Clinical Features of Acute Massive Pulmonary Embolism Complicated by Radiofrequency Ablation

An Observational Study

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Abstract: Although pulmonary embolism (PE) complicated by radiofrequency catheter ablation (RFCA) is rare, it can be life-threatening. Our goal was to elucidate the clinical features of acute massive PE after RFCA.

Of 2386 patients who underwent RFCA for supraventricular tachycardia or idiopathic ventricular arrhythmia, 4 patients (0.16%) whose cases were complicated by acute massive PE were examined.

The 4 patients were female and middle-aged (range 43–52 years), and 2 of the 4 patients had iron-deficiency anemia and reactive thrombocytosis. Ablation in all patients was performed in the left heart ventricle. All of the patients had a long-duration hemostasis procedure and bed rest following femoral arterial sheath removal after RFCA. All of the patients collapsed and lost consciousness during their first attempt at walking after RFCA. The emergent electrocardiogram in 2 of the 4 patients revealed an S1Q3T3 pattern, 1 patient demonstrated new onset of right bundle-branch block (RBBB) and S1Q3 pattern and Qr pattern in V1, and the remaining patient had negative T waves in leads V1, V2, and III. The emergent echocardiogram revealed right ventricular hypokinesis and pulmonary hypertension in the 4 patients with acute PE after ablation. Although all of the patients initially experienced sinus tachycardia when they recovered consciousness, 2 of the 4 patients suddenly developed intense bradycardia and lost consciousness again, and these patients finally died (50% fatality rate). All of the patients were identified by CT pulmonary angiography or pulmonary angiography.

Our report suggests that although acute massive PE is highly rare, there is a real and fatal risk in patients who experienced acute massive PE after RFCA. Particular attention should be paid to avoid this complication, no matter how careful the procedure is, according to the manufacturer’s instructions. All of the patients were verified as having no structural heart disease before catheter ablation by routine biochemistry tests, 12-lead rest electrocardiogram (ECG), x-ray, and color echocardiography examination.

INTRODUCTION

Radiofrequency catheter ablation (RFCA) of symptomatic arrhythmias has enjoyed unprecedented growth over the past 2 decades. This growth has been attributed to its high success rate in the treatment of a variety of arrhythmias and the low complication rate (1.4%–5%) observed in these procedures.1–3 The most severe complications associated with RFCA include death, stroke, complete atrioventricular block, cardiac tamponade, acute myocardial infarction, and thromboembolism.6 The risk of developing symptomatic pulmonary embolism (PE) from electrophysiologic procedures is reportedly 0% to 1.7%.1–6 PE is a very rare complication of RFCA,1–6 but it can be fatal. Only a few isolated cases of PE after RFCA have been reported.7,8 Although careful attention should be paid to avoid this complication, no previous reports have yet systematically addressed this problem. The aim of this study was to elucidate the clinical features of acute massive PE after RFCA.

METHODS

Study Population

From January 2008 to December 2014, a total of 2386 consecutive patients without structural heart disease were presented for catheter ablation for atrioventricular nodal reentrant tachycardia (AVNRT), accessory pathways (AP), and idiopathic ventricular arrhythmias including premature ventricular complexes and ventricular tachycardias (PVC/IVTs) in our hospital. All of the patients were verified as having no structural heart disease before catheter ablation by routine biochemistry tests, 12-lead rest electrocardiogram (ECG), x-ray, and color echocardiography examination.

