

Acute Myocardial Infarction Caused by Multiple Coronary Artery Embolisms

BRENDA N. MANGARIELLO¹, MIGUEL PAYASLIAN², MARCELA ALBORNOZ¹, PABLO MIGUELEZ¹, VANINA GOS RE¹, AGUSTINA BIASCO¹, FABIO MUÑOZ¹, SIMÓN SALZBERG^{MTSAC, 3}, PATRICIA GITELMAN^{MTSAC, 4}

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Address for reprints:

Dra. Brenda N. Mangariello
Cerviño 3356

(1425) Ciudad Autónoma
de Buenos Aires

Phone number: 1565441035

e-mail:

bmangariello@hotmail.com

SUMMARY

Coronary artery embolism is a rare cause of acute coronary syndrome that is scarcely documented. Only a few case reports have been published but lack of solid angiographic evidence. We describe a 32-year old man without history of cardiovascular disease with a first episode of acute myocardial infarction with angiographic evidence of multiple thromboembolisms of the coronary arteries who developed cardiogenic shock and died. We review the causes and the clinical presentations of coronary artery embolism.

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Key words > Embolism - Thrombosis - Myocardial infarction

Abbreviations

CA	Coronary angiography	LCX	Left circumflex artery
RCD	Right coronary artery	LAD	Left anterior descending
CK-MBC	reatine kinase MB fraction	VF/VT	Ventricular fibrillation/ventricular tachycardia
CK	Creatine kinase		

CASE REPORT

A 32-year old man was admitted to the emergency room due to intense (10/10) oppressive chest pain irradiated to the back, lasting for an hour.

The patient did not present signs of heart failure. He had no personal or family history of cardiovascular disease. He was a current smoker of 40 cigarettes/day and had a history of habitual alcohol intake. The electrocardiogram showed ST-segment elevation from V1 to V3 and in aVR, and ST-segment depression in LI and aVL (Figure 1 A). Minutes after being admitted, the patient presented cardiac arrest with VF/VT rhythm and advanced cardiopulmonary resuscitation was initiated. Sinus rhythm was restored after six electrical shocks and the subsequent electrocardiogram showed complete right bundle-branch block, Q waves in inferior leads and ST-segment elevation in inferior and mid-anterior leads (Figure 1 B). Immediately after, the patient developed cardiogenic shock.

A diagnosis of acute coronary syndrome with persistent ST-segment elevation and Killip-Kimball class D was made, and the patient was referred to the catheterization laboratory for primary percutaneous coronary intervention. He was medicated with aspirin, clopidogrel, amiodarone and dopamine from the moment he was admitted.

Coronary angiography (CA) was performed with a time window of 140 minutes after the onset of pain. The study revealed the presence of intraluminal filling defects in the proximal left anterior descending (LAD) artery with TIMI II distal flow, non occlusive lesions in the right coronary artery (RCA) with a radiolucent image in the mid-RCA segment and right posterior descending artery with TIMI III flow, and an occlusive lesion in the circumflex (LCX) artery AV groove continuation segment (Figure 2). The images were consistent with angiographic thrombosis, defined as non calcified intraluminal defects or total occlusion with convex margins and radiolucent image.

During the procedure the patient developed extreme sinus bradycardia followed by cardiac arrest with VF/VT rhythm. Resuscitation was initiated and sinus rhythm was restored. An intraaortic balloon pump and a transient pacemaker were rapidly introduced. Angioplasty with stent implant to the LAD was successfully performed under infusion of tirofiban. Injection of contrast agent after the procedure showed a thrombus in the first diagonal branch and absence of thrombosis in the LCX and mid-RCA. The edges of the coronary arteries were smooth with no evidence of atheromatosis (Figure 2).

