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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.

These 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 via the right femoral arterial approach. 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 rst attempt at walking after RFCA. The emergent electrocardiogram in 2 of the 4 patients revealed an $S_1Q_3T_3$ pattern, 1 patient demonstrated new onset of right bundle-branch block (RBBB) and S_1Q_3 pattern and Qr pattern in V_1, and the remaining patient had negative T waves in leads V_1, 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 identied 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 the first ambulation after RFCA. Acute PE should be strongly suspected when sudden loss of consciousness occurs upon mobilization after RFCA. The new onset of $S_1Q_3T_3$ pattern, RBBB or T wave inversion in the right precordial leads, and early detection of echocardiographic right ventricular dysfunction may be useful for making an early diagnosis of acute PE after RFCA. Early ambulation after left-sided RFCA might be helpful to prevent the formation of deep venous thrombosis and subsequent PE.

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Abbreviations: AP = accessory pathways, AVNRT = atrioventricular nodal reentrant tachycardia, DVT = deep venous thrombosis, ECG = electrocardiogram, IVTs = idiopathic ventricular tachycardias, PE = pulmonary embolism, PVCs = premature ventricular complexes, RBBB = right bundle-branch block, RFCA = radiofrequency catheter ablation, RV = right ventricle.

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.[4] 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.[4] 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.

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TABLE 1. Patient Characteristics, Clinical Manifestation, Treatment, and Outcomes With Acute Massive Pulmonary Embolism After Radiofrequency Catheter Ablation

	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	411×10^9	394×10^9	226×10^9	168×10^9
Fibrinogen, g/L	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	No	Yes	Yes	No
LMWH	Yes	No	No	Yes
Vitamin K antagonist	Yes	No	No	Yes
Catheter thrombectomy	No	Yes	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,