Ethics Approval

Approval was obtained from the Ethics Committee of the Second Affiliated Hospital of Wenzhou Medical University, and all of the patients gave their written informed consent before the procedure.
| Patient 1 | Patient 2 | Patient 3 | Patient 4 |
|----------|----------|----------|----------|
| **Age/sex** | 43 Years/female | 45 Years/female | 48 Years/female | 52 Years/female |
| **BMI** | 23.5 | 23.7 | 22.9 | 23.4 |
| **Arrhythmia** | Idiopathic PVCs, left ASC origin | AVRT, left posteroseptal AP | AVRT, left anterolateral AP | Idiopathic PVC/IVTs, left anterolateral MA origin |
| **Comorbidities** | IDA | IDA, HT | None | None |
| **Hemoglobin, g/L** | 99 | 85 | 125 | 130 |
| **Platelet count, cells/L** | $411 \times 10^9$ | $394 \times 10^9$ | $226 \times 10^9$ | $168 \times 10^9$ |
| **Arrhythmia** | 3.81 | 3.38 | 3.03 | 3.92 |
| **Prior DVT** | No | No | No | No |
| **Prior PE** | No | No | No | No |
| **Femoral arterial sheath placement** | Yes | Yes | Yes | Yes |
| **Femoral venous sheath placement** | No | Yes | Yes | Yes |
| **Ablation** | Left heart | Left heart | Left heart | Left heart |
| **RF Lesions (no.)** | 2 | 1 | 2 | 3 |
| **Procedure time/radiation exposure, min** | 100/12 | 70/6 | 80/9 | 95/11 |
| **ACT during procedure, s** | 270 | 260 | 252 | 284 |
| **Time from the end of procedure to sheath removal, min** | 30 | 30 | 30 | 30 |
| **Immobilization time after sheath removal, h** | 24 | 24 | 24 | 24 |
| **Elastic bandage compression time, h** | 24 | 24 | 24 | 24 |
| **Onset of PE** | 24 h after RFCA | 24 h after RFCA | 24 h after RFCA | 24 h after RFCA |
| **First clinical sign** | Loss of consciousness | Loss of consciousness | Loss of consciousness | Loss of consciousness |
| **Trigger of onset** | First walk after RFCA | First walk after RFCA | First walk after RFCA | First walk after RFCA |
| **HR recovering consciousness, bpm** | 102 | 140 | 110 | 100 |
| **BP recovering consciousness, mmHg** | 80/54 | 85/45 | 90/60 | 98/62 |
| **Emergent ECG** | $S_1Q_3T_3$ pattern | $S_1Q_3$ pattern and RBBB | $S_1Q_3T_3$ pattern | Negative T waves in $V_{1-2}$ and III |
| **RV hypokinesis** | Yes | Yes | Yes | Yes |
| **PASP, mmHg** | 45 | 57 | 52 | 43 |
| **RV dimension, mm** | 31 | 38 | 39 | 32 |
| **DVT** | Yes | No | No | Yes |
| **Thrombus in CTPA or PA** | Bilateral pulmonary arteries | Bilateral lobar pulmonary artery | Bilateral pulmonary arteries | Bilateral pulmonary arteries |
| **Treatment of PE** | Intra-venous thrombolysis | Yes | Yes | No |
| **LMWH** | Yes | No | No | Yes |
| **Vitamin K antagonist** | Yes | No | No | Yes |
| **Catheter thrombectomy** | No | No | No | No |
| **Surgical embolectomy** | No | No | No | No |
| **Recurrent loss of consciousness (Cardiac arrest)** | No | Yes | Yes | No |
| **Outcomes** | Alive | Dead | Dead | Alive |

Act = activated clotting time, AP = accessory pathway, ASC = aortic sinus cusp, AVRT = atrioventricular reentrant tachycardia, BMI = body mass index, BP = blood pressure, bpm = beats/min, CTPA = CT pulmonary angiography, DVT = deep vein thrombosis, ECG = electrocardiogram, HR = heart rate, HT = hyperthyroidism, IDA = iron-deficiency anemia, IVTs = idiopathic ventricular tachycardias, LMWH = low-molecular-weight heparin, MA = mitral annulus, PA = pulmonary angiography, PASP = pulmonary artery systolic pressure, PE = pulmonary embolism, PVCs = premature ventricular contractions, RBBB = right bundle-branch block, RF = radiofrequency, RFCA = radiofrequency catheter ablation, RV = Right ventricle.
Electrophysiologic Study and RFCA

