A 69-year-old man with no history of cardiac disease collapsed at home shortly after chest pain appearance. The ECG performed by paramedics during transportation and sent by the LifeNet system for evaluation to the Cardiology Center shows atrial fibrillation (AF) with a regular slow rhythm of 50 beats/min, suggesting complete heart block, ST-segment elevation in inferior and precordial V3 through V6 leads, ST-segment depression in aVL and in precordial V1 to V2 leads (Figure 1A). The clinical presentation and ECG allow for the diagnosis of ST-segment–elevation myocardial infarction (STEMI) and the patient receives aspirin 300 mg, clopidogrel 600 mg, unfractionated heparin 5000 IU, and morphine 5 mg IV on the way to the hospital. Within 3 hours after the onset of chest pain, the patient is presented to the emergency department of the nearest hospital with a 24/7 cardiac catheterization facility.

**Dr Wąsek:** The clinical presentation and ECG are consistent with acute myocardial infarction (MI) with persistent ST-segment elevation. Networking with the mobile emergency unit, prehospital medical treatment, and invasive strategy implementation within the recommended timelines are in agreement with the current guidelines. Although prasugrel or ticagrelor should preferably be used, in this particular case, clopidogrel seems to be a better option in terms of safety, considering the risk of bradycardia recurrence and possible risk of bleeding following faint and head injury.

**Patient presentation (continued):** On admission, the patient reports on-going chest pain, mild dyspnea, and headache. He denies recent infectious symptoms and bleeding, with the exception of a skin head cut following faint. The relevant past medical history includes recent hip surgery, performed a month ago. The preventive anticoagulation regimen following surgery has not been effectively implemented, most probably because of the patient’s alcohol addiction. On physical examination, he is alert and cooperative, his heart rhythm is irregular AF 60 to 70 beats/min, and the diastolic blood pressure is slightly elevated at 130/100 mmHg. On auscultation, first and second heart sounds are normal. There are no murmurs, gallops, or rubs. He is afebrile; the respiratory rate is 10 breaths per minute. The lungs are clear. The jugular venous pressure is not elevated. The abdomen is soft, and no obvious organomegaly is detected. Peripheral pulses (including radial) are palpable and symmetrical. Mild peripheral edema is detected in the lower left extremity. There are no neurological deficits. The skull skin is wounded superficially with no active bleeding. The ECG shows slow AF, ST-segment elevation in inferior leads, ST-segment depression in I, aVL and in precordial V1 through V3 leads; motion artifacts are also seen (Figure 1B). The patient agrees with the proposed invasive treatment. Surgical and neurological consultations are postponed until the end of the reperfusion therapy.

**Dr Wąsek:** The ECG tracing is the key element in diagnosis and proper treatment initiation at this stage. It leads to immediate coronary catheterization and invasive restoration of flow in the case of the occlusion. Any delay in the initiation of treatment at this stage should be avoided. A careful physical examination is needed, but it should be restricted and focused on the presence of possible mechanical complications of MI and the probable presence of different life-threatening diseases mimicking STEMI. From this perspective, the decision to postpone the head skin cut suture is rational; however, careful neurological examination in this case is mandatory because of the history.
of faint and head injury, which might end up with intracranial hematoma and contraindication to any form of anticoagulation. In case of any neurological suspicion, neurologist consultation and computed tomography scan, if necessary, should precede the intervention. The repetitive ECG tracing in STEMI patients before intervention should be avoided and restricted to cases with dynamically changed clinical presentation only. In this case, repetitive ECG tracing could be avoided. The peripheral edema of the lower left extremity has to be further evaluated in the next days in the context of a high probability for the presence of venous thrombosis.

Patient presentation (continued): The invasive strategy is approved, and the patient is immediately transported directly to the catheterization laboratory. The cardiac angiogram, performed via the transradial approach, reveals an extremely large thrombus in the third and fourth segments of the right coronary artery with no clear angiographic signs of plaque rupture (Figure 2A, Movie IA in the online-only Data Supplement). Proximal and medial segments of the right coronary artery and the left coronary artery are angiographically apparently normal (Figure 2B and 2C, Movie IB in the online-only Data Supplement). The flow around the thrombus to the distal part of the vessel is present, but, yet, it is severely impaired. The angiographic picture suggests the embolic rather than atherosclerotic character of the disease.

