Floating Thrombus Entrapped in a Patent Foramen Ovale Complicating Acute Pulmonary Embolism

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CASE REPORTS


Despite medical progress in its diagnosis and treatment, acute pulmonary embolism (APE) remains an important cause of mortality, 30–35% in the absence of treatment, with delay of the diagnosis remaining the main cause of death from APE.1 Spiral computed tomography as well as transthoracic echocardiography and/or transesophageal echocardiography (TEE) have become fast and successful examinations in the diagnostic strategy for this affliction.2 We report two cases of bilateral APE without hemodynamic instability but complicated by the discovery of a thrombus trapped in a patent foramen ovale (FO) and floating on both sides in the right and left atrium. We also discuss the various therapeutic modalities.

Case Report

Case 1

A 73-yr-old woman was hospitalized after sudden onset of dyspnea. She had a medical history of recent discovery of an ovarian mass in the course of clarification, complicated with lower limb phlebitis treated by low-molecular-weight heparin by low-molecular-weight heparin for some days. At admission, her arterial blood pressure was 110/75 mmHg, and plasma D-dimers increased at 7.8 μg/ml. Spiral computed tomography confirmed clots in the main pulmonary artery and its right and left branches. The Doppler echography of the lower limbs revealed a right popliteal and posterior-tibial deep venous thrombosis without any floating thrombus in the femoral vein; therefore, intravenous heparin therapy was initiated. The next day, routine transthoracic echocardiography showed normal right and left cardiac function but also revealed a mobile structure in the right atrium. TEE confirmed the presence of a long serpentine thrombus trapped in the FO and floating in both the right and left atria (fig. 1). The patient was then transferred to our institution and, after deployment of an inferior vena cava filter by the femoral route, underwent surgical thrombectomy during cardiopulmonary bypass with closure of the FO. A 20-cm-long thrombus was removed from the FO (fig. 2), and others were removed from the pulmonary network. The patient’s course was simple, and she left the intensive care unit after the third postoperative day.

Discussion

The diagnosis of APE remains a challenge for physicians. Lorut et al.2 showed that the combination of ventilation/perfusion lung scan, spiral computed tomography, dosage of plasma D-dimer, and, in some cases, lower limb venous Doppler echography was an effective and noninvasive strategy to confirm APE. Echocardiography (transthoracic echocardiography and/or TEE) is a useful additional examination for the diagnosis of APE, particularly in hemodynamically unstable patients. Echocardiographic abnormalities are indirect signs of pulmonary arterial hypertension and right heart failure signs, which are right cavity dilation, hypokinetic right ventricle, and interventricular septum flattening, with even paradoxical motion.5,6 Embolized thrombus visualization in the right cavities is rare, and its precise incidence is not known. Three types of thrombus can be identified according to their morphology, their origin, and their clinical meaning. The first one, type A, is an intracardiac floating thrombus, serpentine and highly mobile, which can even get through the tricuspid valve or even the pulmonary valve during cardiac beats. Type A thrombus is seen in patients with deep venous thrombosis and corresponds to the migration of a peripheral thrombus. The second type of intracardiac thrombus, type B, is similar to the thrombus of the left cavities and is rather well fixed to the cardiac walls. This thrombus is rarely associated with peripheral deep venous thrombosis but is generally seen in patients with cardiac, potentially thrombogenic, pathologies such as heart failure with cardiac blood flow stasis.

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because it does not have the shape of a snake, and unlike type B, because it is very mobile and looks like a myxome. The short-term prognosis of patients with type A thrombus is significantly worse than those with type B. In type A, early mortality (<8 days) related to the thrombus is 40%, versus only 9% for type B. Therefore, discovery of an intracardiac type A thrombus must be considered as a real emergency, and treatment must be initiated immediately. The cause of death for type A is generally a massive and quickly fatal embolism, which is rarely the case for type B. Mortality is intermediate with intracardiac type C thrombus.