The electrocardiogram after the procedure did not show any changes. The patient remained in cardiogenic

Department of Cardiology, Hospital Juan A. Fernández. Buenos Aires, Argentina

^{MTSAC} Full Member of the Argentine Society of Cardiology

¹ Department of Cardiology

² Chief of the Cardiac Catheterization Laboratory

³ Chief of the Department of Cardiology

⁴ Chief of the Coronary Care Unit



Fig. 1. Electrocardiogram **A.** Admission in the ER: ST-segment elevation from V1 to V3 and in aVR, and ST-segment depression in I and aVL. **B.** After cardiac arrest: complete right bundle-branch block, Q waves in inferior leads and ST-segment elevation in inferior and mid-anterior leads.

shock and required inotropic drugs, vasopressors and implant of intraaortic balloon pump. He died 12 hours after hospitalization. A post mortem study was not authorized.

During his short stay at the ER, a urine specimen was collected for qualitative determination of drugs of abuse (cocaine, amphetamines, marihuana, methylenedioxyamphetamine, morphine, phencyclidine, barbiturates, benzodiazepines and tricyclic antidepressants). The study was positive for benzodiazepines. Plasma levels of CK and CK-MB were 6337 and 417 U/L, respectively (Table 1).

DISCUSSION

Coronary artery embolism is a rare cause of myocardial infarction. (1, 2) The infrequency of coronary embolism has been attributed to the proximal take-off of the coronary arteries from the aorta, the fact that the majority of coronary filling occurs during diastole and to the discrepancy between the calibers of the aorta and that of the coronary artery. (1)

Cheng classified coronary embolism in direct, paradoxical, and iatrogenic according to the pathophysiological mechanism. (1)

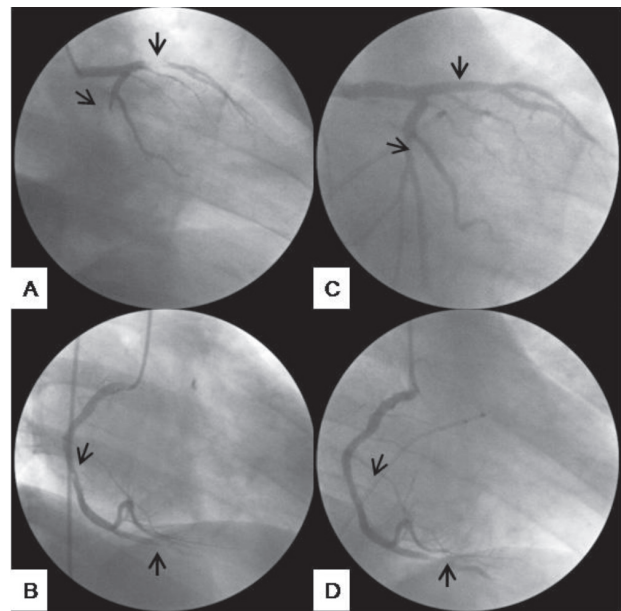


Fig. 2. Coronary angiography. **A.** Image suggestive of non occlusive thrombus (arrow) in the proximal left anterior descending (LAD) artery with TIMI II distal flow. An occlusive thrombus in the circumflex artery AV groove continuation segment is also seen (arrow). **B.** Thrombus in the mid-RCA segment and right posterior descending artery (arrows) with TIMI III distal flow. **C.** Angiography following stent (arrow) implantation plus tirofiban infusion in the left anterior descending coronary artery. Spontaneous reperfusion of the circumflex artery (arrow). **D.** Resolution of the thrombus in the mid-RCA segment with persistence of the thrombus in the right posterior descending artery (arrows).

Direct embolism occurs most commonly in patients with aortic valve endocarditis, atrial fibrillation, mural cardiac thrombus, mitral valve prolapse, prosthetic valves or atrial myxoma. (1, 3) The absence of febrile syndrome or anorexia-cachexia syndrome in our patient excludes the diagnosis of infective endocarditis. The absence of history of cardiovascular diseases rules out mural cardiac thrombus. Physical examination findings are not suggestive of mitral valve prolapse or atrial myxoma. Atrial myxoma is more frequent in women and is associated with constitutional symptoms and abnormal laboratory tests that were not present in this case. (4)