Dr Wąsek: The crucial question is whether we really can rely on angiography in the proper evaluation of the embolic background of the vessel occlusion. The discrete intraluminal filling defect with a lucent area within the lumen vessel strongly suggests the presence of thrombus. The presence of thrombus in otherwise normal arteries is believed to be consistent with an embolic origin; however, the proper diagnosis requires the exclusion of a complex plaque rupture. The pathohistological correlations of angiographic lesion morphologies have demonstrated that lesions with overhanging edges and irregular or scalloped borders and those that remained contrast-stained for several cardiac cycles corresponded to complex plaques. The
small craters consisting of a discrete luminal widening with luminal irregularity suggest ulceration. Radiolucent extension of the vessel wall into the arterial lumen corresponds with the intimal flap. Despite the diagnostic accuracy of angiography, this imaging modality is not free from limitations. The preserved flow to the distal part of coronary artery (as in this case) allows for a reliable evaluation of the vessel borders, but yet, the abrupt closure requires reevaluation after wiring and makes the final confirmation more complicated and doubtful. The sensitivity and specificity of angiographic features of coronary plaque rupture have been the subject of comparisons with intravascular ultrasound (IVUS) and optical coherent tomography. The complex lesion morphology on angiography has been shown to strongly correlate with plaque ruptures on IVUS. In the study by Maehara et al⁴ there were no false-positive angiographic interpretations of complex plaques, still the sensitivity of angiography was clearly lower, leaving ≈10% of plaque ruptures depicted on IVUS undetectable by angiography. The diagnostic capability and accuracy of optical coherent tomography in complex plaques imaging is even more advanced than IVUS. The distinct advantage of optical coherent tomography over IVUS is not only limited to almost a twice higher sensitivity in evaluating plaque disruption, but also in a capability to identify plaque erosions, imaging unavailable for IVUS.⁵ The imaging limitations of angiography should be considered in making the definite diagnosis of the embolization into the coronary arteries; however, in this case, the coronary angiogram shows apparently normal arteries and no clear angiographic signs of plaque rupture with concomitant enormously large thrombotic burden, unusual picture, especially for a short-duration MI, suggesting the embolic or inflammatory character of the disease.

Patient presentation (continued): The right coronary artery is wired followed by the manual thrombus aspiration. The double bolus of eptifibatide is administered intravenously and continued by weight-adjusted infusion. The thrombectomy and repetitive multilevel balloon dilatation are ineffective, and the flow deteriorates and finally stops (Figure 3A and 3B, Movie II in the online-only Data Supplement). At this stage, the decision of triple-antiplatelet and anticoagulation therapy is made with an intention to continue invasive treatment within a few hours following medical thrombus passivation. Along with antithrombotic treatment, atorvastatin and captopril are administered and the patient is monitored in the Intensive Cardiac Care Unit. The clinical status remains unchanged. The patient experiences recurrent chest pain episodes that are relieved by morphine. The ECG recorded 2 hours after the intervention reveals 50% ST-segment resolution in inferior leads (Figure 4A).
The angiographic picture suggests the need of further differential diagnosis evaluation; however, it does not change the routine immediate invasive approach, aiming for the restoration of flow and myocardial perfusion. The angiographic evidence of massive thrombus, no reflow following the failure of manual thrombus aspiration, justifies the intensification of platelet inhibition; still, in terms of underlying bleeding risk, it “navigates therapy between Scylla and Charybdis.” Unrestored flow, despite the efforts made, leaves 3 possible solutions: percutaneous coronary intervention (PCI) termination, intracoronary
Patient presentation (continued): The next PCI attempt is performed within 8 hours. No flow in the right coronary artery is detected before the intervention (Figure 5A, Movie IIIA in the online-only Data Supplement). Multilevel balloon angioplasty and finally stenting at the level of crux cordis bifurcation restore the flow in the posterior descending artery (Figure 5B, Movie IIIB in the online-only Data Supplement). The posterior lateral branch remains occluded owing to the thrombus distal dislodgement. The patient feels a substantial relief from his chest pain following the revascularization; shortness of breath appears to be the remaining concern. The sinus rhythm is spontaneously restored and blood pressure is normal 130/90 mm Hg. The ECG recording following PCI is comparable to the one performed after the first intervention and demonstrates Q waves and >70% ST-segment resolution in inferior leads (with remaining 1-mm ST elevation) and almost complete ST-segment normalization in precordial leads (Figure 4B). The chest x-ray shows mild to moderate congestion and leads to the initiation of diuretic therapy. Unfractionated heparin and epifibatide infusions are intended to be maintained for the next 24 hours. Statin therapy and angiotensin-converting enzyme inhibition remain unchanged.

