ABSTRACT: A 34-year-old male patient was referred for primary percutaneous coronary intervention for ST-segment elevation myocardial infarction with cardiogenic shock and was found to have embolic left coronary artery occlusion and subsegmental pulmonary artery emboli as a consequence of venous thrombosis to trauma to the thigh in the presence of a patent foramen ovale.

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Key words: STEMI, PFO closure

Case Description
A 34-year-old male patient was referred to our hospital for primary percutaneous coronary intervention (PCI) for ST-segment elevation myocardial infarction (MI) with cardiogenic shock. He had been healthy with an unremarkable past medical history. Family history of premature death and cardiovascular disease was negative. The patient had suffered an apple-size bruise of the left thigh during a soccer match the day before. After getting up in the morning, he experienced severe chest tightness associated with worsening shortness of breath at rest and rapidly progressing diaphoresis. Cardiogenic shock developed rapidly. He received endotracheal intubation and...
Death by PFO in a Soccer Player

high-dose catecholnergic support by a medical rescue team at home. He was flown by helicopter to our catheterization laboratory within 90 minutes of symptom onset. The electrocardiogram revealed extensive ST-segment elevation in leads V1-V6, I, and aVL (Figure 1). Left ventriculography showed markedly impaired systolic function with anterosepto-apical akinesia and severe ischemic mitral regurgitation. Coronary angiography performed under manual cardiac massage demonstrated complete thrombotic occlusion of the left anterior descending (LAD) coronary artery, the left circumflex (LCX) coronary artery, and a large intermediate branch (Figure 2). The right coronary artery was normal. Thrombus aspiration from the left coronary artery was performed with a manual suction device followed by several balloon dilatations for further thrombus fragmentation and administration of a weight-adjusted intravenous abciximab bolus, heparin, and acetylsalicylic acid. Patency of all 3 branches was achieved (Figure 2), but flow could only be re-established after percutaneous insertion of a left ventricular assist device (TandemHeart; CardiacAssist, Inc) via transseptal puncture. Transesophageal echocardiography (TEE), performed to exclude papillary muscle rupture as a cause for severe mitral regurgitation, depicted

Figure 3. Transesophageal echocardiogram revealing the patent foramen ovale (arrow) and the TandemHeart cannula (arrow head) placed through a transseptal puncture.

Figure 4. Autopsy findings. (A) Left femoral vein with friable thrombotic material (arrow) and wall injury. (B) Large myocardial infarction involving the anterolateral and septal segments of the left ventricle. (C) Subsegmental pulmonary artery emboli. (D) Patent foramen ovale (PFO).
a patent foramen ovale (PFO) with spontaneous bidirectional shunt (Figure 3). No work-up for a hypercoagulable state was performed. The following night, the treatment was discontinued due to brain death. Autopsy demonstrated wall injury of the left femoral vein at the site of trauma with a fresh thrombus, bilateral subsegmental pulmonary artery emboli, and a large PFO (Figure 4).

Discussion

Paradoxical embolism, typically through a PFO, is entertained in the differential diagnosis of acute myocardial infarction.\(^1\)\(^\text{18}\) It is generally only considered in the realm of otherwise normal coronaries although the presence of coronary artery disease is entirely independent of this mechanism. The detection of thrombus in the venous system or right atrium should not be considered a prerequisite for this diagnosis, as non-detectable small clots are common culprits. The direct detection of thrombus within a PFO being rare,\(^9\)\(^\text{11}\) the diagnosis of paradoxical embolism is usually assumptive. A strong association between the presence of a PFO and the risk for paradoxical embolism has been confirmed in several studies.\(^\text{12-16}\) More than a decade ago, a seminal report identified the PFO as an independent predictor of mortality and stroke in 139 patients with clinically significant pulmonary embolism.\(^9\) A field study confirmed this later without, however, pinpointing the PFO as culprit.\(^1\)\(^\text{18}\) It proved a striking incidence of stroke and confirmed this later without, however, pinpointing the PFO as culprit.\(^1\)\(^\text{18}\)

Non-surgical closure of PFO has been demonstrated feasible\(^19\)\(^,\)\(^\text{20}\) and safe.\(^21\)

The case should cause reconsideration of the current Food and Drug Administration restriction regarding indication for PFO closure to patients with a recurrent event despite medical treatment.\(^22\)

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References