Patients were taken off anti-arrhythmic drugs for at least 5 half-lives before electrophysiologic studies. All ablation procedures were done under local anesthesia. Under fluoroscopic guidance, 3 standard multielectrode catheters were inserted via the internal jugular and femoral veins and located at the coronary sinus, His bundle region, high right atrium, or right ventricle (RV) for patients with AVNRT or AP. An additional 7F sheath was introduced in the femoral artery and advanced into the left ventricle for patients with left-sided AP or ventricular arrhythmias. The standard techniques and protocols were used for the electrophysiologic study. Diagnosis of AVNRT, AP, and PVC/IVTs was made based on standard criteria.9 If the clinical arrhythmia did not occur spontaneously and was not induced at baseline, intravenous isoproterenol (2–4 μg/min) was administered to induce arrhythmia. A 7F quadripolar deflectable catheter with a 4-mm-tip electrode was used for mapping and ablation for patients with AVNRT, AP.

FIGURE 1. Serial ECGs in case 1. (A) ECG after RFCA. (B) ECG performed after onset of acute pulmonary embolism demonstrating T wave inversion in leads III, V1, and V2 and an S1Q3T3 pattern. (C) ECG performed 2 days after onset of acute pulmonary embolism showing a resolution of the T wave abnormalities in lead III. (D) ECG performed 5 days after onset of acute pulmonary embolism showing no S1Q3T3 pattern.
and PVC/IVTs. Bolus injection of 2000 U unfractionated heparin was done after right-sided catheter insertion, and bolus injection of 3000-U unfractionated heparin followed by maintenance infusion of 1000 U/h was done after left-sided catheter insertion. All catheters and sheaths were removed at the end of the procedures. Hemostasis was maintained by hand compression, which was subsequently maintained by elastic bandage for 12 hours. Commonly, the immobilization time was 6 hours if only venous sheath had been placed, and 12 hours if an arterial sheath had also been placed. All of the patients underwent 24-hours ECG monitoring after the procedure. After RFCA, patients lay in bed for 6 to 12 hours if only venous sheath had been placed and 12 to 24 hours if an additional arterial sheath had also been placed.

RESULTS

Prevalence of Acute Massive PE

Acute massive PE occurred in 4 patients, whose clinical characteristics are shown in Table 1. The incidence of acute massive PE was 0.16%. Acute massive PE was defined as systolic arterial pressure <90 mmHg or at least 2 occluded lobar pulmonary arteries. All of the patients were identified by CT pulmonary angiography or pulmonary angiography. PE was suspected in 2 other patients who were excluded from this study because the diagnosis of PE was not finally confirmed.

Clinical Characteristics of Acute Massive PE

All of the patients were female, and the mean age was 47 ± 3.9 years (range 43–52 years). No obesity was observed (BMI 23.3 ± 0.33 [range 22.9–23.7]). The medical history was significant for iron-deficiency anemia without any treatment in 2 patients (case 1 and 2) and hypertension in 1 patient (case 2). All of the patients had no history of past surgeries and thromboembolic events. After admission, a routine blood test in cases 1 and 2 revealed a red blood cell count of 5.03 × 10¹² cells/L and 4.54 × 10¹² cells/L (normal range: 3.5–5.0 × 10¹²), hemoglobin of 99 and 85 g/L (normal range: 110–150), hematocrit 0.32 and 0.30 (normal range: 0.37–0.47), mean RBC volume 64.2 and 65.4 fl (normal range: 80–100), mean corpuscular hemoglobin 19.7 and 18.7 pg (normal range: 27–34), mean corpuscular hemoglobin concentration 307 and 286 g/L (normal range: 320–360), platelet count of 411 × 10⁹ and 394 × 10⁹ cells/L (normal range: 100–300 × 10⁹), and thrombocytocrit 0.45 and 0.35 (normal range: 0.11–0.28), respectively. Blood coagulation tests including cardiac troponin I levels, brain natriuretic peptide levels, a chest x-ray, and echocardiographic parameters were within the normal range in all of the patients.