Within the few hours following PCI, the patient presents anxiety and abnormal behavioral reactions with impaired logical communication. No changes in neurological examination are found, suggesting a mental, neurotic background of his behavior. The patient shows lack of insights; his psychomotor disturbances are deemed with severe alcohol addiction, leading to the initiation of therapy with diazepam and haloperidol. The therapy is effective, and 2 days later haloperidol is stopped and the patient is left on a low-dose diazepam. He still reports shortness of breath. In the meantime, his temperature increases to 38°C, and white blood cell count increases from 12 000 to 16 000 cells/mm³. Although the chest radiograph demonstrated no infiltrates, amoxicillin and clavulanic acid are given intravenously. The antithrombotic therapy with enoxaparin (1 mg/kg twice daily) is continued following unfractionated heparin termination.

Dr Wąsek: The angiographic success following the second intervention does not go along with the clinical progress. The lack of clinical progress requires further laboratory and echocardiographic evaluation in terms of making a differential diagnosis. The shortness of breath appears to be the main remaining concern, which cannot be explained by chest x-ray, showing no infiltrates and mild congestion only. The differential diagnosis should mainly focus on the pulmonary embolism (PE) as the most probable cause of the deterioration in his clinical course. This brings us back to the necessity of careful consideration of the possibly embolic character of MI and the necessity to look for the etiology of PE and MI coexistence in a high-risk patient with deep venous thrombosis (DVT).

Patient presentation (continued): The blood gases show hypoxia, hypocapnia and respiratory alkalosis, the D-dimer levels are markedly increased, and the troponin levels are decreasing. Transthoracic echocardiogram shows normal size of the left ventricle (51 mm) with 3 acinetic segments: the basal and medium of the inferior wall and the basal of the posterior wall with well-preserved systolic function, ejection fraction 48%, by volume-based quantitation. The flattening of the interventricular septum at end-diastole and recovering of shape at end-systole are detected, with a normal left ventricle diastolic and systolic eccentricity index (Movie IVA in the online-only Data Supplement). Border size left atrium (40 mm) and moderate mitral regurgitation of 32 mL are measured. The septal and posterior wall thickness are within the limits (10 mm). The ascending part of the aorta is mildly dilated (46 mm). The right ventricle (RV) is moderately enlarged (43 mm) with severe hypokinesia of the mid-free wall, associated with normally contracting RV apex: McConnell sign (Movie IVB in the online-only Data Supplement), and tricuspid annular plane systolic excursion of 17 mm. Tricuspid insufficiency is detected with a jet velocity of 2.7 m/s and RV systolic pressure of 50 mm Hg. The vena cava inferior is not dilated (14 mm) and collapses with inspiration. The trunk of the pulmonary artery is dilated proximally to 26 mm.
mm and distally to 31 mm. The short acceleration time (55 ms) with no mid-systolic deceleration of flow velocity in the RV outflow tract is measured. There is no pericardial effusion. The echocardiographic evaluation is highly suggestive of nonmassive PE. The ultrasound examination of the lower extremities demonstrates an occluded left fibular vein and the sonographic features of the endured right deep popliteal venous thrombosis. Computed tomography shows multiple clots in both main pulmonary arteries with a floating part in a main trunk (Figure 6A–6C). There is a massive thrombosis of the left inferior lobe artery and segmental arteries, occluding their lumen by 90% to 100%. The massive thrombosis of the intermediate artery is also present. There are infiltrates in the inferior lobe of the left lung and radiological features suggesting 1-segment infarction.

