Acute right ventricular failure caused by concomitant coronary and pulmonary embolism: successful treatment with endovascular coronary and pulmonary thrombectomy

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Abstract

Patent foramen ovale (PFO) is present in approximately 25% of the general population. PFO is characterized by intermittent shunting of blood from the right to the left atrium, especially in the context of increased right-sided filling pressures, with risk of paradoxical embolism. We describe a 69-year-old woman presenting with acute chest pain, severe dyspnoea, and acute inferolateral ST-segment elevation on the electrocardiogram. The patient was diagnosed with myocardial infarction and failure of the right cardiac ventricle, which was considered to be secondary to extensive pulmonary embolism leading to increased filling pressures and paradoxical coronary embolism. The patient underwent emergent percutaneous interventions with coronary thrombus extraction and pulmonary thrombus fragmentation and local thrombolysis. The patient was free of symptoms at follow up 6 months later and echocardiography showed substantially improved right ventricular function. We discuss issues related to the diagnosis, treatment, and secondary prevention for patients with concomitant pulmonary and coronary arterial thrombosis.

Keywords

Myocardial infarction, patent foramen ovale, percutaneous coronary intervention, pulmonary artery intervention, pulmonary embolism, right heart failure

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Introduction

Respiratory distress and hypoxia in the acute phase of ST-segment elevation myocardial infarction (STEMI) is generally seen in large anterior infarctions resulting in acute heart failure and pulmonary oedema or with acute mechanical complications, including rupture of the ventricular septum, papillary muscle, or free ventricular wall.¹ However, angiographic evidence of occlusion only in smaller coronary branches in conjunction with echocardiography without signs of mechanical complication should lead the clinician to consider concomitant complicating disease. We describe a case of inferolateral STEMI, presenting with chest pain, severe dyspnoea, and hypoxia, related to extensive pulmonary embolism.

Case report

A 69-year-old woman presented with sudden onset of severe retrosternal chest pain. She had a history of hypertension and treatment with 5 mg felodipine daily, but had never smoked and was otherwise healthy. An electrocardiogram recorded in the ambulance showed significant ST-segment elevations in inferior (II, aVF, III) and lateral

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Figure 1. Signal-averaged standard 12-lead electrocardiogram in the acute phase of interolateral ST-segment elevation myocardial infarction, showing significant ST-segment elevation in leads II, aVF, III and V3–V6.

(V3 through V6) leads (Figure 1). In the ambulance, she received a loading dose of aspirin (500 mg) and ticagrelor (180 mg) and was admitted directly from the ambulance to the cardiac catheterization laboratory at our institution. Emergent coronary angiography through radial access revealed occlusion of a right posterior descending branch and two posterolateral branches (Figure 2a). The patient was

treated with thrombus aspiration and balloon dilation two hours after the onset of pain, which resulted in complete reperfusion (Thrombolysis in Myocardial Infarction criteria, grade 3) (Figure 2b). During the procedure, the patient received anticoagulation with 5000 IU heparin and 250 mg bivalirudin. In the absence of remaining stenosis after thrombus aspiration and balloon dilation, no stent was implanted.

A peak troponin T of 4946 ng/l measured 12 hours after the sudden onset of pain indicated a relatively large infarction. However, already at the initial presentation, the patient had severe dyspnoea and an oxygen saturation of only 80% with an oxygen flow rate of 12 l/min through a reservoir mask. Saturation improved only slightly to 85–90% when supported with continuous positive airway pressure of 7.5 cm H₂O during the procedure in the catheterization laboratory, but the chest X-ray was normal, without signs of pulmonary congestion. The patient was also hypotensive with an initial systolic blood pressure of 80 mmHg, which improved to 100 mmHg after the administration of 2 l of intravenous crystalloid volume expander along with infusion of noradrenaline. Inotropic therapy with a low dose of levosimendan (0.05 µg/kg/min) was initiated and infused over 48 hours, and noradrenaline was discontinued. The patient remained slightly hypotensive with a systolic blood pressure of around 100 mmHg, sinus tachycardia with a heart rate of around 120 bpm, and a respiratory rate over 20 per minute with poor oxygen saturation. Attempts to discontinue positive airway pressure therapy resulted in desaturation to below 80% even with an oxygen flow of 12 l/min. Further evaluation with echocardiography did not indicate any mechanical complication but showed a severely dilated right ventricle (basal diameter 54 mm) with severely reduced systolic function (fractional area change 10%).² The left ventricle showed at most a modest

Figure 2. Coronary angiogram with selective catheterization of the right coronary artery: (a) acute phase of interolateral ST-segment elevation myocardial infarction, showing occlusion of a right posterior descending branch and two posterolateral branches; and (b) after thrombus aspiration and balloon dilation, showing complete reperfusion (TIMI grade 3) without any signs of underlying coronary stenosis.