The 4 patients had different ablative procedure types; case 1 used ablation of PVCs arising from the left aortic sinus cusp, case 2 had ablation of the left posteroseptal AP, case 3 had ablation of the left anterolateral AP, and case 4 had ablation of PVC/IVTs arising from the anterolateral portion of the mitral annulus. Only the arterial sheath was inserted in case 1, and both venous and arterial accesses were used in the remaining 3 patients. Activated clotting time during procedure was 266.5 ± 13.8 seconds in the 4 patients. Ablation was successfully performed in the left heart via the right femoral arterial approach in all of the patients. No complications occurred during the mapping or ablation procedure. Procedure duration (from puncture to removal of sheath catheter) and radiation exposure were comparable across patients.
exposure time were 86.2 ± 13.7 (range 70~100) and 9.5 ± 2.6 minutes (range 6~12), respectively. The number of radiofrequency applications required for a successful ablation was 2.0 ± 0.82 (range 1–3). Time from the end of procedure to sheath removal was 30 minutes in all patients. Small hematomas were observed around the puncture sites of the right femoral artery after ablation in the 4 patients. To achieve complete hemostasis, all of the patients had a long-duration hemostasis procedure (elastic bandage compression time for 24 hours and immobilization time for 24 hours) and bed rest (24 hours) following femoral arterial sheath removal after RFCA.

Onset of Acute Massive PE
All of the patients suddenly collapsed and lost consciousness (syncope) at PE onset. All of the patients demonstrated their symptoms during their first attempt at walking 24 hours after RFCA. Of 4 patients, 3 recovered spontaneously after 30 seconds to 1 minute, and the remaining patient (case 2) recovered after minutes of cardiac compression. Hypotension (<100 mmHg), tachycardia (rest heart rate ≥100 beats/min), and low arterial oxygen saturation (<96%) were observed in all of the patients when they recovered consciousness.

Acute PE Diagnosis
Acute PE was suspected at PE onset in all of the patients. An emergent ECG was performed in each of the 4 patients (Figures 1–4). The ECG in 2 of the 4 patients (cases 1 and 3) revealed a S1Q3T3 pattern (Figures 1 and 3), and in the case 2 demonstrated new onset of right bundle-branch block (RBBB) and a S1Q3 pattern and Qr pattern in V1 (Fig. 2), and in the case 4 demonstrated negative T waves in leads V1, V2, and III (Fig. 4). To differentiate between PE and cardiac tamponade, an emergent bedside echocardiogram was performed in all of the patients, which revealed right ventricular hypokinesis and dilation (RV dimension 35.0 ± 4.1 mm), pulmonary hypertension (pulmonary artery systolic pressure 49.3 ± 6.4 mmHg), and no pericardial tamponade (Table 1). Deep venous thrombosis (DVT) was detected by duplex ultrasonography in 2 of 4 patients. Of the 4 patients, 3 underwent emergent CT pulmonary angiography, which demonstrated bilateral massive PE, and the remaining patient (case 2) underwent emergent pulmonary angiography and autopsy, which revealed occlusive clots in the upper segments of left lobar pulmonary artery (Figures 5–8).

Treatment and Outcomes for Acute Massive PE After RFCA
Of the 4 patients, 2 (Cases 1 and 4) maintained stable hemodynamics after inotropic support (dopamine) and standard anticoagulation treatment (low-molecular-weight heparin and vitamin K antagonist). A repeated CT pulmonary angiography later revealed a significantly improved filling defect within either pulmonary artery in cases 1 and 4 (Figures 5 and 8). However, intense bradycardia (heart rate <35 beats/min) suddenly occurred, and consciousness was lost again in the
remaining 2 patients (cases 2 and 3). Despite efforts at resuscitation and treatment (urokinase administration and catheter thrombectomy in case 2, and urokinase administration in case 3), their consciousness was not recovered and their hemodynamics could not be stably maintained, and the 2 patients were finally pronounced dead.

**DISCUSSION**

One of the most feared complications of catheter ablation procedures is the development of a thromboembolism. PE complicated by RFCA was very rare in previous reports, but it may be underestimated and life-threatening. Asymptomatic or mild PE is likely much more common than symptomatic PE, complicating electrophysiologic procedures. In this series, acute massive PE complicated 0.16% of RFCA with very high case fatality rate (50%). In our patients, acute massive PE clearly occurred after RFCA. PE might be related to the development of DVT after RFCA rather than the procedure itself. Several studies have reported that asymptomatic femoral DVT following venous sheath placement occurred in 5% to 44% of patients. However, none of these patients with DVT experienced symptomatic PE. Although femoral DVT was not detected in 2 of 4 patients in the study, it was likely that the whole thrombus had already detached and embolized.

