

## Paradoxical Embolism in the Left Main Coronary Artery: Diagnosis by Transesophageal Echocardiography

HANS K. MEIER-EWERT, MD; SHERIF B. LABIB, MD; EDGAR C. SCHICK, MD; DAVID E. GOSSMAN, MD; MICHAEL S. STIX, MD; AND CHRISTINA A. WILLIAMSON, MD

We describe a patient with a paradoxical coronary embolism diagnosed by transesophageal echocardiography. The patient developed a stroke followed by a myocardial infarction. Coronary angiography showed an obstruction of the left main coronary artery. Transesophageal echocardiography showed the mechanism of the neurologic and cardiac events to be a paradoxical embolism. Emergency surgical retrieval of the thrombus lodged in the left main coronary ostium and of a separate thrombus traversing a patent foramen ovale was performed. To our knowl-

edge, direct visualization of the paradoxical coronary embolism by echocardiography has not been reported previously. We discuss mechanisms responsible for paradoxical coronary embolism and review the literature pertaining to this condition.

*Mayo Clin Proc.* 2003;78:103-106

CT = computed tomography; LAD = left anterior descending; PFO = patent foramen ovale; TEE = transesophageal echocardiography

Although embolism in a coronary artery is a recognized cause of myocardial infarction in patients otherwise not known to have coronary heart disease, paradoxical coronary embolism is rare. In an autopsy series of 1050 patients with myocardial infarctions, Prizel et al<sup>1</sup> found only 55 patients who had had coronary embolisms, none of which were paradoxical embolisms. We describe a patient with paradoxical embolism in the left main coronary artery and the diagnosis made by transesophageal echocardiography (TEE).

### REPORT OF A CASE

A 62-year-old woman who had a medical history of hypertension, trigeminal neuralgia, and a left ankle fracture treated with a cast 2 weeks earlier presented for elective radiofrequency ablation of the left trigeminal nerve. The procedure was complicated by a subarachnoid hemorrhage. Computed tomography (CT) showed a small amount of subarachnoid blood, and cerebral angiographic findings were normal. She was admitted to the hospital for observation. Serial CT scans showed decreasing subarachnoid blood with mild enlargement of ventricles consistent with hydrocephalus. Except for having frontal headaches, the patient's general neurologic status remained unremarkable;

speech was fluent, and motor function was intact throughout the postprocedure period.

On the seventh day in the hospital, the patient developed new right hemiplegia and expressive aphasia. An emergent CT scan showed no interval change in the extent of intracranial hemorrhage. Approximately 2 hours after onset of neurologic symptoms, the patient developed dyspnea and chest pain. An electrocardiogram revealed sinus tachycardia with acute ST elevation in leads I, aVL, V<sub>1</sub>, and V<sub>2</sub>. An urgent cardiology consultation was requested.

Emergency coronary angiography was performed. Injection of the left main coronary artery showed near total occlusion of the entire left main segment by an intraluminal radiolucency consistent with thrombus. The left anterior descending (LAD) coronary artery was totally occluded, and the left circumflex coronary artery was partially occluded (Figure 1). Angiographic findings of the right coronary artery were normal.

The patient underwent emergency bypass surgery within 3 hours of the onset of chest pain. A TEE was performed to investigate possible sources of an embolus. An extensive, mobile echodensity was found in the right atrium traversing the patent foramen ovale (PFO) into the left atrium (Figure 2, A). A separate mobile echodensity was evident in the proximal aorta, anchored in the ostium of the left main coronary artery (Figure 2, B). Left ventricular function was severely depressed with hypokinesis of anterior and lateral walls. The right ventricle was normal in size and function. There was no evidence of thrombus in the main pulmonary artery or in the proximal portion of its right or left branches. A single 10-mL injection of agitated saline, which was administered while the patient was intu-

From the Department of Cardiovascular Medicine (H.K.M.-E., S.B.L., E.C.S., D.E.G.), Department of Anesthesiology (M.S.S.), and Department of Cardiothoracic Surgery (C.A.W.), Lahey Clinic Medical Center, Burlington, Mass.

Address reprint requests and correspondence to Sherif B. Labib, MD, Department of Cardiovascular Medicine, Lahey Clinic Medical Center, 41 Mall Rd, Burlington, MA 01805 (e-mail: sherif.b.labib@lahey.org).

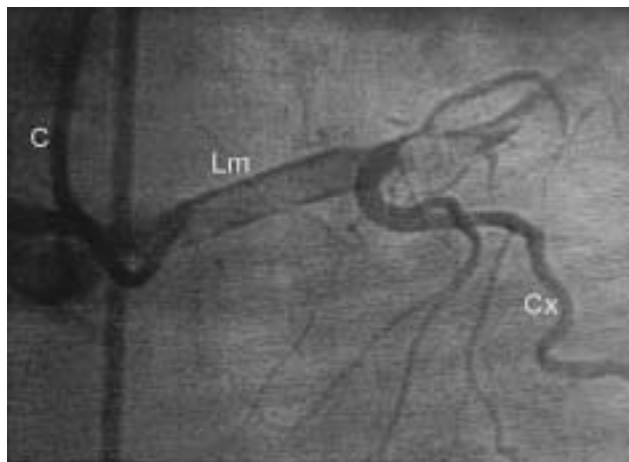


Figure 1. Angiogram of the left coronary artery showing obstruction of the left main segment by a radiolucency with occlusion of the left anterior descending coronary artery and obstruction of circumflex branches. C = catheter; Cx = circumflex coronary artery; Lm = left main coronary artery.

bated, provided excellent opacification of the right heart chambers; no bubbles appeared in the left atrium.