We think that the systematic performance of TEE in all patients presenting with central APE, even those without hemodynamic compromise, is a minimally invasive and safe examination that would allow the early detection of an intracardiac floating thrombus and would allow one to choose the best therapeutic option. Echocardiographic studies including patients presenting with APE estimate the presence of an intracardiac floating thrombus from 3 to 23%, with, in these cases, short-term significant increased mortality of 26–50%, which is sharply superior to the 8–10% mortality associated with “isolated” APE. Intracardiac tumor, vegetation, interatrial aneurysm, and embryonic remainder, such as the Chiari network, which is an embryologic membrane extending from the ostium of the inferior vena cava and the coronary sinus to the interatrial septum and the tricuspid valve or the thebesian and eustachian valves, are the main differential diagnoses to exclude.

The prognosis of a typical type B thrombus is good, and intravenous heparin therapy is generally sufficient. However, there is currently no consensus for the ideal therapeutic management of APE associated with type A thrombus, and data in the literature vary greatly. On the basis of a comparable mortality for intravenous heparin therapy, systemic fibrinolysis, or surgical thrombectomy, Kinney and Wright concluded in 1989 that intravenous heparin therapy was the first treatment of choice. Later, Rose et al. reviewed all of the cases of intracardiac thrombus published in the English-speaking literature between 1966 and 2000 that included sufficient clinical data. They thus analyzed 177 patients, among whom 98% presented with APE. The mortality rates associated with the absence of treatment, with anticoagulation therapy, with surgical thrombectomy, and with fibrinolysis were 100, 29, 24, and 11%, respectively, which suggests that thrombolytic treatment is the treatment of choice in the absence of any contraindication. However, it is important to note that the great majority of the reported cases corresponded to right cavity floating thrombi and that the rate of thrombus entrapped through the FO was extremely weak, even not clarified. It was not possible for us to find specific recommendations in the therapeutic management of these particular cases with high risks not only of pulmonary embolism but also of paradoxical embolism. Given the high risks of massive pulmonary and systemic embolism, we chose the surgical option for the patients in these cases. This choice also allowed resection of the intracardiac thrombus, treatment of the clots located in the pulmonary arterial trunk and its main branches, and closure of the FO. Intravenous anticoagulation therapy with heparin was then introduced immediately in the postoperative period. We chose to use a vena cava filter in these two patients, but we recognize that there is considerable debate on this issue. The recognized indications are the extension of venous thrombosis or an ongoing pulmonary embolism in appropriately treated patients for more than 48 h and contraindications to anticoagulant therapy, whereas temporary filters may be discussed immediately after surgical embolectomy because of the increased risk of bleeding during the first 24–72 postoperative hours with full intravenous heparin therapy.
Lower Esophageal Sphincter Pressure Measurement during Cardiac Arrest in Humans: Potential Implications for Ventilation of the Unprotected Airway

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IN an unprotected airway, distribution of ventilation volume between the lungs and stomach depends mainly on patient variables such as lower esophageal sphincter pressure (LESP), airway resistance, and respiratory system compliance.1 The combination of these variables and ventilatory techniques determines upper airway pressure and therefore gas distribution between the lungs, esophagus, and stomach.2 In an animal model, investigators showed that LESP decreases during cardiac arrest from approximately 20 H2O to approximately 5 cm H2O.3,4 When such a pressure is used in a bench model of simulated ventilation of an unprotected airway,5 stomach ventilation is almost inevitable. Stomach inflation increases intragastric pressure,6 elevates the diaphragm, restricts lung movement, and in turn reduces respiratory system compliance and lung ventilation.7,8

which may cause severe complications such as aspiration, pneumonia, and possibly death.9,10

Unfortunately, knowledge about LESP physiology during cardiopulmonary resuscitation is limited to one animal model.3 The objective of the current study was to verify the rate of decline of LESP before, during, and after cardiovascular collapse and cardiac arrest in humans. After institutional review board approval, we measured LESP in three patients undergoing withdrawal of life support in an intensive care unit. This study may be as close as it is possible to get to realistically assessing human LESP physiology immediately before, during, and after sudden cardiac arrest.

Case Reports

We studied three adult individuals, ranging in age from 53 to 77 yr, all affected by devastating hemorrhagic strokes and in deep coma. Arterial blood gases or arterial oxygen saturation was compatible with good arterial oxygenation during mechanical ventilation.