An important finding was that the first clinical sign of acute massive PE after RFCA was sudden loss of consciousness (syncope) in the 4 cases listed in Table 1. The loss of consciousness in the 4 cases occurred during their first attempt at walking after RFCA. The results are in agreement with an earlier study demonstrating that most patients caught PE on their first walk after surgery for lung cancer. The first ambulation might cause detachment of the thrombus from the femoral vein and subsequent PE. Another important finding was that all of the patients had a long-duration hemostasis procedure (elastic bandage compression time for 24 hours and immobilization time for 24 hours) and bed rest (24 hours) for the femoral artery sheath. Hand compression and the elastic bandage at the punctured sites of the right femoral artery may also compress the right femoral vein simultaneously leading to

![FIGURE 4](image-url). Serial ECGs in case 4. (A) ECG performed after onset of acute pulmonary embolism demonstrating T wave inversion in leads III, V1, and V2. (B) ECG performed 2 hours after onset of acute pulmonary embolism demonstrating progressive T wave inversion in leads III, V1, and V2. (C) ECG performed 12 days after onset of acute pulmonary embolism demonstrating a resolution of the T wave abnormalities in leads III, V1, and V2.)
venous stasis, especially in case 1. The patient without femoral vein cannulation in case 1 developed acute massive PE after RFCA. Commonly, the elastic bandage compression time and the immobilization time were only 12 hours for most patients who had undergone RFCA after femoral arterial puncture in our hospital. But because small hematomas were observed around the puncture sites of the right femoral artery, the elastic bandage compression time and the immobilization time were 24 hours for the 4 patients with acute massive PE after ablation so as to achieve complete hemostasis, and were longer than other patients who had undergone catheter ablation without PE after femoral arterial puncture. The long immobilization time of the patients because of the femoral artery hemostasis procedure facilitated the formation of a thrombus in the femoral vein, which caused a subsequent PE upon mobilization. In addition, all patients in the report were female and middle-aged (range 43–52 years). Similar to our cases, Bauer et al.17 also reported a middle-aged woman (age 47 years) who developed a massive PE with cardiac arrest after an intracardiac electrophysiological study and found that the long immobilization time postprocedure was a cause for DVT formation and subsequent PE. In the present report, 2 of the 4 patients had iron-deficiency anemia and reactive thrombocytosis. There is substantial evidence that iron-deficiency anemia induces a hypercoagulable state.18–23 Jiménez et al.22 reported that patients with anemia had a higher risk of fatal PE. Our report and other studies22,23 support that anemia may play a role in the development of venous thrombosis and PE. All the 4 patients had also no history and family history of thromboembolic events. Therefore, acute PE after ablation may not be related with genetic disorders in the present study.

Early PE diagnosis is often difficult to establish. Clinical alertness is important for PE diagnosis complicated by RFCA. It may be necessary to differentiate acute PE from other forms of circulatory and respiratory failure after RFCA such as acute pericardial tamponade and acute myocardial infarction. When syncope, circulatory arrest, or sudden respiratory distress accompanied by changes in clinical signs are observed after RFCA, especially on the first walk after RFCA, acute PE should be strongly suspected. In the report, emergent ECG in cases 1 and 3 demonstrated an S1Q3T3 pattern, and case 2 demonstrated new onset of RBBB and S1Q3 pattern and Qr pattern in V1, and case 4 demonstrated negative T waves in leads V1, V2, and III. Chou24 suggested that typical ECG findings in PE are as follows: an S1Q3 or S1Q3T3 pattern; a rightward shift of the QRS axis; transient, incomplete or complete RBBB; and T wave inversion in the right precordial leads. The S1Q3T3 pattern or RBBB or T wave inversion in the right precordial leads are more common with massive embolism than with smaller emboli and correlate with worse short-term prognosis in acute PE.10 Tachycardia is the most common ECG abnormality in PE.15,25 Each patient initially experienced sinus tachycardia when they recovered consciousness, then 2 of the 4 patients suddenly developed intense bradycardia and finally died. The new onset of the S1Q3T3 pattern or RBBB or T wave inversion in the right precordial leads was helpful for making an early diagnosis of acute massive PE after RFCA. In addition, emergent bedside echocardiogram revealed right ventricular hypokinesis and dilation and pulmonary hypertension in all patients with acute massive PE after ablation in the report. Thus, early detection of echocardiographic right ventricular dysfunction is also of importance for making an early diagnosis of acute massive

