Paradoxical Coronary Embolism: A Rare Cause of Acute Myocardial Infarction

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Since it was first described in 1877, paradoxical embolism has become widely accepted as an etiology of stroke and peripheral vascular thrombotic occlusion. Less common are paradoxical emboli to the coronary artery. On the basis of limited pathologic and clinical series, it appears that paradoxical coronary emboli account for 5%–10% of all paradoxical emboli. Paradoxical coronary emboli have been described in all age groups, from neonates to the elderly, reflecting a wide spectrum of cardiac structural abnormality and clinical circumstance. Strategies for management should focus on treatment of acute coronary occlusion as well as prevention of future emboli.
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Key words: Acute myocardial infarction • Paradoxical coronary embolism • Patent foramen ovale • Atrial septal defect

A 57-year-old woman was admitted to the hospital with a 1-day history of intermittent chest discomfort. She had felt well until the morning of admission, when she noted the onset of severe exertional chest pressure that resolved spontaneously over 30 minutes. Several hours later she developed another episode of chest pressure on minimal exertion that persisted at rest, accompanied by shortness of breath. She became frightened and called for emergency assistance.

Emergency Medical Services personnel found her anxious and uncomfortable with a blood pressure of 150/80 mm Hg, heart rate of 98 beats/min, and respiratory rate of 14 breaths/min. Physical examination in the field was remarkable only for mild diffuse wheezing. She was administered a single sublingual nitroglycerin and had prompt and complete relief of the chest pressure. She was brought to the emergency department (ED) for further evaluation.

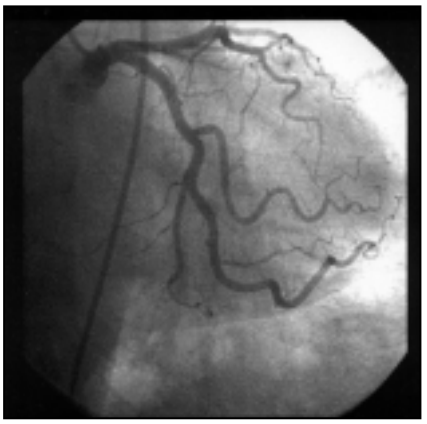

Figure 1. *Single-frame cineangiogram of the left coronary artery in the right anterior oblique with caudal projection.*

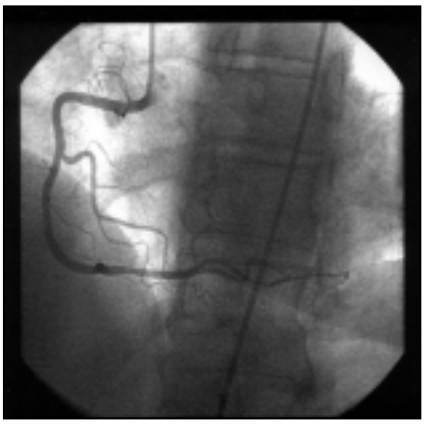

Figure 2. *Single-frame cineangiogram of the right coronary artery in the left anterior oblique projection.*

History

The patient had a history of mild chronic obstructive pulmonary disease that had never required hospitalization, as well as mild hypertension. There was no prior history of angina and no known cardiac disease. She had no history of hyperlidemia or diabetes and no family history of premature coronary artery disease. Her outpatient medications included lisinopril, albuterol, ipratropium bromide (Atrovent), and fluticasone.

Examination, Initial Laboratory Data

In the ED, she was found to have no residual chest discomfort. Her blood pressure was 130/70 mm Hg and her heart rate was 84 beats/min. Her oxygen saturation was 100% on room air. The physical examination was otherwise remarkable only for mild wheezing. Her electrocardiogram showed normal sinus rhythm with an atrial premature beat, and was otherwise normal. Complete blood cell count and electrolytes were normal. Her initial creatine kinase (CK), CK-MB, and troponin I were all within normal limits. Chest film showed no acute pulmonary process. She was admitted to the hospital for further observation.

Hospital Course

The patient was treated with aspirin, unfractionated heparin, metoprolol, and inhaled bronchodilators. At the next assessment of cardiac enzymes, the CK had risen to 236 U/L with a CK-MB of 12.3 U/L (5.2%) and troponin I of 3.3 ng/mL, all above normal. She was started on an intravenous infusion of eptifibatide. She continued to remain symptom-free, and her cardiac enzymes trended downward. Repeat electrocardiograms showed no new abnormalities.

On the second day of hospitalization, left heart cardiac catheterization was performed. The coronary arteries were angiographically normal (Figures 1 and 2). Left ventriculography revealed mild posterobasal hypokinesis, with a calculated ejection fraction of 55% (Figure 3). Right heart catheterization was subsequently performed (Table 1).