Figure 3. Pulmonary angiogram and intervention in the right pulmonary artery: (a) extensive pulmonary embolism involving right main and the lower lobe artery; (b) thrombectomy device in the pulmonary artery; and (c) reduced clot burden following mechanical thrombus fragmentation.

systolic dysfunction with a normal diameter. Echocardiography also indicated pulmonary hypertension with a slightly elevated peak tricuspid regurgitation velocity, corresponding to a systolic pulmonary pressure of 30 mmHg, and evidence of increased right-sided filling pressure with a dilated right but not left atrium. The inferior vena cava was of normal dimensions with inspiratory collapse. A chest computer tomography performed the same day showed extensive pulmonary embolism, involving all lobar arteries to the right lung and several segmental and subsegmental arteries in both lungs. A prominent pulmonary artery and dilated right ventricle was also noted.

Thrombolytic therapy was considered. However, as the patient had already been treated with dual antiplatelet therapy including both aspirin and ticagrelor, the risk of bleeding was substantial. A percutaneous intervention was therefore considered as a more favourable alternative. Ticagrelor was discontinued and 19 hours after coronary angiography the patient underwent pulmonary angiography with interventional treatment. The pulmonary angiography, performed after placement of a vena cava filter (Celect; William Cook, Bjaeverskov, Denmark), showed extensive pulmonary embolism in segmental and subsegmental branches of both pulmonary arteries, but foremost on the right side (Figure 3a). Clot fragmentation was performed using an over-the-wire Arrow-Trerotola Percutaneous Thrombectomy Device (Arrow International) (Figure 3b) and aspiration with an 8-F guide catheter. At the end of the procedure, a local intra-arterial injection of thrombolytics in the right lower lobe arteries was done. The intervention resulted in a clinical improvement. The blood pressure stabilized at 120 mmHg, positive airway pressure could be discontinued, and an oxygen saturation >95% could be maintained with 5–10 l/min of oxygen.

Figure 4. Contrast echocardiogram in the apical 4-chamber view with injection of agitated saline for contrast, showing large amounts of microbubbles in the dilated right atrium and ventricle (asterisk) and rapid passage of contrast to the left atrium and ventricle (arrow), even without the valsalva manoeuvre.

Following the pulmonary intervention, transthoracic echocardiography with injection of agitated saline microbubbles for contrast revealed a patent foramen ovale and regression of right ventricular dilation and systolic dysfunction (Figure 4). The patient was treated with aspirin 75 mg once daily and warfarin treatment was initiated. After initial infusion of heparin, anticoagulant therapy was continued with enoxaparin 1 mg/kg twice daily until oxygen therapy could be discontinued, warfarin therapy reached a therapeutic level and the vena cava filter could be retrieved. An ultrasound examination of the lower extremity showed

no evidence of venous thrombi. The patient did not have a family history of thrombosis and a blood screening did not indicate any coagulopathy. Therapy with atorvastatin and metoprolol was initiated. The lipid profile was good, with LDL cholesterol of 1.8 mmol/l, HDL cholesterol of 1.3 mmol/l, triglycerides of 0.6 mmol/l, and total cholesterol of 3.5 mmol/l, as were fasting blood glucose levels, liver function tests, renal function, and blood parameters. The patient was discharged after 11 days without need of extra oxygen.

The patient was seen again at the outpatient clinic 1.5 and 6 months later and was completely asymptomatic, without dyspnoea, chest pain, or palpitations with daily physical activity. The pharmacological treatment was well tolerated and no bleeding events were reported. An echocardiographic evaluation 6 months after the initial presentation showed a slightly dilated right ventricle (basal diameter 44 mm) with slightly reduced systolic function (fractional area change 28%) and a slightly elevated peak regurgitation velocity, corresponding to a systolic pulmonary pressure of 30 mmHg. The left ventricle had a normal diameter and a moderately reduced left ventricular ejection fraction (35%). The patient now also recalled that she had experienced increasing dyspnoea on exertion over the 2 weeks before the sudden deterioration and onset of chest pain, indicating that some degree of pulmonary embolism had occurred before the acute presentation with severe pulmonary embolism and coronary embolism. Treatment with warfarin, a statin, and a beta-blocker was continued and treatment with an angiotensin-converting enzyme inhibitor was initiated with regard to left ventricular dysfunction. Biannual examinations at the outpatient clinic were scheduled.

