Uncommon Electrocardiographic Changes Corresponding to Symptoms during Recurrent Pulmonary Embolism as Documented by Computed Tomography Scans

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Summary: Electrocardiographic (ECG) findings of pulmonary embolism (PE) include $S_1Q_3T_3$ pattern, right bundle-branch block, right-axis deviation, and T-wave inversion in medial precordial leads. We report other uncommon ECG changes associated with various symptoms during recurrent PE as documented by computed tomography (CT) scans in a single patient. An 83-year-old woman was admitted with PE secondary to deep venous thrombosis in the left leg. During episodes of chest pain, ECG showed QTc prolongation (480 ms) with new T-wave inversion in leads III, aVF, and V_1 – V_3 , and ST-segment depression in leads V_5 – V_6 . Despite adequate anticoagulant therapy, recurrent episodes of PE occurred in the hospital. When the patient experienced sudden chest tightness, ECG showed a new S-wave notch in lead V_1 and clockwise rotation with sinus tachycardia. She also experienced transient syncope with hypotension. At this time, ECG showed transient atrioventricular junctional rhythm followed by sinus arrest, and CT scan showed a new massive embolus in the main pulmonary trunk with right ventricular dilatation, as demonstrated by echocardiography. The mechanism responsible for QTc prolongation with ST-T changes, the S-wave notch in lead V_1 with clockwise rotation, or atrioventricular junctional rhythm with sinus arrest during PE may be associated with myocardial ischemia, acute right ventricular overload, or vagal reflex, respectively.

Key words: pulmonary embolism, ST-T changes, QTc prolongation, clockwise rotation, S-wave notch in lead V_1 , atrioventricular junctional rhythm

Introduction

Acute pulmonary embolism (PE) is one of the causes of sudden death along with acute myocardial infarction, hypertrophic cardiomyopathy, myocarditis, and ventricular fibrillation. The diagnosis of PE depends primarily on a high degree of clinical suspicion because of the nonspecific presenting features. Electrocardiographic (ECG) changes in PE have been extensively documented.^{1–4} Typical findings include acute S_1Q_3 or $S_1Q_3T_3$ pattern, incomplete or complete right bundle-branch block, right-axis deviation, clockwise rotation, and T-wave inversion in medial precordial leads.⁵ Although ECG abnormalities in acute PE are often considered of minor importance in the diagnostic approach, the combination of ECG findings with clinical signs can increase the diagnostic sensitivity.³ We report uncommon ECG changes associated with various symptoms during recurrent PE as documented by computed tomography (CT) scans in a single patient, and discuss the underlying mechanisms responsible for these ECG changes.

Case Report

An 83-year-old Japanese woman with a history of hypertension was admitted to our hospital with chest pain. The same symptom recurred twice within a 12-h period. Physical examination revealed tachycardia (100/min), tachypnea (24/min), cyanosis (oxygen saturation 70%, room air), and signs of acute right ventricular (RV) failure—distended neck vein and right-sided S_3 gallop augmented with inspiration. Systolic blood pressure was 50 mmHg. Electrocardiogram showed QTc prolongation (480 ms), T-wave inversion in

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FIG. 1 Electrocardiogram recorded on admission, showing QTc prolongation (480 ms), T-wave inversion in leads III, aVF, and V₁–V₃, and ST-segment depression in leads V₅–V₆.

leads III, aVF, and V₁–V₃, and ST-segment depression in leads V₅–V₆ (Fig. 1), which were absent on an ECG taken 2 months earlier. Chest x-ray revealed cardiomegaly and oligemic lung field (Westermarck's sign⁶). Hemodynamics were measured with a Swan-Ganz catheter. Pulmonary artery pressure was 28/16 mmHg, right atrial pressure was 7 mmHg, and cardiac index was 1.72 l/min/m². Intravenous dopamine and oxygen therapy were started. Computed tomography scan detected some emboli in the proximal right and left pulmonary artery (Fig. 2A). Ventilation/perfusion lung scintigraphy scans indicated the presence of multiple large segmental perfusion defects that ventilated normally, the so-called ventilation-perfusion mismatch. Cardiac enzymes remained normal, and activated partial thromboplastin time (APTT) was 32.9 s. An initial dose of 80 U/kg heparin was administered intravenously, followed by 18 U/kg/h for 10 days to maintain APTT between 50 to 70 s. Oral warfarin was administered concomitantly with heparin at a dose of 5 mg/day for 4 days, followed by 3 to 2 mg/day to maintain an international normalized ratio between 2.0 and 3.0. Hemodynamics and blood gas values improved (blood pressure 120/80 mmHg, pulmonary artery pressure 20/8 mmHg, cardiac index 2.90

FIG. 2 (A) Some emboli (arrow) are shown by computed tomography (CT) to be attached to the left proximal pulmonary artery at the initial episode of pulmonary embolism. (B) A new massive embolus (arrow) is shown by CT to involve the main pulmonary trunk at the second episode in the hospital.

