CASE REPORT

Correlation Between ST-Segment Elevation and Negative T Waves in the Precordial Leads in Acute Pulmonary Embolism: Insights into Serial Electrocardiogram Changes

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Background: Acute pulmonary embolism (APE) is often misdiagnosed as acute coronary syndrome because of the similarity of the presenting symptoms and of the electrocardiogram (ECG) manifestations. In APE, ST-segment elevation (STE) in leads V₁ to V₃/V₄, mimicking anteroseptal myocardial infarction, is not a rare phenomenon. Negative T waves (NTW) in the precordial leads mimicking the “Wellens’ syndrome” is an important ECG manifestation of APE. The evolution of these ECG changes—STE and NTW—in APE has not been thoroughly studied.

Methods: We present two patient cases with APE and their evolving serial ECGs to analyze the correlation between STE and NTW.

Results: NTW developed later than STE in these two patient cases.

Conclusions: NTW might represent a “postischemic” ECG pattern indicating a previous stage with transmural myocardial ischemia.

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acute pulmonary embolism; electrocardiogram; myocardial ischemia; right ventricular dysfunction

INTRODUCTION

Acute pulmonary embolism (APE) continues to be associated with significant morbidity and mortality. Prompt and proper diagnosis and therapy is of paramount significance to reduce mortality. APE is often misdiagnosed as acute coronary syndrome because of the similarity of the presenting symptoms and of the electrocardiogram (ECG) manifestations.¹ Growing evidence indicates that ST-segment elevation (STE) in leads V₁–V₃/V₄ mimicking anteroseptal myocardial infarction is

not a rare phenomenon in patients with APE.^{2–10} Negative T waves (NTW) in the precordial leads mimicking the “Wellens’ syndrome” is an important ECG manifestation of APE.¹¹ To be able to improve the ECG diagnosis of APE, it is important to explore the pathogenesis of these ECG manifestations.

NTW in the precordial leads were associated with right ventricular dysfunction (RVD)^{12,13} and right ventricular strain (RVS).¹⁴ Precordial NTW were superior to biomarkers of myocardial injury and B-type natriuretic peptide (BNP) for the early

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detection of RVD and was a reliable finding to identify RVD in APE.¹² Improvements in RVD can be predicted by the normalization of the precordial NTW. All these facts seem to suggest that precordial NTW is an important ECG sign of RVD or RVS. The evolution of these ECG changes—STE and NTW—in APE has not been thoroughly studied. Here, we present two patient cases with APE, who had repeated ECGs to analyze the correlation between STE and NTW in APE.

CASE PRESENTATIONS

Case 1

A 63-year-old female was admitted to Shiyan Taihe hospital due to progressive dyspnea, tightness in the middle chest and repetitive syncope for 5 days. She had no history of cardiopulmonary disease. ECG on admission showed sinus tachycardia, SI-SII-SIII pattern, qs pattern in V₃R–V₅R and V₁, the transitional zone in the precordial leads was dislocated to the left, beyond lead V₅ (clockwise rotation around the longitudinal axis), minor STE in leads aVR, V₁–V₃, and V₃R–V₅R, and NTW waves in leads III, V₃R–V₅R, V₁–V₄ (Fig. 1A). The blood pressure (BP) was 80/50 mmHg. Troponin I was 200 ng/mL (normal value <50 ng/mL) and also the NT-proBNP level was elevated: 2778 pg/mL (normal value <300 pg/mL). STE myocardial infarction was suspected by the consultant doctor and emergency coronary artery angiogram was performed. It showed only minor atherosclerosis in the right coronary artery and distal left anterior descending coronary artery. The conus branch of the right coronary artery was normal. Dopamine was initiated to maintain BP and tissue perfusion and the patient was given aspirin, clopidogrel, and low-molecular-weight heparin. A transthoracic echocardiogram revealed RV dilatation, free wall hypokinesia, D-shaped left ventricle (LV), and severe tricuspid regurgitation (Figs. 2A–D). Hence, APE was highly suspected and computed tomography (CT) pulmonary artery angiography confirmed bilateral pulmonary arterial thrombus. Subsequently, the patient suffered from sweating, profound dyspnea and lost consciousness; the BP was 30/20 mmHg. A repeated ECG showed sinus tachycardia, obvious STE in leads III, aVR, V₁–V₃, and V₃R–V₅R, and emerging ST-segment depression (STD) in leads I, II, aVL, and V₅–V₆