After confirmation of irreversible devastating neurologic injury in all patients and family decision to withdraw care, informed consent was obtained. While the patient was still supported by mechanical ventilation, esophageal manometry was performed via the nares with a low-compliance perfusion esophageal manometry system (Arndorfer Infusion, Greenwich, WI; Medtronic Polygraph, Minneapolis, MN). After recording a baseline measurement, continuous pressure measurements of the LESP were then taken upon extubation until the patient expired (asystole) and for several minutes after the cardiac arrest up to reaching a steady state nadir. Normal LESP in our laboratory is 15–30 mmHg. Withdrawal of care was performed through discontinuation of all medications except morphine and extubation of the trachea to room air. In all cases, the patients’ peripheral oximetry desaturated rapidly, and they became bradycardic and then asystolic while esophageal pressure was recorded (fig. 1).

Lower esophageal sphincter pressure decreased rapidly from normal baseline during cardiocirculatory collapse in all three patients under-

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going withdrawal of life support, which is similar to previous observations of LESP in an animal model of cardiac arrest.³

Discussion

Lower esophageal sphincter pressure is one the mechanisms that prevents stomach inflation during positive-pressure ventilation. If our observations of LESP physiology before, during, and after cardiovascular collapse in three intensive care unit patients can be extrapolated to critically ill victims or during cardiac arrest, emergency ventilation management should be always performed in a manner that assumes that the LESP may be unable to prevent air from entering the stomach. Therefore, emergency ventilation should be performed in a manner that minimizes peak inspiratory pressure, because respiratory physiology favors substantial stomach inflation when LESP is critically decreased.² For example, any ventilation strategy generating peak airway pressures above 5 cm H₂O in a cardiac arrest patient with an unprotected airway may cause massive stomach inflation before the airway can be protected with an advanced airway device.⁵ Stomach inflation can also be prevented in many patients by applying pressure over the cricoid to seal off the esophagus, when feasible.¹¹,¹² A recent study of out-of-hospital cardiac arrest showed that professional rescuers use ventilation techniques that would make stomach inflation very likely in the presence of an unprotected airway.¹³ Furthermore, even after a period of intensive retraining, rescuers continued to apply ventilation that resulted in excessive airway pressure. Therefore, basic and advanced life support training courses may require special emphasis on teaching the psychomotor skills necessary for good airway management practices to prevent harm.

Previously, we have developed several strategies to prevent stomach inflation during ventilation of a cardiac arrest victim with an unprotected airway by decreasing peak airway pressure with small instead of large tidal volumes,¹⁴ decreasing peak inspiratory flow,¹⁵,¹⁶ or using pure oxygen instead of room air.¹⁷ For example, proper oxygenation and carbon dioxide elimination could be achieved with tidal volumes of 350 ml pure oxygen in anesthetized supine patients.¹⁸ Because ventilation requirements during cardiopulmonary resuscitation may be less than during intact circulation¹⁹ and excessive ventilation may decrease the time for life-saving chest compressions and decrease venous return,¹⁵ it may be important to limit tidal volume to 300–500 ml, a goal usually achieved by observing an adequate chest rise after resuscitation using bag ventilation.²⁰

Although the role of ventilation strategies to prevent stomach inflation in an unprotected airway has been discussed in general during cardiac arrest,¹–³,⁵,⁸ it may be important to extrapolate ventilation strategies for unintubated cardiac arrest patients to other unintubated
patients with severe shock states, who often need basic life support ventilation before undergoing intubation. Intuitively, cricoid pressure should be always attempted when an extra set of hands is available.

There are several limitations to the current case report series. First, our patients had severe underlying neurologic injury and may not be exactly comparable to patients with other prehospital conditions of sudden cardiac arrest, dysrhythmia, or hypoxia. Second, two of three patients received vasoactive medications before life support was withdrawn, which may have affected measurements. Third, the mechanism of death in our patients reflects initial hypoxia, hypercapnia, and a rapid decreasing perfusion always resulting in asystolic cardiac arrest, which may be different when compared with out-of-hospital patients with sudden ventricular fibrillation.

In conclusion, after withdrawal of life support in intensive care unit patients, LESP decreased rapidly before cardiac arrest, from a baseline of approximately 20 cm H₂O. Invariably, at the time of cardiac arrest, LESP averaged 5 cm H₂O.

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