l/min/m², oxygen saturation 95%). Cardiopulmonary rehabilitation proceeded well. However, on Day 16 of her hospital stay, the patient suddenly developed chest tightness, and ECG showed sinus tachycardia (110/min), clockwise rotation, and a new S-wave notch in lead V₁ (Fig. 3). This was followed by a transient syncopal episode with systolic blood pressure of 40 mmHg. Electrocardiogram showed transient sinus arrest and atrioventricular (AV) junctional rhythm at a rate of approximately 55 beats/min (Fig. 3). Atropine sulfate was administered. After the rhythm returned to normal, the patient regained consciousness and her blood pressure increased to 110/70 mmHg. Simultaneous echocardiography showed RV dilatation (RV endodiastolic diameter 40 mm, M-mode) with global hypokinesis, and CT showed a new massive embolus in the main pulmonary trunk (Fig. 2B). Eventually she underwent angiography, which showed that PE originated from left leg deep venous thrombosis. Because she was at risk for recurrent and possibly fatal PE with oral warfarin alone, a Greenfield filter (Boston Scientific Co., Boston, Mass.) was placed in the inferior vena cava percutaneously via the right femoral vein. During the 3 years after the operation, the patient has experienced no recurrent PE.

FIG. 3 All electrocardiograms were recorded on Day 16 of the patient's hospital stay. The patient developed chest tightness at 9:55 A.M., soon falling into a transient syncope with bradycardia. After atropine injection, she regained consciousness and heart rate returned. The 9:55 A.M. tracing reveals sinus tachycardia (100 beats/min), clockwise rotation, and a new notch in the S wave (the rS duration 80 ms) in lead V₁. The 10:00 A.M. tracing reveals atrioventricular junctional rhythm at a rate of approximately 55 beats/min. The 10:09 A.M. tracing again reveals sinus tachycardia (120 beats/min).

Discussion

The ECG is helpful in evaluating patients who have had massive PE complicated by acute cor pulmonale. Because one or more of the traditional manifestations of acute cor pulmonale, that is, S₁Q₃T₃ pattern, right bundle-branch block, or right-axis deviation, occur only in 26% of patients with arteriographically proven acute massive or submassive PE, one cannot rely exclusively on these ECG abnormalities for the diagnosis of PE.² The most common ECG abnormalities were sinus tachycardia and nonspecific ST-T changes.² To our knowledge, the ECG changes observed with various symptoms in this patient during recurrent PE are rare.

Possible mechanisms underlying ECG abnormalities observed during PE attacks may involve the following. The sudden rise in pulmonary artery pressure due to anatomical obstruction, neurohumoral activation, or stretching of pulmonary artery mechanoreceptors leads to RV dilatation and eventually dysfunction.⁷ Right ventricular dysfunction leads to decrease in RV cardiac output with reduced left ventricular (LV) preload. Right ventricular dilatation and decreased LV preload lead to a shift of the intraventricular septum toward

the LV. With underfilling of LV, systemic output and pressure decrease, potentially compromising coronary perfusion and producing myocardial ischemia.⁷ ST-T changes and QTc prolongation during chest pain on admission may be associated with myocardial ischemia.^{1,5,8,9} Acute myocardial ischemia may induce an initial, transient QTc shortening followed by extreme QTc prolongation.⁸ Changes in the QTc interval often accompany T-wave inversion.⁹ The QTc interval usually returns to its normal value within 72 h of acute ischemic event, whereas inverted T waves may persist.⁸ In our patient, the QTc interval also returned to normal value 3 days after admission. It is also possible that QTc prolongation may be related to proteins, released during the thromboembolic process, that depress myocardial cell function delaying repolarization. Clockwise rotation and the S-wave notch in lead V₁ during chest tightness that were observed on Day 16 of the hospital stay may be associated with acute RV overload in the setting of recurrent PE.^{4,10} The S-wave notch in lead V₁ is thought to be identical with an rSr' pattern in lead V₁ that is occasionally noted in acute PE.¹⁰ Right ventricular conduction defect induced by pressure and volume overload is probably the responsible mechanism.¹⁰ Sinus tachycardia may be caused by

increased sympathetic tone from stimulation of pulmonary artery mechanoreceptors, decreased LV filling, and chest pain.¹¹ Atrioventricular junctional rhythm with sinus arrest during syncope may be secondary to a vasovagal reflex. Akinboboye *et al.*¹¹ and Simpson *et al.*¹² reported that AV block or marked sinus bradycardia during syncope may be caused by the vagal reflex in patients with recurrent PE as documented at autopsy. The increase in sympathetic tone leads to heightened cardiac inotropy and stimulation of cardiac mechanoreceptors and C-fibers. Signals then travel to the brainstem where the autonomic nervous system is modulated to produce either a predominantly cardioinhibitory or vasodepressor response.¹¹ A parasympathetic reflex would tend to cause sinus slowing, AV conduction disturbance, and hypotension.¹² Jaeger *et al.*¹³ reported that AV junctional rhythm was observed in 23% of the 31 patients who developed vasovagal syncope during head-up tilt-table test. In addition, the response to atropine suggests that the arrhythmia may be caused by the vagal reflex.

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