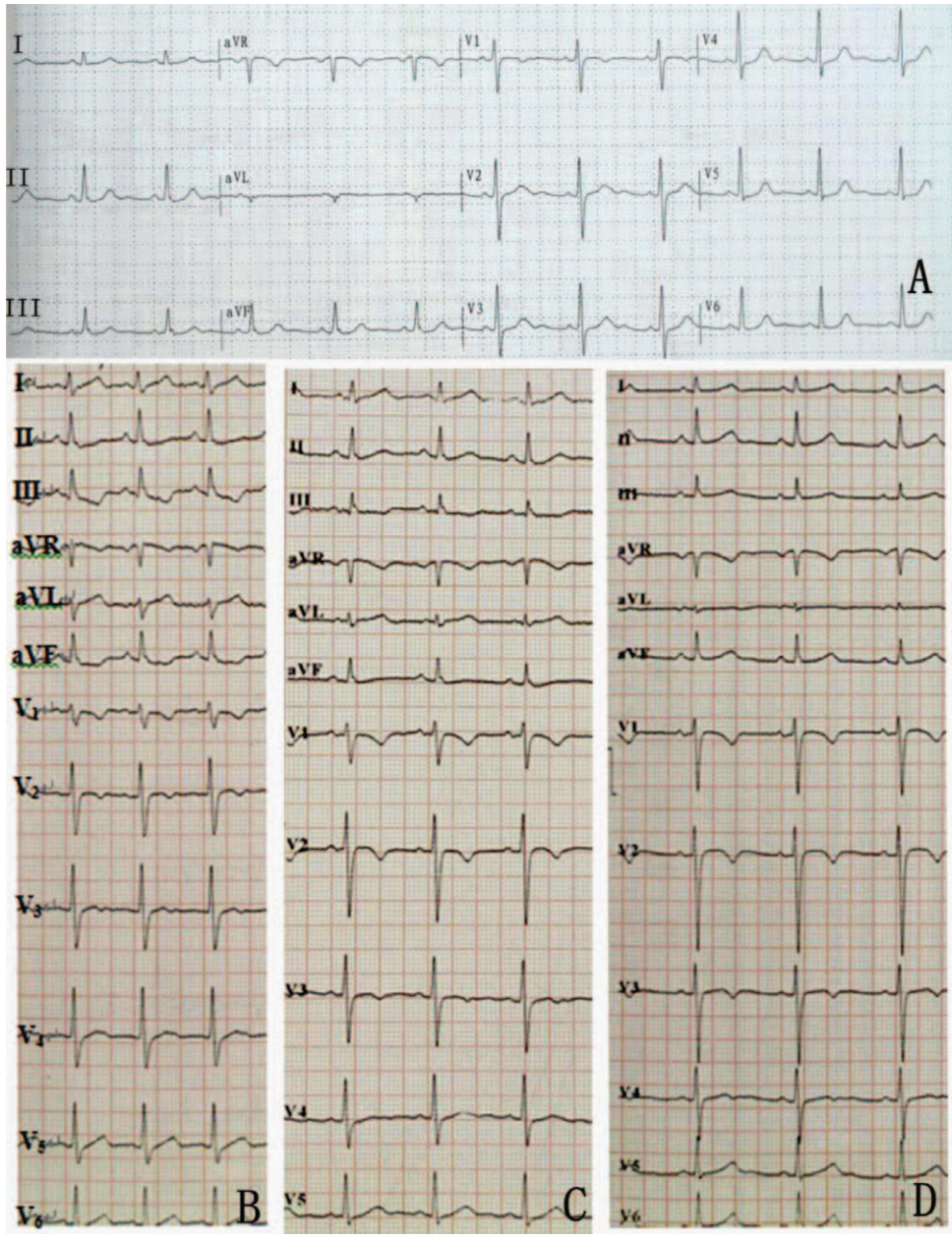

FIGURE 1. Serial ECGs in case 1. (A) ECG after RFCA. (B) ECG performed after onset of acute pulmonary embolism demonstrating T wave inversion in leads III, V1, and V2 and an $S_1Q_3T_3$ pattern. (C) ECG performed 2 days after onset of acute pulmonary embolism showing a resolution of the T wave abnormalities in lead III. (D) ECG performed 5 days after onset of acute pulmonary embolism showing no $S_1Q_3T_3$ 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.[10] 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 5.03×10^{12} cells/L and 4.54×10^{12} cells/L (normal range: $3.5–5.0 \times 10^{12}$), 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 corpsular 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 411×10^9 and 394×10^9 cells/L (normal range: $100–300 \times 10^9$), 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

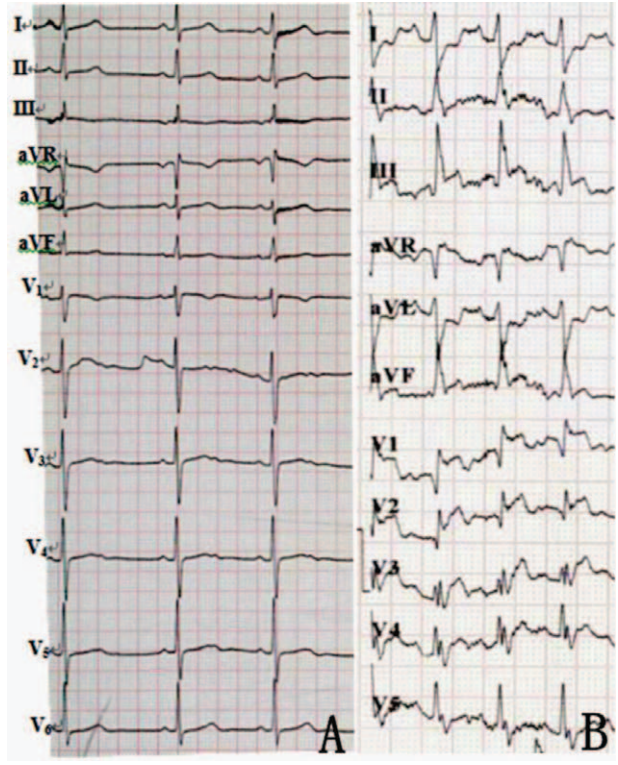

FIGURE 2. ECGs in case 2. (A) ECG after RFCA. (B) ECG performed after onset of acute pulmonary embolism demonstrating a sinus tachycardia with new onset of right bundle branch block, q wave and slight ST elevations in leads III, V1, and V2, S waves in leads I and V5–V6.