On the basis of the step-up in oxygenation at the level of the high right atrium, a small left-to-right shunt was considered possible, although, given the normal pulmonary artery saturation, any shunt would be very small. The arteriotomy was sealed, and the patient was removed from the table. Unfractionated heparin was continued.

Transthoracic echocardiography revealed normal biventricular size and systolic function. A small patent foramen ovale (PFO) or atrial septal defect (ASD) was noted, with right-

Table 1
Right Heart Catheterization

Site	Pressure, mm Hg	Oxygen Saturation, %
Inferior vena cava	—	72.9
Superior vena cava	—	72.1
High right atrium	13/10/9	79.5
Right ventricle	34/10	66.3
Pulmonary artery	34/15/23	68.5
Pulmonary capillary	17/20/13	—

to-left flow present at rest after intravenous injection of agitated saline (Figure 4). Lower extremity Doppler showed a left lower extremity, calf-level, deep vein thrombosis. Computed tomography–pulmonary angiography was performed and revealed a pulmonary embolism in the right main pulmonary artery, extending into the right upper lobe artery. The patient was maintained on heparin, and oral anticoagulation was started. She was discharged on hospital day 6 and continues to recover uneventfully.

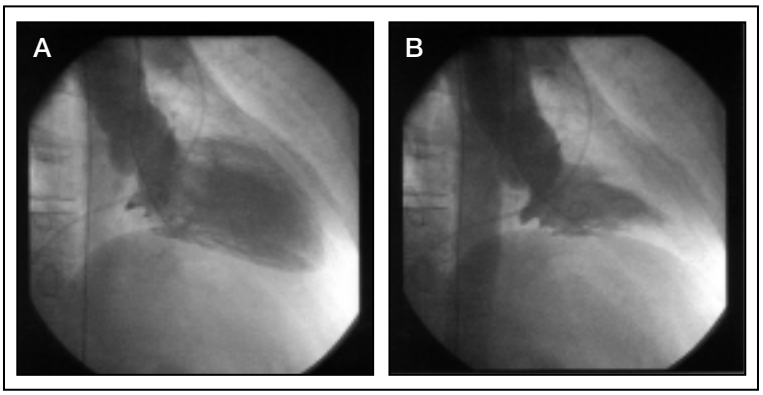

Figure 3. *Left ventriculography in the right anterior oblique projection at end-diastole* **(A)** *and end-systole* **(B)**.

Discussion

Common left-to-left emboli, which occur most often in patients with atrial fibrillation but also in other patient populations, generally manifest in the form of stroke. Less common in these patients are emboli to other sites, including the aorta, mesentery, renal arteries, and extremities, with rare reports of coronary emboli.[1,2] Similarly, patients with prosthetic heart valves most often embolize to the brain, although embolization to the coronary artery may account for approximately 15% of embolic episodes.[3]

Far less common are paradoxical emboli. Paradoxical embolism refers to the passage of embolic material from the venous to arterial circulation across a cardiac defect. Although several definitions exist, most require the following:

- Clinical, radiologic, or postmortem evidence of arterial embolic occlusion in the presence of:
  - Pulmonary embolism or a right-sided source of embolus and
  - A constant or inducible right-to-left shunt

A proven rather than presumptive diagnosis of paradoxical embolism mandates that, in addition to the above criteria, embolic material is identified in situ, straddling the cardiac defect.[4]

Since it was first described in 1877,[5] paradoxical embolism has become widely accepted as an etiology of stroke and peripheral vascular thrombotic occlusion. Less common are paradoxical emboli to the coronary artery. On the basis of limited pathologic and clinical series,[4,6] it appears that paradoxical coronary emboli account for 5%–10% of all paradoxical emboli. Paradoxical coronary emboli have been described in all age groups, from neonates to the elderly,[4,7] reflecting a wide spectrum of cardiac structural abnormality and clinical circumstance. Given the transient perinatal predisposition toward right-to-left shunting, the phenomenon of paradoxical coronary emboli differs considerably between the very young patient and patients in all other age groups. Excluding the neonate, based on review of databases and pertinent bibliographies, 32 cases of proven or presumptive paradoxical coronary emboli have been identified.[4,6,8-12] Important features of these cases are shown in Table 2.

Common left-to-left emboli, most often in patients with atrial fibrillation but also in other patient populations generally manifest in the form of stroke.

Patients most often present with classic symptoms and electrocardiographic evidence of acute myocardial

Table 2
Features of Paradoxical Coronary Emboli (N = 32)

Age	16–77 years
Sex (male/female)	9/23
Embolic material	Thrombus, 23; air, 7; other, 2
Coronary artery site	Left main, 5; left anterior descending, 11; left circumflex, 1; right, 7; diffuse, 3; unknown, 5*
Right-to-left conduit	PFO, 21; ASD, 5; other, 2; unknown, 4

*Electrocardiographic or enzyme evidence of paradoxical coronary emboli.
ASD, atrial septal defect; PFO, patent foramen ovale.

