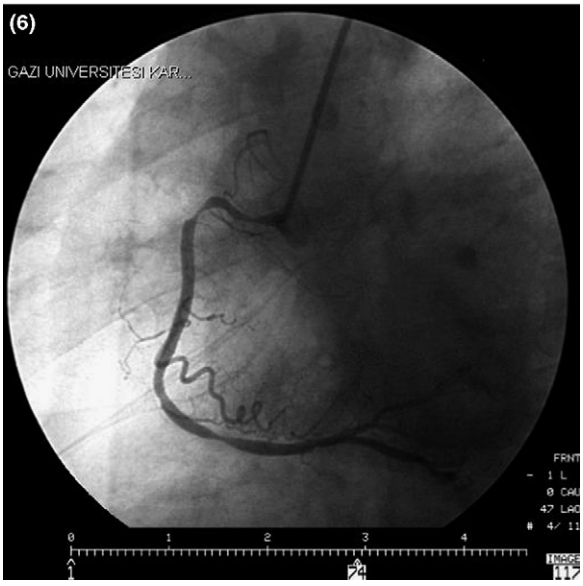


Figure 4 ECG findings after PCI.





Figures 5 and 6 Coronary angiographic evaluation after nitrate administration.

the time of the last dose was at midnight. Her hemodynamic status improved after 6 h of this treatment. She was treated with Tiotropium Br (1× 18 μg), Formoterol fumarat (2× 12 μg), Budesonid (200 μg/day) for asthma. Her complaints of chest pain and dyspnea improved. She was discharged on 13th day with same treatment.

After 2 months she was admitted with aggravating chest pain under treatment with nitrate and CCB in a maximum dosage which the patient tolerate and other preceding medications to another hospital. Her ECG demonstrated anterolateral ST elevation and subsequently VF and cardiac arrest developed in coronary care unit. After defibrillation and resuscitation, her chest pain diminished and ST elevation on ECG resolved. Her physical examination and laboratory findings were normal. Then she was referred to our hospital. The patient underwent single chamber ICD implant and was discharged from hospital 3 days after the procedure.

Discussion

The etiology and pathogenesis of acute myocardial infarction in patients with normal coronary arteries is still controversial. Coronary vasospasm, thrombosis, embolization, inflammation and endothelial dysfunction are the multiple proposed pathogenetic mechanisms [1,2]. The prevalence of this entity is 1—12% [3]. Coronary artery vasospasm and thrombosis are the most commonly encountered clinic problems and appear in localized coronary segments. It has been shown that cocain abuse, ethanol intoxication, angiotensin II receptor gene polymorphism, smoking and endothelial dysfunction had a crucial role in coronary vasospasm [4]. Clinical presentation as acute coronary syndromes, life-threatening arrhythmias, mechanical complications due to ischemia and sudden death has been demonstrated [5]. Our patient had no history of smoking, mental stress which have possible effects on coronary vasospasm.

The incidence of cardiovascular disease is increased in asthmatic patients. β_2-Adrenergic agonists use is associated with increased heart rate, myocardial oxygen demand and reduced potassium levels which lead to arrhythmias, ischemia and congestive heart failure in patients with asthma bronchiale [6]. In some studies the possible relationship through parasympathetic system activation between coronary vasospasm and asthma has been suggested [7]. Beta agonists use may contribute to occur of severe ischemia due to increased heart rate and myocardial oxygen demand. Myocardial ischemia during asthma has been described in literature. Acute myocardial infarction with normal coronary arteries in patients with asthma bronchiale is very rare. Noike et al. reported a patient with asthma bronchiale and acute inferior myocardial infarction [8]. Suissa et al. suggested that increased cathecolamine levels had a crucial role in developing cardiovascular events in patients with asthma [9]. In our patient severe bronchial asthma contributed to occur coronary vasospasm. Her severe dsypnea required β_2-adrenergic medication.

Patients with severe coronary vasospasm in allergic situations may benefit from corticosteroid therapy [10]. Our patient was receiving inhaler cor-

ticosteroid therapy therefore we did not prefer oral treatment.

In our case, our patient's clinical presentation was cardiogenic shock with acute inferior and right ventricular MI. Therefore the decision was made to perform primary PCI. Severe multifocal, multivessel obstructive coronary artery disease was revealed in coronary angiographic examination. We did not administer nitrate due to severe hypotension and cardiogenic shock and we inserted intraaortic balloon pump. Nitrate injection during primary PCI in cardiogenic shock is not indicated even contraindicated however it led to a diagnostic pitfall in our case leading to stent implantation to a vasospastic vessel. It has been suggested that coronary stent placement might be a treatment approach in patients with vasospastic angina unresponsive to medical therapy [11]. To establish a true-coronary diameter with PCI is an important issue which prevents the coronary artery from thrombosis. The clinical course of our patient did not seem to benefit from stent placement. Her hypotension, chest pain and life-threatening arrhythmia continued. Due to ongoing chest pain and hemodynamic instability; an early bypass surgery was planned. After intracoronary nitrate administration during control angiogram all lesions disappeared. Therefore the cause of this situation was not due to coronary atherosclerosis. On control angiogram posterobasal and inferior hypokinesias were seen. On 2nd hospitalization transthoracic echocardiographic examination demonstrated same hypokinetic areas without any additional findings. Only after cessation of dopamine infusion and administration of high doses of CCBs and oral nitrates her hemodynamic status improved.

After 2 months she suffered VF and cardiac arrest during readmission with chest pain under treatment with vasodilator agents. She was resuscitated and ECG demonstrated anterolateral ST elevation, cardiac enzymes, troponins were negative. In that time she had no additional symptoms and laboratory findings. Under maximum dosage of CCB and nitrates, due to recurrent VF cardioverter intracardiac defibrillator was implanted. Hypokalemia which occurs with β-adrenergic stimulation mal lead severe ventricular arrhythmias in patients with asthma. Our patient had normal laboratory findings.

In our patient coronary vasospasm involved all coronary vasculature and caused recurrent ventricular fibrillation due to the extensive ischemia.

Coronary vasospasm is a misleading angiographic finding. There are several case reports in the literature about patients undergoing coronary bypass surgery with vasospastic lesions mimicking true stenosis which later were found to have normal coronary angiograms. Our case may be the first report of coronary vasospasm in all the three coronary vessels leading to inferior and right ventricular MI, cardiogenic shock and recurrent ventricular fibrillation which required intracardiac defibrillator in a patient with long-standing asthma.

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