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 $S_1Q_3T_3$ pattern (Figures 1 and 3), and in the case 2 demonstrated new onset of right bundle-branch block (RBBB) and a S_1Q_3 pattern and Qr pattern in V_1 (Fig. 2), and in the case 4 demonstrated negative T waves in leads V_1, V_2, 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

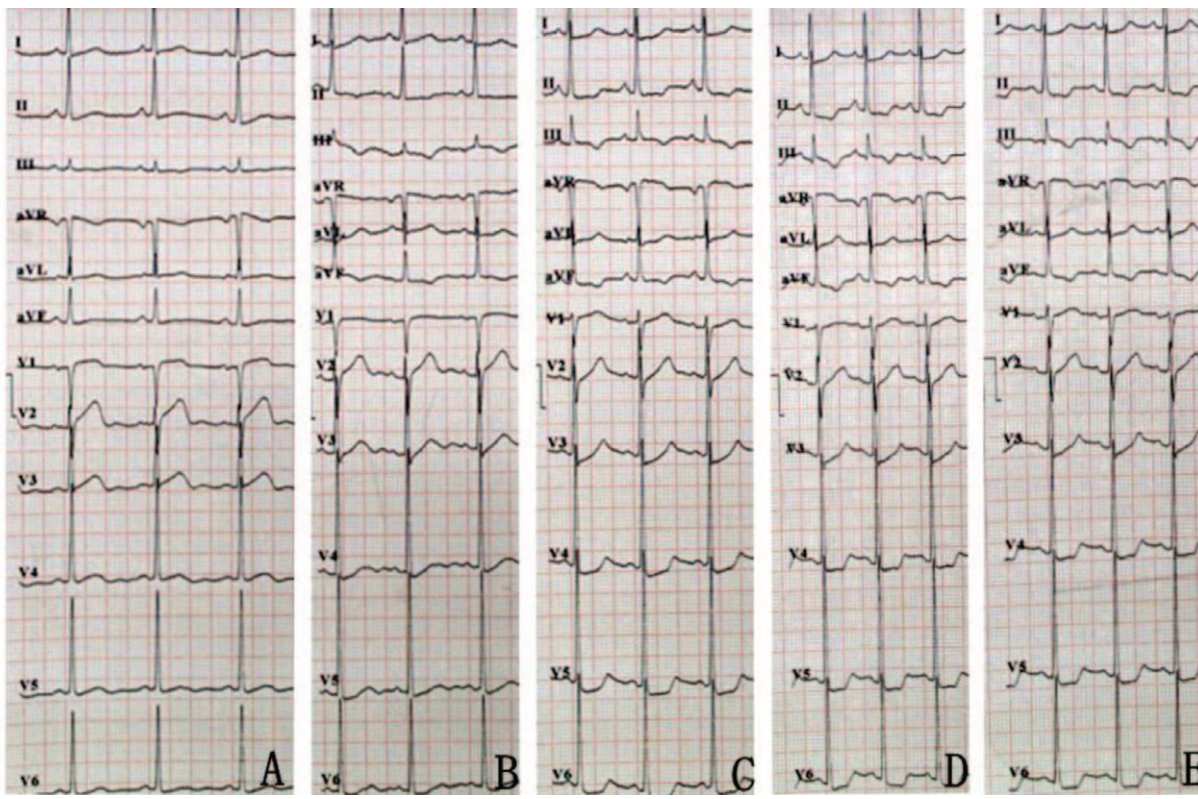

FIGURE 3. Serial ECGs in case 3. (A) ECG after RFCA. (B) ECG performed after onset of acute pulmonary embolism showing T wave inversion in leads III and aVF with ST depressions in leads V3–V6. (C) ECG performed 30 minutes after onset of acute pulmonary embolism demonstrating progressive T wave inversion in leads II, III, and aVF with prominent ST depressions in leads V3–V6. (D) ECG performed 1 hour after onset of acute pulmonary embolism showing an $S_1Q_3T_3$ pattern with prominent ST depressions in leads V3–V6. (E) ECG performed 2 hours after onset of acute pulmonary embolism.