Iatrogenic embolism is the most common cause of coronary embolism that is observed in patients undergoing myocardial revascularization surgery, coronary angiography and/or thrombolysis or anticoagulant therapy. (1, 5) One of the mechanisms involved is the dissemination of multiple small deposits of cholesterol crystals from ulcerated atheromatous plaques, producing occlusion of small vessels (atheroembolization). The clinical presentation ranges from subclinical embolization to multiple organ failure. (5) Air embolism is another type of iatrogenic coronary embolism and it results from the iatrogenic introduction of gas

Table 1. Laboratory tests at admission

Hematocrit	44.2%	Creatinine	0.88 mg/dl
Hemoglobin	14.8 mg/dl	Sodium	143 mmol/L
White blood cell count	14.000 U/dl	Potassium	3.7 mmol/L
Plaquet count	463.000 U/dl	Total bilirubin	1.2 mg/dl
Prothrombin time	100%	AST	48 U/L
aPTT	34 seg	ALT	23 U/L
Glycemia	184 mg/dl	Alkaline phosphatase	384 U/L
VBun	26 mg/dl	CK-MB	248/24.4 U/L

bubbles into the coronary circulation and is detected by fluoroscopy. (6) Our patient had no evidence of atherosclerotic disease as the edges of the coronary arteries were smooth. Obviously, air coronary embolism is excluded as the patient had symptoms before undergoing coronary angiography.

Paradoxical embolism is uncommon and refers to the passage of a thrombus via a patent foramen ovale in patients with deep venous thrombosis or pulmonary embolism. (1, 7) The simultaneous compromise of arterial and venous systems is rare. (7) In our patient, the diagnosis of paradoxical coronary embolism cannot be ruled out despite the absence of embolism in other arterial territories: the neurological status could not be evaluated and we could not perform a post mortem study.

Tung described that hypercoagulability states may produce in-situ thrombosis. (3)

The hypercoagulability states predispose to the development of venous and arterial thrombosis and should be suspected in cases of paradoxical embolism and regional thrombosis. (2, 3)

Neoplasms, nephrotic syndrome, myeloproliferative disorders, hemoglobin disorders and cocaine abuse are associated with hypercoagulability states. (3, 8) Data from physical examination and laboratory tests of our case are not suggestive of any of the conditions mentioned above. Despite our patient had no history of neoplasms, we should not forget that a thromboembolic episode may be the first manifestation of cancer.

Inherited or acquired thrombophilia, factor V Leiden, deficiency of protein C or S, hyperhomocysteinemia and antiphospholipid syndrome are conditions that should also be considered. Antiphospholipid syndrome presents compromise of the coronary arteries in 25% of patients, yet coronary events are not the first manifestation of the disease. (9, 10)

In conclusion, we were not able to make a correct diagnosis due to the impossibility of performing a histopathological study. However, after doing this dialectical exercise, we think that this young, apparently healthy patient without a history of cardiovascular disease, had a first episode of multiple coronary artery embolism probably related to some type of inherited or acquired thrombophilia, with in-situ thrombosis in the coronary circulation, or to deep venous thrombosis or pulmonary embolism and paradoxical embolism via a patent foramen ovale.

Finally, isolated cases of this condition have been reported without angiographic documentation. We

have presented the case of a rare disease supported by angiographic evidence that opens a range of etiological possibilities.

RESUMEN

Tromboembolia coronaria múltiple como causa de infarto agudo de miocardio

La tromboembolia coronaria es una causa infrecuente y poco documentada de síndrome coronario agudo. En la bibliografía sólo se encuentran comunicaciones aisladas con escasa fundamentación angiográfica. En esta presentación se describe el caso de un varón de 32 años sin antecedentes cardiovasculares, que debuta con un cuadro de infarto agudo de miocardio con evidencia angiográfica de múltiples tromboembolias coronarias y evoluciona a shock cardiogénico y muerte. Se realiza, asimismo, una revisión de las causas y de las formas de presentación clínica de la tromboembolia coronaria.

Palabras clave > Embolia - Trombosis - Infarto del miocardio

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