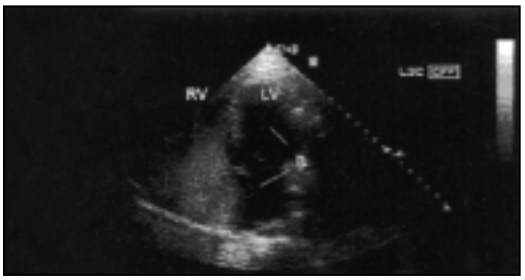

Figure 4. *Still-frame echocardiogram in the apical four-chamber view during intravenous injection of agitated saline revealing the presence of bubbles in the left atrium and left ventricle. B, bubbles; LV, left ventricle; RV, right ventricle.*

infarction, though these may be obscured by important dysfunction of other organs involved in embolic phenomena or by coexisting disease. Patients are often hypoxic, reflecting the frequent presence of concomitant pulmonary emboli, an ongoing right-to-left shunt, or congestive heart failure. Angiography or pathologic examination reveals coronary occlusion, although when performed remote from the acute event, angiography may be notable only for the absence of significant coronary disease. When pathologic specimens are available, embolic material is most often found to be thrombus arising from a fragmented deep vein thrombosis, although in more unusual clinical settings, tumor or other materials have been found.[8,13] Air has been implicated as the source of coronary artery embolism in various settings, including after neurosurgery, abdominal surgery,[14-16] and even simple venous cannulation.[17] Paradoxical coronary emboli have been found in all major divisions of the epicardial coronary circulation, although emboli to the left coronary circulation are more common than emboli to the right coronary artery.

A right-to-left shunt is required to satisfy the definition of paradoxical coronary emboli, which mandates a pathway and a pressure gradient for paradoxical flow. Most often, a PFO or an ASD is the conduit for embolic material, although paradoxical coronary emboli may occur through more unusual congenital cardiac defects.[17,18]

The pressure gradient driving blood from right to left usually results from common conditions that raise right-sided pressures either transiently (Valsalva or cough) or on a chronic basis (chronic obstructive pulmonary disease, pulmonary embolism, positive-pressure ventilation). When searching for the diagnosis, it is important to note that shunt flow across a PFO may be absent at rest. In these cases, provocative maneuvers with either coughing or Valsalva are necessary to elevate right-sided pressures and demonstrate the presence of shunt flow.[19] This flow is most easily demonstrated echocardiographically by color Doppler or intravenous injection of contrast or agitated saline ("bubble study").

Paradoxical coronary emboli have been found in all major divisions of the epicardial coronary circulation, although emboli to the left coronary circulation are more common than emboli to the right coronary artery.

Although no evidence-based treatment guidelines exist for paradoxical coronary emboli, strategies for management should focus on treatment of acute coronary occlusion as well as prevention of future

Main Points

- A proven diagnosis of paradoxical embolism requires clinical, radiologic, or postmortem evidence of arterial embolic occlusion in the presence of pulmonary embolism or a right-sided source of embolus, with a constant or inducible right-to-left shunt, as well as embolic material identified in situ, straddling the cardiac defect.
- When pathologic specimens are available, embolic material is most often found to be thrombus arising from a fragmented deep vein thrombosis, although in more unusual clinical settings, tumor or other materials have been found.
- Air has been implicated as the source of coronary artery embolism in various settings, including after neurosurgery, abdominal surgery, and even simple venous cannulation.
- Anticoagulation is a mainstay of therapy, reflecting the predominance of thrombotic emboli and their frequent association with deep vein thrombosis and pulmonary emboli.
- When anticoagulation is contraindicated or considered inadequate for long-term management of embolic risk, inferior vena cava filter placement may be appropriate; surgical or percutaneous repair of anatomic deformities may also be appropriate, depending on anatomy and the clinical situation.

emboli. With ongoing thrombotic occlusion and ischemia, thrombolytic therapy and percutaneous coronary intervention with a goal of disrupting the embolus have been utilized. Anticoagulation is a mainstay of therapy, reflecting the predominance of thrombotic emboli and their frequent association with deep vein thrombosis and pulmonary emboli. When anticoagulation is contraindicated or considered inadequate for long-term management of embolic risk, inferior vena cava filter placement may be appropriate. Surgical or percutaneous repair of anatomic deformities may also be appropriate, depending on anatomy and the clinical situation.

In conclusion, paradoxical coronary embolism is a rare etiology of acute myocardial infarction and is found in a wide variety of clinical scenarios. Patients usually have a presentation consistent with an acute myocardial infarction. Management addresses an acute, usually thrombotic occlusion and prevention of further episodes. The diagnosis should be considered in all patients with the correct anatomic and clinical substrate, particularly in the setting of normal coronary arteries. ■

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