FIGURE 5. CT pulmonary angiography (CTPA) in case 1. CTPA with 2-dimensional imaging (A) and 3-dimensional imaging (B) performed after acute pulmonary embolism onset demonstrating thrombi in bilateral main pulmonary artery (arrow) that were more prominent on the right side. Repeated CTPA with a 2-dimensional imaging (C) and a 3-dimensional imaging (D) performed 10 days after onset of acute pulmonary embolism demonstrating a significantly improved filling defect within either pulmonary artery.
PE after RFCA. In the report, the 4 patients were finally identified by CTPA or pulmonary angiography. The imaging studies had greater sensitivity and specificity for detecting PE, but it took more time and more risk to finish the imaging studies than ECG. When the patient’s condition was not stabilized and the patient was moved unsafely, transesophageal echocardiography may confirm the diagnosis by showing emboli in the main pulmonary arteries.

Although acute massive PE after RFCA was identified and treated early, the case fatality rate was 50% (2/4) in the present report. Therefore, active prevention for PE after RFCA is very important. Early ambulation and the shortened time that the
patients were immobilized and the puncture sites were compressed after RFCA were vital to prevent DVT formation, especially in patients with femoral arterial cannula. In the report, ablation in all of the patients was performed in the left heart via the right femoral arterial approach, which led to the prolonged compression for femoral artery hemostasis and bed rest following femoral arterial sheath removal after RFCA. Special attention should be paid to patients during their first attempt at walking after RFCA. A duplex ultrasonography before getting up to walk to evaluate thrombus formation at the compressed and/or punctured femoral veins may be appropriate for patients with a potential risk for DVT.7

In conclusion, our report suggests that although acute massive PE is very rare, there is a real and fatal risk in patients who experienced acute massive PE after RFCA. Particular attention should be paid to the first ambulation after RFCA. Acute PE should be strongly suspected when a sudden loss of consciousness occurs upon mobilization after RFCA. The new onset of the S1Q3T3 pattern, RBBB, or T wave inversion in the right precordial leads may be useful for making an early diagnosis of acute PE after RFCA. Early ambulation after left-sided RFCA might be helpful to prevent DVT formation and subsequent PE.

REFERENCES

1. Zipes DP, DiMarco JP, Gillette PC, et al. Guidelines for clinical intracardiac electrophysiological and catheter ablation procedures. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Clinical Intracardiac Electrophysiologic and Catheter Ablation Procedures), developed in collaboration with the North American Society of Pacing and Electrophysiology. J Am Coll Cardiol. 1995;26:555–573.

2. Hindricks G. The Multicentre European Radiofrequency Survey (MERFS): complications of radiofrequency catheter ablation of arrhythmias. The Multicentre European Radiofrequency Survey (MERFS) investigators of the Working Group on Arrhythmias of the European Society of Cardiology. Eur Heart J. 1993;14: 1644–1653.

3. O’Hara GE, Philippon F, Champagne J, et al. Catheter ablation for cardiac arrhythmias: A 14-year experience with 5330 consecutive patients at the Quebec Heart Institute, Laval Hospital. Can J Cardiol. 2007;23(Suppl B):67B–70B.

4. Calkins H, Yong P, Miller JM, et al. Catheter ablation of accessory pathways, atrioventricular nodal reentrant tachycardia, and the atrioventricular junction: final results of a prospective, multicenter clinical trial. The Atakr Multicenter Investigators Group. Circulation. 1999;99:262–270.