Discussion

This case illustrates how an initial presentation with acute myocardial infarction can mask another underlying condition, in this case pulmonary embolism, where prompt recognition and treatment of both disorders are of major importance.^{3–5} Another reason should be expected in cases of myocardial infarction with severe distress and only thrombotic occlusion of smaller coronary branches, especially with a short time to reperfusion. Severe dyspnoea and hypoxia in patients with inferior STEMI generally suggests mitral insufficiency resulting from rupture of the posteromedial papillary muscle, which typically receives blood supply singly from the posterior descending artery.⁶ Other mechanical complications of myocardial infarction include rupture of the ventricular septum or ventricular wall. Mechanical complications typically present on day 2–3 following myocardial infarction. In our patient, a short duration of the relatively acute symptoms and echocardiographic appearance of a severely dilated, dysfunctional right ventricle did not correlate to the distal coronary occlusions observed on angiography. These findings as well as the absence of the pulmonary

congestion on the chest X-ray did not indicate any mechanical complication. A computed tomography of the chest confirmed the diagnosis of pulmonary embolism. Further evaluation with contrast echocardiography revealed a patent foramen ovale, suggesting paradoxical embolism as the cause of myocardial infarction.

The proposed criteria for diagnosis of paradoxical embolism include evidence of (i) arterial embolism in the absence of a source in the left heart, (ii) a source of embolism in the venous system, and (iii) communication between pulmonary venous and arterial circulation.⁷ A patent foramen ovale is considered common, with a reported prevalence of 25–35% in autopsy series,⁸ but paradoxical coronary embolisms are considered rare, responsible for 5–10% of all paradoxical embolisms. It is a less common cause of coronary embolisms than prosthesis-derived coronary embolisms or coronary embolisms resulting from endocarditis, atrial fibrillation and iatrogenic causes. The great majority, up to 90%, of arterial embolisms occurs in cerebral arteries or in the lower extremities. Until 1953, only 54 cases of coronary embolism had been described in the literature. In recent years, with the more widespread availability of coronary angiography, larger series of cases have been reported.^{5,9} These included at least 17 presumed cases of paradoxical coronary embolism, diagnosed post mortem, of which at least five occurred simultaneously with diagnosed pulmonary embolism.^{10–15} It has been recently suggested that myocardial infarction resulting from paradoxical coronary embolism may be under-diagnosed, in patients with pulmonary embolism a patent foramen ovale.⁵ Coronary embolisms have in autopsy series been reported to lodge distally and occur 3–4-times more often in the left than the right coronary artery.⁹

Our case illustrates also how concomitant coronary and pulmonary embolisms can be successfully treated with dual endovascular interventions with a good outcome. Although percutaneous coronary interventions are now well established as the first line of treatment in STEMI, percutaneous pulmonary interventions have been more seldom reported.¹⁶ These interventions should only be performed by experienced operators in patients with haemodynamic instability or right ventricular failure when there are contraindications to thrombolysis, surgical embolectomy, or in patients who remain unstable after systemic thrombolysis.¹⁷ To our knowledge, sequential coronary thrombectomy and pulmonary thrombus fragmentation and local thrombolysis has not been reported previously. In our patient, coronary thrombus aspiration and balloon dilation were performed. A stent placement was not indicated in the absence of underlying stenosis, which limited the need of long-term antiplatelet therapy. Three cases of successful thrombus aspiration for paradoxical coronary emboli without stenting have been reported previously.^{11–13} While dual antiplatelet therapy is central in the prevention of stent thrombosis and secondary prevention after myocardial infarction, the current standard

in secondary prevention of pulmonary embolism is anticoagulation with warfarin¹⁷ or administration of the novel oral anticoagulants.¹⁸ This is problematic since the combined use of dual antiplatelet therapy and oral anticoagulation (triple antithrombotic therapy), confers substantial risk of bleeding.¹⁹ In the case of a coronary embolus, it is possible that antiplatelet therapy is not necessary at all after successful aspiration, but no data are available to support this hypothesis. In our patient, we elected to use aspirin in combination with warfarin. Recent data indicate that the use of warfarin and clopidogrel are associated with less bleedings compared to triple therapy, without an increase in rate of thromboembolism after PCI.²⁰ Although no randomized studies have compared aspirin with clopidogrel in warfarin-treated patients, the combination of warfarin with clopidogrel might be associated with less bleeding, as aspirin is well known to increase risk of spontaneous gastrointestinal bleeds²¹ and a majority of the bleeding events seen with triple therapy are spontaneous gastrointestinal bleeds.¹⁹

Long-term use of beta-blockers and cholesterol-lowering therapy are indicated to improve outcomes after STEMI. However, some previous studies have suggested detrimental effects of beta-blockers in pulmonary hypertension and right ventricular failure²² but only few studies have examined this issue and shown inconsistent findings.²³ The role of cholesterol-lowering therapy is also unclear in patients with coronary embolism. Some studies have reported a lower incidence of venous thromboembolism with lipid-lowering therapy,²⁴ but the lack of effect in meta-analyses makes it unlikely that statins would provide any meaningful clinical benefits with regard to risk of venous thromboembolic disease,²⁵ why statin therapy could probably be discontinued in our patient – especially in the light of a good lipid profile even without treatment.

Conflicts of interest

The authors declare that there are no conflicts of interest.

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