*Dr Wąsek*: The echocardiographic assessment of individuals with suspected non–high-risk PE is not supported by the current European Society of Cardiology guidelines; however, in patients without previous cardiorespiratory diseases, indirect signs of RV pressure overload might reasonably be interpreted as specific for PE. The signs truly dependent on RV pressure overload, such as increased tricuspid jet velocity and short acceleration time (60/60 sign) associated with increased RV/left ventricle end-diastolic dimension ratio and the presence of the McConnell sign, are together highly sensitive and specific for PE. In this case, the coronary angiogram also helps in the differential diagnosis between RV MI and PE. Undisturbed through RV branches, flow, documented on coronary angiography, allows for the exclusion of the RV infarction. The everyday clinical practice, however, always requires performing a computed tomography scan in PE diagnosis making and, in our case, finally confirms PE. Clinically nonmassive PE does not require thrombolytic or catheter-based invasive treatment. Prolonged anticoagulation is a rational therapeutic option.

The coronary angiogram suggesting the embolic origin of MI in a patient with AF might suggest an atrial origin of the thrombotic material; still, the simultaneous occurrence of MI and PE in a patient with confirmed DVT leads rather to the clinical suspicion of the paradoxical embolization and requires transeophageal echocardiographic examination for the confirmation of the presence of a patent foramen ovale.

*Patient presentation (continued):* Anticoagulation therapy with enoxaparin is clinically effective. Transeophageal echocardiographic confirmed the presence of a large tunnel-like patent foramen ovale (PFO) with spontaneous left-to-right shunt and a large volume of contrast flow from the right to the left atrium on Valsalva maneuver (Movie V in the online-only Data Supplement), but the question whether PFO should or should not be occluded still remains open.

*Dr Wąsek*: The main issue requiring comment is related to the sequence of coexisting life-threatening illnesses. The protective anticoagulation therapy refusal, following hip surgery, related to the patient’s severe alcohol addiction resulted

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**Figure 6.** Angio-CT image. **A**, Massive thrombosis of the pulmonary artery main trunk; the arrow is pointing at the thrombus (horizontal view). **B**, Massive thrombosis of the pulmonary artery main trunk; the arrow is pointing at the thrombus (frontal view). **C**, Three-dimensional CT image reconstruction: dilatation of the pulmonary trunk; the arrow is pointing at the dilated medial part of the trunk. CT indicates computed tomography.
in DVT and subsequently PE. The relation of DVT to STEMI, however, is also highly probable. The suspicion of paradoxical embolization as a cause of STEMI is mainly based on the coronary angiographic picture and related to the diagnosis of DVT and PFO. On the other hand, prolonged immobilization of a patient with STEMI frequently leads to DVT and may cause PE as well. The question is, if the differential diagnosis between 2 clinical scenarios, DVT-related paradoxical embolism resulting with STEMI and simultaneous PE, or a regular STEMI case complicated by DVT and PE, is possible. The timing of PE seems to be the key question to answer. The value of implicit clinical judgment in the diagnosis of PE has been shown in several large series; however, at any stage of this case, implicit clinical judgment could not be of help in PE-onset prediction.8,9 The Wells score and the revised Geneva score both predicted a low clinical probability of PE, and, in our case, both are misleading.10,11 Unfortunately, the Geneve score both predicted a low clinical probability of PE, however, is also highly probable. The suspicion of paradoxical embolization as a cause of STEMI is mainly based on the coronary angiographic picture and related to the diagnosis of DVT and PFO. On the other hand, prolonged immobilization of a patient with STEMI frequently leads to DVT and may cause PE as well. The question is, if the differential diagnosis between 2 clinical scenarios, DVT-related paradoxical embolism resulting with STEMI and simultaneous PE, or a regular STEMI case complicated by DVT and PE, is possible. The timing of PE seems to be the key question to answer. The value of implicit clinical judgment in the diagnosis of PE has been shown in several large series; however, at any stage of this case, implicit clinical judgment could not be of help in PE-onset prediction.8,9 The Wells score and the revised Geneva score both predicted a low clinical probability of PE, and, in our case, both are misleading.10,11 Unfortunately, the severe alcohol addiction resulted in neurotic behavior and hindered the ability to follow up on patient concerns. The temporal sedation impaired patient communication skills and made the clinical assessment even more difficult. Based on ultrasound examination of inferior extremities, however, strongly suggested endured deep popliteal venous thrombosis, we may assume the longer than 4-day history of DVT and reject the hypothesis of prolonged immobilization-dependent DVT occurrence. Besides, it is unlikely that DVT and PE occurred when the patient was on anticoagulants and on triple-antiplatelet agents. It is much more probable that long-lasting DVT attributable to the lack of anticoagulation therapy following hip surgery resulted in nonmassive, meagerly symptomatic PE and paradoxical embolism, leading to STEMI. Cardiac troponin dynamic changes within the first week of hospitalization also support this concept. Within the first day, the troponin I level increased from baseline 0.4 to 130 ng/mL and, during the following days, gradually decreased from 110, 54, 10 to 5 ng/mL. Although the RV myocardium is certainly not its only source, PE would most probably disturb the gradual decrease of elevated plasma troponin attributable to a transient increase of wall stress. And finally, transient right-to-left shunt through a PFO can, indeed, occurs even in the presence of normal right-side hemodynamics, but this phenomenon becomes especially prominent in the setting of elevated right-side pressures.12,13 The answer to the key question, mentioned before, is not just academic, because it paves the way toward the implantation of a PFO occluder.