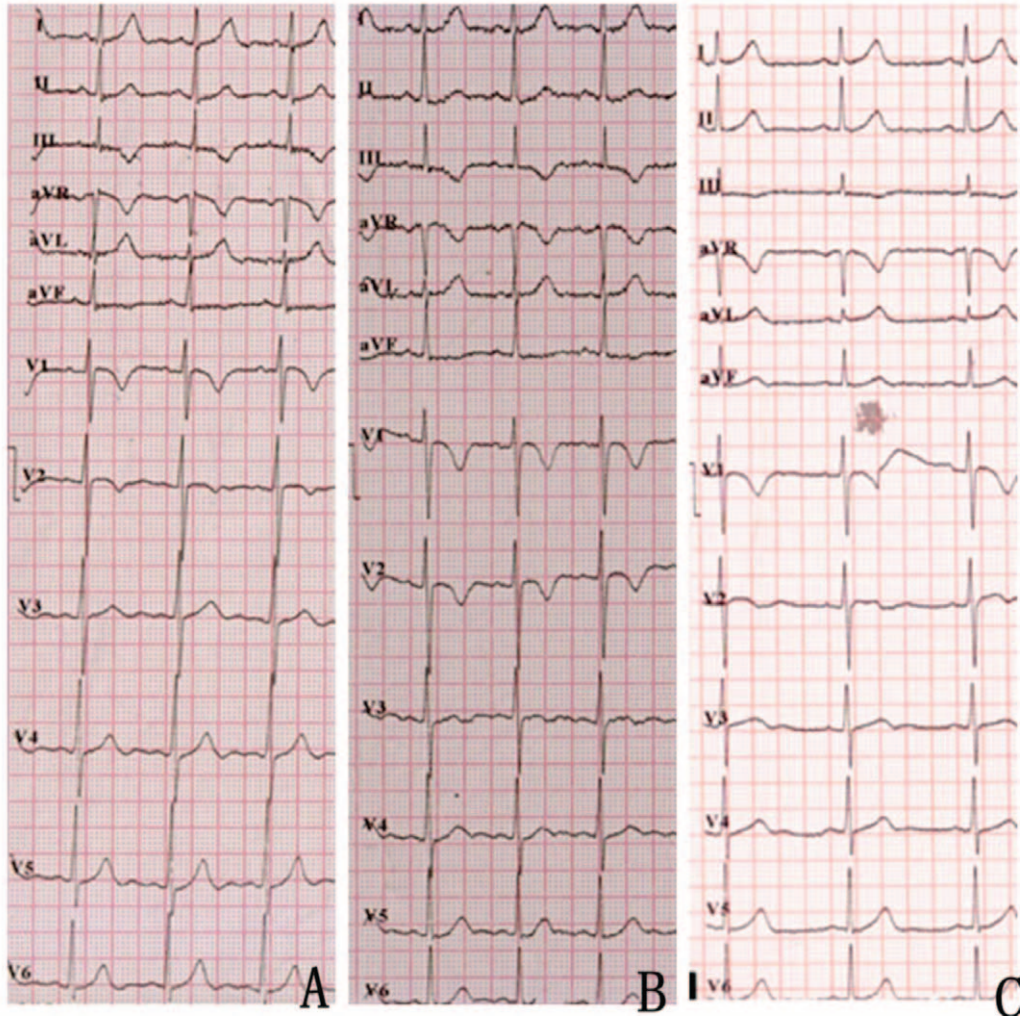

FIGURE 4. Serial ECGs in case 4. (A) ECG performed after onset of acute pulmonary embolism demonstrating T wave inversion in leads III, V1, and V2. (B) ECG performed 2 hours after onset of acute pulmonary embolism demonstrating progressive T wave inversion in leads III, V1, and V2. (C) ECG performed 12 days after onset of acute pulmonary embolism demonstrating a resolution of the T wave abnormalities in leads III, V1, and V2.

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,[1–6] but it may be underestimated and life-threatening.[4] Asymptomatic or mild PE is likely much more common than symptomatic PE, complicating electrophysiologic procedures.[11] 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.[12–14] However, none of these patients with DVT experienced symptomatic PE.[12–14] 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.[15]

An important nding was that the rst 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 rst 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.[16] 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[7] leading to