with pseudo-normalization of the NTW in leads V₃R–V₅R, V₁–V₄ (Fig. 1B). The patient received cardiopulmonary resuscitation to maintain vital signs and simultaneous infusion of 100 mg tissue plasminogen activator (tPA) over 2 hours. The patient's general condition improved and she was hemodynamically stable 1 hour after the tPA infusion. The ECG (Fig. 1C) 2 hours after the infusion (BP 100/70 mmHg) showed reappearance and, compared to the ECG at admission, deepening of the NTW in the leads V₁–V₅ without ST deviation in any lead. The patient remained stable. Four days after admission, the ECG (Fig. 1D) showed NTW in leads II, III, aVF, V₁–V₅, and V₃R–V₅R. A repeat transthoracic echocardiogram showed normal RV and LV morphology and function (Figs. 2E and F). The ECG 1 month later showed disappearance of NTW in leads II, III, aVF, V₂–V₅.

Case 2

A 48-year-old female was admitted to the same hospital with new onset of dyspnea, tachycardia, dizziness and diaphoresis in relation to activity. Her medical history revealed antihypertensive medication for 3 years, but no dyspnea or chest discomfort. A previous ECG 1 month before admission, when the patient was free of symptoms, was normal, except for leftward deviation of the frontal plane QRS axis (Fig. 3A). On admission the heart rate was 118 beats/min, the respiratory rate 25/min, and the BP 90/60 mmHg. The ECG on admission showed the SI-QIII pattern, minor STD in leads I, II, aVF, V₄–V₆ and minor STE in leads aVR and V₁ (Fig. 3B). She was diagnosed with acute coronary syndrome and received aspirin, clopidogrel, and low-molecular-weight heparin. Coronary angiogram was performed and showed only minor atherosclerosis in the right coronary artery. The conus branch of the right coronary artery was normal. One day later she deteriorated with pallor, sweating, and profound dyspnea and the BP dropped to 80/40 mmHg. The ECG showed deepening of the STD in leads I, II, aVF, V₄–V₆ and increased STE in the leads aVR, V₁ and V₂ (Fig. 3C). Emergent transthoracic echocardiogram revealed RV dilatation, free wall hypokinesia, and severe tricuspid regurgitation (Figs. 4A and B). APE was highly suspected and the CT pulmonary angiography confirmed bilateral pulmonary artery embolism. The patient was given urokinase infusion over 2 hours and was hemodynamically

Figure 1. (A) The ECG on admission showed sinus tachycardia, SI-SII-SIII pattern, qs pattern in V_3R , V_4R , V_5R and V_1 , transitional zone in precordial leads is dislocated to left, minor STE in leads aVR, V_1 - V_3 , and V_3R - V_5R , and NTW waves in leads III, V_3R - V_5R , V_1 - V_4 . (B) The ECG during cardiogenic shock showed sinus tachycardia, obvious STE in leads III, aVR, V_1 - V_3 , and V_3R - V_5R , newly emerging ST-segment depression in leads I, II, aVL and V_5 - V_6 , and pseudo-normalization of NTW in leads V_3R - V_5R , V_1 - V_4 . (C) The ECG after thrombolytic therapy showed reappearance of NTW in leads V_1 - V_5 , deeper than that at admission, without ST deviation in any lead. (D) The ECG 4 days later showed NTW in leads II, III, aVF, V_1 - V_5 , and V_3R - V_5R . (E) The ECG 1 month later showed disappearance of NTW in leads II, III, aVF, V_2 - V_5 .

stable after thrombolytic therapy. A repeated ECG after successful thrombolytic therapy, when the BP recovered to 110/60 mmHg, showed NTW in leads V_1 - V_4 (Fig. 3D). The rest of the hospital stay was uneventful. Eight days after admission, the ECG showed NTW in leads III and V_1 to V_2 (Fig. 3E). Repeated transthoracic echocardiogram showed mild tricuspid regurgitation without other abnormalities (Figs. 4C and D).