Patient presentation (continued): The patient was discharged home on day 13 and returned to his baseline functional status. He was rehospitalized within 2 weeks for PFO occlusion. The implantation procedure was successful and the patient returned home on the following day (Movie VI in the online-only Data Supplement). He continued to recover uneventfully.

Discussion

Indications for PFO Closure

The clinical importance of a PFO still continues to be the subject of debate. Since its first description by Cohnheim in 1877, the entity of paradoxical embolism through a PFO has remained a diagnostic challenge. The diagnosis of paradoxical embolism is almost always presumptive and relies on the occurrence of an arterial thromboembolic event in the absence of AF, absence of disease in the left side of the heart, absence of severe atherosclerosis of the thoracic aorta; the detection of right-to-left shunt (usually through a PFO or an atrial septal defect); and the presence of venous thrombosis or pulmonary embolism. Definite confirmation of paradoxical embolism essentially requires the detection of a right atrial thrombus crossing PFO. However, direct observation of this phenomenon during life is rarely possible and remains confined to isolated echocardiographic reports. These clinical realities call into question the possibility and propriety in diagnosing paradoxical embolism and raise the question of the correct indications for PFO closer. Currently, most of the indications for PFO closure are debated (Table). Cryptogenic strokes, migraine syndrome with aura, decompression sickness and asymptomatic neurological events, right-sided cardiac...
diseases, and peripheral embolism are the clinical scenarios where paradoxical embolism may play clinically meaningful role. The indications for PFO closure in individuals with the history of stroke and clinical suspicion of cerebral paradoxical embolization have attracted particular attention. The meta-analysis of case control studies, recruited patients who had had a stroke, found an increased risk with PFO (odds ratio, 3.10; 95% confidence interval, 2.29–4.21) and even higher risk with PFO accompanied by atrial septal aneurysm (odds ratio, 15.59; 95% confidence interval, 2.83–85.87) in patients ≤55 years of age.\textsuperscript{14} The results of the Evaluation of the STARFlex Septal Closure System in Patients With a Stroke or TIA Due to the Possible Passage of a Clot of Unknown Origin Through a Patent Foramen Ovale (CLOSURE I), Randomized Evaluation of Recurrent Stroke Comparing PFO Closure to Established Current Standard of Care Treatment (RESPECT), and Clinical Trial Comparing Percutaneous Closure of Patent Foramen Ovale Using the Amplatzer PFO Occluder with Medical Treatment in Patients with Cryptogenic Embolism (PC) trials challenged the credibility of a substantial body of observational evidence strongly favoring mechanical closure over medical therapy. In the pooled, intention-to-treat meta-analysis of these trials, the rate of nonfatal ischemic cerebrovascular events and transient ischemic attack was 3.7% with PFO closure and 5.3% with medical therapy, and did not reach a significant difference (odds ratio, 0.70; 95% confidence interval 0.47–1.05).\textsuperscript{15} According to the current recommendations on stroke and on transient ischemic attacks secondary prevention strategies, there are insufficient data to make a recommendation regarding PFO closure in patients with stroke and PFO (class IIb; level of evidence C).\textsuperscript{16} The recommendation reflects a high level of uncertainty in reaching the reliable diagnosis of paradoxical embolism in patients with cryptogenic strokes. The conservative approach is also supported by data showing a trend toward device-related AF inducibility; still, despite the main outcome of the trials, the uncertainty in the evaluation of true clinical significance of the PFO closure still remains. The intention-to-treat analysis was negative, yet the analysis of patients who actually received the assigned treatment, PFO closure was effective. In the as-treated analysis, the rate of recurrent events was 3.6% with PFO closure and 5.8% with medical therapy and reached statistical differences (odds ratio, 0.62; 95% confidence interval, 0.41–0.94).