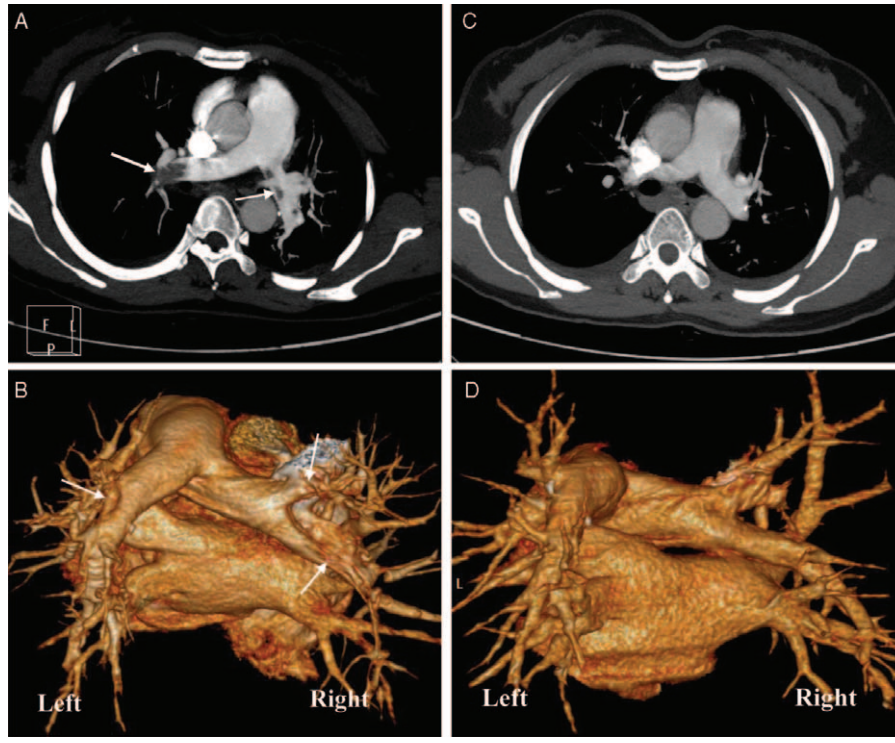

FIGURE 5. CT pulmonary angiography (CTPA) in case 1. CTPA with 2-dimensional imaging (A) and 3-dimensional imaging (B) performed after acute pulmonary embolism onset demonstrating thrombi in bilateral main pulmonary artery (arrow) that were more prominent on the right side. Repeated CTPA with a 2-dimensional 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.

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 women (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 studies[22,23] 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 $S_1Q_3T_3$ pattern, and case 2 demonstrated new onset of RBBB and S_1Q_3 pattern and Qr pattern in V_1, and case 4 demonstrated negative T waves in leads V_1, V_2, and III. Chou[24] suggested that typical ECG findings in PE are as follows: an S_1Q_3 or $S_1Q_3T_3$ pattern; a rightward shift of the QRS axis; transient, incomplete or complete RBBB; and T wave inversion in the right precordial leads. The $S_1Q_3T_3$ 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 $S_1Q_3T_3$ 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

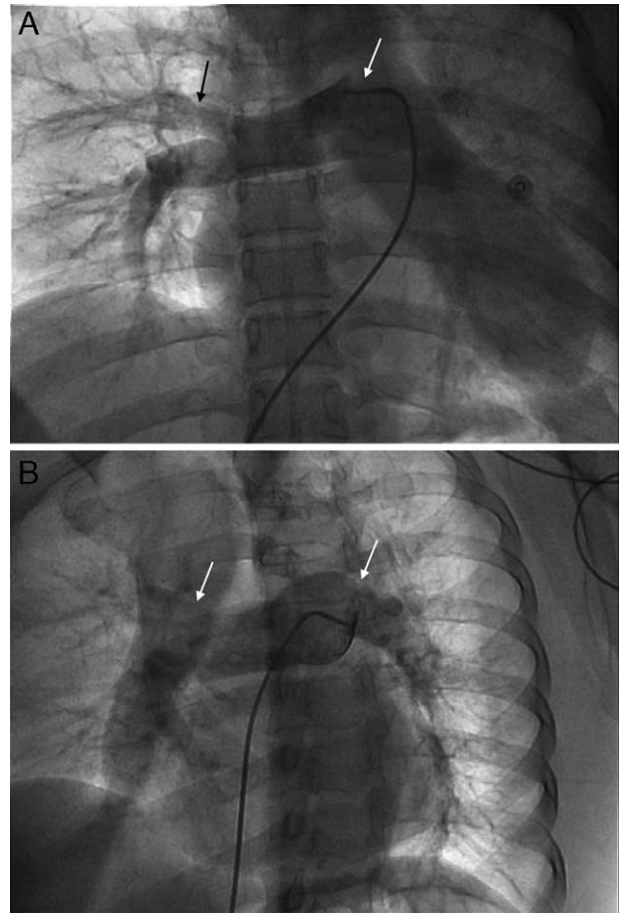

FIGURE 6. Pulmonary angiography in case 2. Pulmonary angiography in a RAO projection (A) and a LAO projection (B) demonstrating thrombi in the upper segments of bilateral lobar pulmonary artery (arrow).