The patient was placed on cardiopulmonary bypass. An aortotomy was performed, and a thrombus was found protruding from the ostium of the left main coronary artery. The thrombus was removed, which resulted in free backflow of blood from the ostium while retrograde cardioplegia was administered via the coronary sinus (Figure 3, A). The LAD coronary artery was opened, and a 2.5-mm probe could be passed proximally and distally with no further retrieval of the clot. The right atrium was then opened, and a long thrombus trapped in a PFO was re-

moved (Figure 3, B). The separation between the 2 layers of the interatrial septum measured nearly 1 cm. The PFO was sutured, and the LAD coronary artery was bypassed with a saphenous vein graft. A second TEE done before decannulation showed another density extending from the inferior vena cava into the right atrium, and this thrombus was removed on reexploration before the chest was closed. Postoperatively, venous studies showed a residual thrombus in the left popliteal vein, and a filter was placed in the inferior vena cava.

The patient's subsequent course was complicated by prolonged cardiogenic shock, pulmonary edema, sepsis, heparin-induced thrombocytopenia, and adult respiratory distress syndrome. Computed tomography of the brain 7 days after surgery showed a large stroke in the distribution of the left middle cerebral artery with midline shift. On the 22nd postoperative day, hemorrhage into the ischemic territory with uncal herniation was observed, and the patient died. Autopsy showed no evidence of pulmonary embolism.

## DISCUSSION

More than 150 cases of paradoxical embolisms have appeared in the literature since this condition was first described by Cohnheim in 1877.<sup>2</sup> Because of the known high prevalence of PFO in individuals without embolic events, the diagnosis of paradoxical embolism is often difficult to prove in an individual patient. Indeed, Johnson<sup>3</sup> suggested that a paradoxical embolism can be "presumed" when the following criteria are met: (1) evidence of arterial embolization in the absence of a source in the left heart, (2) a source of embolism identified in the venous system, and (3) demonstration of an abnormal communication between the venous and arterial circulations. Paradoxical embolism can

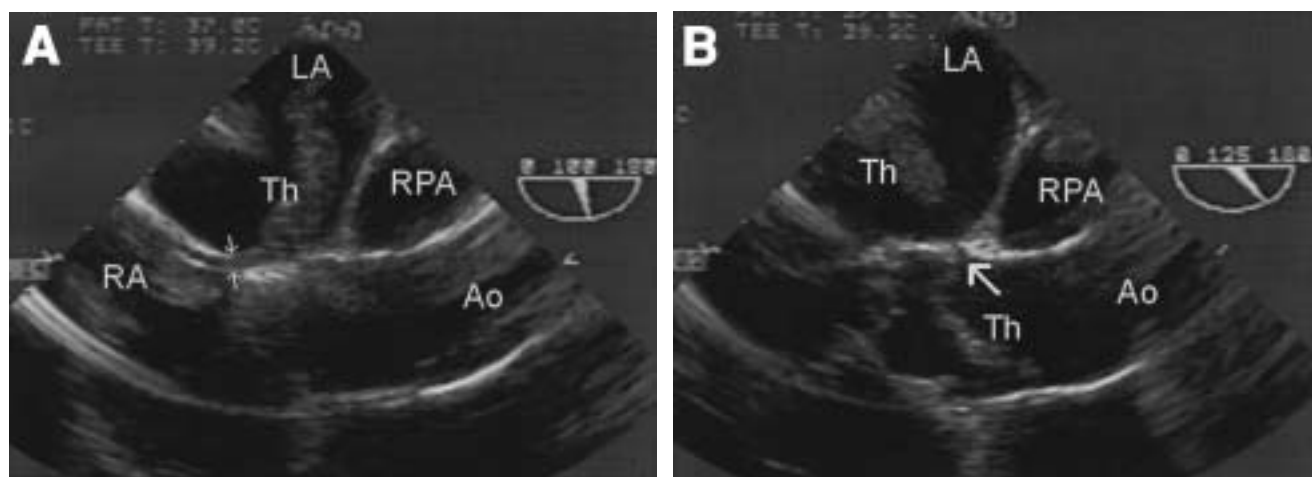


Figure 2. Transesophageal echocardiogram. A, Thrombus traversing patent foramen ovale (small arrows) from right atrium into left atrium. B, Long-axis view of the ascending aorta, showing mobile echodensity in the ascending aorta with attachment to the left main coronary ostium (large arrow). Ao = ascending aorta; LA = left atrium; RA = right atrium; RPA = right pulmonary artery; Th = thrombus.