5. Horowitz L. Safety of electrophysiologic studies. Circulation. 1986;73:28–31.

6. Horowitz L, Kay H, Kutalek S, et al. Risks and complications of clinical cardiac electrophysiologic studies: a prospective analysis of 1,000 consecutive patients. J Am Coll Cardiol. 1987;9:1261–1268.

7. Hung CY, Lin TC, Hsieh YC, et al. Acute massive pulmonary embolism after radiofrequency catheter ablation: a rare complication after a common procedure. J Chin Med Assoc. 2012;75:409–412.
8. Lu CR, Chen JY, Hsu CH, et al. Acute massive pulmonary embolism after radiofrequency catheter ablation: a rare but devastating complication. *Tex Heart Inst J.* 2010;37:498–499.

9. Nakagawa H, Jackman WM. Catheter ablation of paroxysmal supraventricular tachycardia. *Circulation.* 2007;116:2465–2478.

10. Jaff MR, McMurtry MS, Archer SL, et al. Management of massive and submassive pulmonary embolism, iliofemoral deep vein thrombosis, and chronic thromboembolic pulmonary hypertension: a scientific statement from the American Heart Association. *Circulation.* 2011;123:1788–1830.

11. Hockstad E, Gornick CC. Mildly symptomatic pulmonary emboli associated with electrophysiologic procedures. Indications for anticoagulant use. *Chest.* 1994;106:1908–1911.

12. Moubarak G, Bonhomme S, Vedrenne G, et al. Femoral vein thrombosis after right-sided electrophysiological procedures. *J Interv Card Electrophysiol.* 2013;38:155–158.

13. Chen JY, Chang KC, Lin YC, et al. Safety and outcomes of short-term multiple femoral venous sheath placement in cardiac electrophysiological study and radiofrequency catheter ablation. *Jpn Heart J.* 2004;45:257–264.

14. Duvuoglu V, Kervancioglu S, Dinokal H, et al. High incidence of occult femoral vein thrombosis related to multiple venous sheaths during electrophysiological studies. *Heart.* 2004;90:1061–1062.

15. Tapson VF. Acute pulmonary embolism. *N Engl J Med.* 2008;358:1037–1052.

16. Sakuragi T, Sakao Y, Furukawa K, et al. Successful management of acute pulmonary embolism after surgery for lung cancer. *Eur J Cardiothorac Surg.* 2003;24:580–587.

17. Bauer MP, Vliegen HW, Huismans MV. Massive pulmonary embolism with cardiac arrest after an intracardiac electrophysiological study: a strong case for venous thromboprophylaxis. *Blood Coagul Fibrinolysis.* 2006;17:57–58.

18. Balci K, Utku U, Asil T, et al. Deep cerebral vein thrombosis associated with iron deficiency anaemia in adults. *J Clin Neurosci.* 2007;14:181–184.

19. Stolz E1, Valdueza JM, Grebe M, et al. Anemia as a risk factor for cerebral venous thrombosis? An old hypothesis revisited. Results of a prospective study. *J Neurol.* 2007;254:729–734.

20. Kinoshita Y, Taniura S, Shishido H, et al. Cerebral venous sinus thrombosis associated with iron deficiency: two case reports. *Neurol Med Chir (Tokyo).* 2006;46:589–593.

21. Nicastro N, Schneider A, Leemann B. Iron-deficiency anemia as a rare cause of cerebral venous thrombosis and pulmonary embolism. *Case Rep Med.* 2012;2012:497814.

22. Jiménez D, Escobar C, Martí D, et al. Association of anaemia and mortality in patients with acute pulmonary embolism. *Thromb Haemost.* 2009;102:153–158.

23. Donzé J1, Labaire J, Méan M, et al. Prognostic importance of anaemia in patients with acute pulmonary embolism. *Thromb Haemost.* 2011;106:289–295.

24. Chiou T. Electrocardiography in Clinical Practice. 2nd ed. Orlando, FL: Grune Stratton; 1986:309–317.

25. Lin JF, Li YC, Yang PL. A case of massive pulmonary embolism with ST elevation in leads V1–4. *Circ J.* 2009;73:1157–1159.

26. Agnelli G, Becattini C. Acute pulmonary embolism. *N Engl J Med.* 2010;363:266–274.