PE after RFCA. In the report, the 4 patients were finally identied 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.[15] 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.[26]

Although acute massive PE after RFCA was identied 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

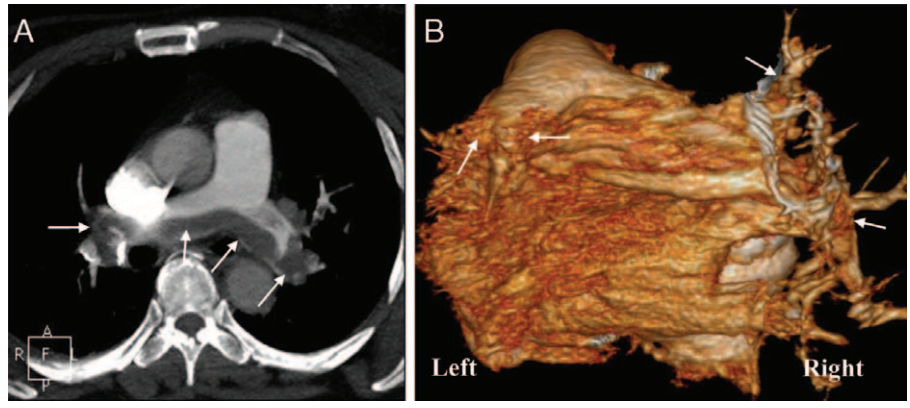

FIGURE 7. CT pulmonary angiography (CTPA) in case 3. CTPA with 2-dimensional imaging (A) and 3-dimensional imaging (B) performed 2 hours after onset of acute pulmonary embolism demonstrating thrombi in the bilateral main pulmonary artery (arrow).

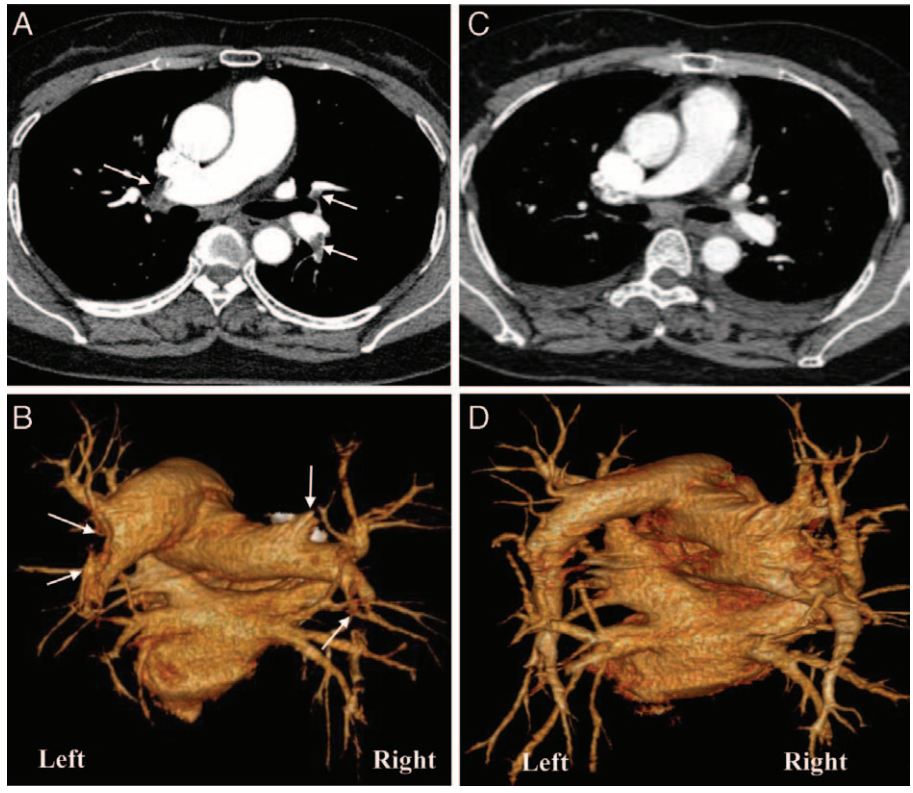

FIGURE 8. CT pulmonary angiography (CTPA) in case 4. CTPA with 2-dimensional imaging (A) and a 3-dimensional imaging (B) performed 2 hours after onset of acute pulmonary embolism demonstrating bilateral thrombi in the main pulmonary artery (arrow). Repeated CTPA with a 2-dimensional imaging (C) and a 3-dimensional imaging (D) performed 12 days after onset of acute pulmonary embolism demonstrating a significantly improved filling defect within either pulmonary artery.

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 $S_1Q_3T_3$ 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.

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