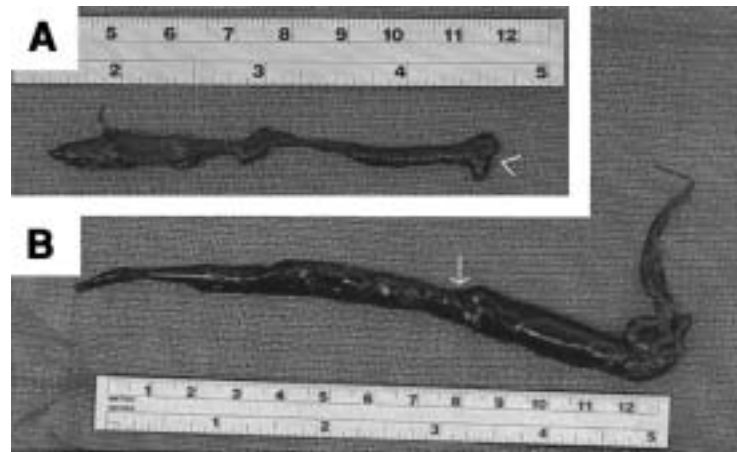


Figure 3. A, Thrombus removed from the left main coronary ostium showing molding of the embolus by the coronary artery anatomy, allowing clear distinction of left main coronary artery segment and bifurcation into left anterior descending and circumflex coronary branches (arrowhead). B, Thrombus removed from the right atrium. The region where the thrombus had lodged in the patent foramen ovale can be distinguished (arrow), as can the leading edge from which the emboli that traveled to the brain and coronary artery were presumably detached.

be considered “proven” only when the embolus is found lodged in the abnormal communication between the venous and arterial circulations.

The advent of echocardiography, and in particular TEE, has made antemortem diagnosis of paradoxical embolism more common, but the direct imaging of a thrombus entrapped in the PFO, the so-called impending paradoxical embolism, is still extremely unusual. The first such case was reported in 1985,<sup>4</sup> and in a recent review of the English language literature, 29 cases were summarized.<sup>5</sup> A broader review of the world literature indicates the occurrence of an actual arterial embolic event in approximately 50% of such cases.<sup>6</sup>

Pulmonary embolism is often present and is frequently implicated in the increased right heart pressures that set the stage for right-to-left shunting via a PFO. However, in a summary of all 44 case reports of entrapped emboli in the world literature, 53% of patients had pulmonary embolism alone, 40% had both pulmonary and paradoxical embolism, and 7% had paradoxical embolism without pulmonary embolism.<sup>6</sup> Pulmonary embolism was not present in our patient. Other mechanisms implicated in facilitating paradoxical embolization are chronic pulmonary hypertension, Valsalva maneuver, cough with transient reversal of the interatrial pressure gradient, and positive pressure ventilation.<sup>7</sup> Transient right-to-left shunting also has been shown to occur spontaneously and may be explained by the preferential direction of venous flow from the inferior vena

cava into the fossa ovalis.<sup>8</sup> Thus, although evidence of pulmonary embolism always should suggest a PFO when systemic embolization occurs, its absence does not preclude the diagnosis.

Paradoxical embolism in a coronary artery is a recognized clinical entity, but it is rare and usually definitively established only at autopsy. In a comprehensive review by Jungbluth<sup>9</sup> in 1988, only 27 cases were reported in the world literature, including 8 cases of paradoxical air embolism. In only 4 cases was an antemortem diagnosis of paradoxical coronary thromboembolism made using electrocardiography and/or angiography in patients with intracardiac communication.<sup>10-12</sup> Subsequent to Jungbluth’s publication, we found only 4 other cases in which a diagnosis of paradoxical coronary embolism was made during the patient’s life.<sup>13-16</sup> According to the criteria proposed by Johnson,<sup>3</sup> all 8 cases would be considered presumed rather than proven because of the lack of demonstrable thrombus at the intracardiac communication site.

Treatment options for impending paradoxical embolism include anticoagulation, thrombolysis (alone or in combination with vena caval interruption), and surgical thrombectomy. Surgery, which allows for simultaneous closure of the PFO, is associated with improved outcomes.<sup>5,6</sup> More frequent diagnoses of impending paradoxical embolisms by TEE, when this diagnosis is suspected, may provide greater opportunity for therapeutic intervention.

Many aspects of our case have not been reported previously. Among patients with demonstrable thrombus entrapped in the PFO, our case represents the first proven antemortem diagnosis of coronary embolism leading to acute myocardial infarction. To our knowledge, TEE visualization of an embolus lodged in a coronary ostium is unprecedented. Finally, appreciation of the embolic nature of the myocardial infarction led to the surgical retrieval of the paradoxical embolism from a coronary artery.

In summary, we report the diagnosis of a paradoxical embolism in the left main coronary artery by TEE. Although this diagnosis is uncommon, we believe that it should be actively sought when myocardial infarction occurs concurrently with systemic arterial embolization in the context of a predisposition to venous thrombosis. Concomitant pulmonary embolization does not need to be present. A TEE is vitally important for accurate diagnosis and appropriate management, once this condition is suspected.

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