\textsuperscript{15} The recognition of an association between migraine and aura and PFO appears to have come full circle over the past 2 decades, and there is not sufficient evidence to recommend PFO closure for this indication. High-volume divers, compressed-air tunnel workers, high-altitude aviators and astronauts may suffer from decompression sickness and asymptomatic neurological events regardless of the presence of PFO. Multiple recurrences in individuals who wish to continue diving or to continue their high-risk jobs may warrant PFO closure in centers maintaining closure registries or participating in trials. Patients with right-sided cardiac disease affected by compliance abnormalities of right-sided filling and who have remarkable elevation of right atrial pressure are predisposed to right-to-left shunting at the level of PFO. Targeting the treatment at the level of PFO is the mainstay of therapy for these individuals. The control of cyanosis by means of PFO closure may be an intermediate-term palliation; yet, the longer-term worsening of RV muscle function may occur. The noncerebral paradoxical embolisms account for 5% to 10% of all paradoxical embolisms and are much less studied.\textsuperscript{17,18} These events have been described as a cause of MI, renal infarction, and limb ischemia and mainly reported as challenging cases or intriguing cardiovascular images. The presumptive causal connections between benign anatomic alteration and life-threatening diseases has no support from evidence-based medicine.

Paradoxical Embolism as a Probable Cause of Acute Coronary Syndromes

The Young Adult Myocardial Infarction and Ischemic Stroke: The Role of Paradoxical Embolism and Thrombophilia (YAMIS) study, the biggest to date clinical evaluation of relations between PFO and the risk of MI in a group of young survivors with a particularly low prevalence of atherosclerosis, found no relationship between right to left cardiac shunting and MI.\textsuperscript{19} The YAMIS study results were consistent with a previous smaller study on the frequency of PFO in patients with MI and normal coronary arteries.\textsuperscript{20} In the autopsy-studied infarcts at the Johns Hopkins Hospital over a 10-year period, paradoxical embolism to the coronary arteries has also scarcely been reported.\textsuperscript{21} On the other hand, in a study by Dao and Tobis, 2% of patients with highly probable MI of embolic origin were reported in a series of 416 patients referred for evaluation of PFO-related conditions.\textsuperscript{22} The authors, however, raised the hypothesis that some of the myocardial infarctions associated with PFO may not necessarily be related to thrombus embolization itself, but rather to intense coronary vasospasm possibly initiated by vasoactive substances that ordinarily would be metabolized in the lungs. The appreciation of these results and the clinical scenario of the disease make the diagnosis of MI considered with paradoxical embolism extremely difficult, leaving in any case high level of uncertainty. However, extremely rarely diagnosed, paradoxical embolism in a coronary artery is a recognized clinical entity. The most spectacular example was the case of coronary thrombotic embolization described in the left main coronary artery with subsequent surgical intervention that demonstrated a clot in-transit through the PFO.\textsuperscript{23} The other presumptive reports of paradoxical embolism described history of patients with no risk factors for coronary artery disease who presented with acute MIs and were subsequently found to have a right-to-left shunt through PFO. These reports also include cases deemed with air, tumor, and brain tissue embolization.\textsuperscript{24–32} In 1988 Jungbluth et al identified 27 published cases of paradoxical coronary embolism, and 21 additional cases have been reported until 2004.\textsuperscript{33,34} The unique subgroup particularly prone to experience paradoxical embolism comprises patients with PE and PFO.\textsuperscript{13} The elevation of right-sided cardiac chamber pressures from the PE keeps the PFO opened large enough to accommodate the thrombus and to promote its crossing to the left atrium. Subsequently, the thrombus may propagate into the coronary tree. This phenomenon has been described in a very limited number of clinical reports.\textsuperscript{35,36} Rovner et al described the case of a 70-year-old woman with a recent PE who presented 1 week

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\textsuperscript{1} Wąsek et al

\textsuperscript{2} STEMI and PE Attributable to DVT and PFO

\textsuperscript{3} Downloaded from http://circ.ahajournals.org/ by guest on July 31, 2017
later with an acute embolic MI, presumably resulting from a second thrombotic transit causing a paradoxical coronary embolism through a PFO.\(^3\) Haghi and colleagues published the case of a 61-year-old woman with DVT, who presented with PE followed by an acute coronary embolism on the next day.\(^3\) Uchida and colleagues described a case of a 38-year-old man who developed PE and an acute MI simultaneously after laparoscopic surgery.\(^3\) These 3 shortly characterized cases illustrate the difference of the time frame between PE and MI symptoms onset, focusing attention on the similarity in clinical signs and symptoms of both entities and emphasizing the difficulties in diagnosis making when they coincide.

A high level of clinical suspicion should be recommended in light of the data proving PFO related right-to-left atrial shunts in patients with acute major PE being associated with difficulties in diagnosis making when they coincide.

There are no trials assessing whether persons found to have a PFO possibly associated with other than cerebrovascular symptoms and deemed with paradoxical embolism benefit from specific medical or interventional treatments. It is unlikely, such recommendations will ever be published.

Currently, PFO occlusion in suspicion of both cerebral and noncerebral embolization is neither strong nor definitely established. However, uncertain and intuitive with no strong guidelines background, an individualized approach to PFO closure maybe recommended. The implantation of the septal occluder should be considered and maybe justified as a potentially valuable therapeutic option after careful consideration.

**Conclusions**

The elevation of right-sided cardiac chamber pressures from the PE may promote thrombus crossing through the PFO. The simultaneous occurrence of MI and PE in a patient with confirmed DVT should lead to the clinical suspicion of the rarely diagnosed paradoxical embolization. The differential diagnosis between DVT-related PE and simultaneous paradoxical embolism resulting with MI or MI case complicated by DVT and PE in the course of prolonged immobilization is challenging. The presence of right-to-left intracardiac shunt is mandatory, but the timing of the events and the confirmation of the embolic origin of the coronary vessel occlusion are the key elements necessary for the proper evaluation. The imaging limitations of angiography should be considered in making the definitive diagnosis of the paradoxical embolization into the coronary arteries. The current recommendations regarding PFO closure in patients with cryptogenic stroke and PFO do not apply to patients with suspicion of noncerebral paradoxical embolization. The no-guidelines land require an individualized approach and justifies PFO closure after careful consideration.

**Disclosures**

Dr. Wąsek currently holds lectures and provides training courses sponsored by Boston Scientific.

**References**


Key Words: embolism, paradoxical • foramen ovale, patent • myocardial infarction • pulmonary embolism
Unique Case of ST-Segment–Elevation Myocardial Infarction Related to Paradoxical Embolization and Simultaneous Pulmonary Embolization: Clinical Considerations on Indications for Patent Foramen Ovale Closure in No-Guidelines Land

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