DISCUSSION

In submassive embolism 23% of the patients have a normal ECG. According to a previous study the traditional manifestations of APE—SI-QIII-TIII, RBBB, P-pulmonale, or right axis deviation—occurred in only 26% of cases. The most common

ECG features were nonspecific T-wave changes (42%) and nonspecific abnormalities (elevation or depression) of the ST segment (41%). Left axis deviation occurred in 7% and was as frequent as right axis deviation. Low voltage QRS complexes were noted in 6%. Atrial fibrillation or atrial flutter typically developed in patients with APE, who had preexistent cardiac disease. All of the varieties of ECG abnormalities disappeared in some of the patients by 2 weeks. Inversion of the T wave was the most persistent abnormality.¹⁵

A few case reports describe ECG presentation of APE as STE in the precordial leads, but the mechanism is not clear. STE in leads V_1 - V_3 / V_4 , mimicking anteroseptal myocardial infarction, is not a rare phenomenon in the presenting ECG of patients with APE. Some patients have accompanying STE in lead III and/or aVF and II.^{4,8,9} The

Figure 2. (A) and (B) Color Doppler examination showed severe tricuspid regurgitation and 83 mmHg of the regurgitation pressure gradient. (C) The apical 4-chamber view at the apex of the heart revealed enlarged right ventricle, which was greater than the left ventricle. (D) Parasternal short-axis view at end diastole showed interventricular septal flattening and thus formed the D-shaped left ventricle. (E) The apical 4-chamber view at the apex of the heart after treatment revealed normal right ventricle and left ventricle morphology. (F) Parasternal short-axis view at end diastole after treatment showed disappearance of the D-shaped left ventricle.

right-sided accessory precordial leads V_3R – V_5R may also show STE, like in our first case and the case reported by Ozer et al.⁸ Moreover, STE in leads V_1 and/or III are common ECG presentations.¹⁶ Lead III faces the inferior region of the RV and lead V_1 faces the anterior region of the RV.¹¹ We have also described that the right accessory lead V_3R showed STE in 3 patients with hemodynamic instability during APE, even without STE in lead V_1 .¹⁷ Transmural acute RV myocardial infarction secondary to massive pulmonary embolism has been proven by autopsy.^{18,19} All these facts suggest that STE in these leads are the results of transmural ischemia in RV. Apparently, there

are two main possible explanations for the STE in APE. In the presence of APE with a proven atrial septal defect or patent foramen ovale with a right to left shunt, paradoxical embolism to the conus coronary artery is an obvious explanation for ST segment elevation from V_1 to V_4 . Goslar et al.⁶ reported a case of APE presenting with STE in the precordial leads V_1 – V_4 , where there was paradoxical embolism into the conus artery. However, this explanation has limitations. First, there is no reason for a paradoxical embolus to specifically occlude the conus artery. On the contrary, there should be variation in the distribution of the STE, if the general mechanism

Figure 3. (A) A previous ECG showed left-axis deviation of the frontal plane QRS axis, but was otherwise normal. (B) The ECG on admission showed SI-QIII pattern, minor STD in leads I, II, aVF, V₄-V₆ and minor STE in leads aVR and V₁. (C) The ECG during cardiogenic shock showed deeper STD in leads I, II, aVF, V₄-V₆ and higher STE in the leads aVR, V₁ and V₂. (D) The ECG after successful thrombolytic therapy showed NTW in leads V₁-V₄ without ST-segment deviation. (E) The ECG 8 days later showed disappearance of SI-QIII and NTW in lead V₄.

would be paradoxical embolism. In APE, STE is uniformly confined to the leads adjacent to the RV, such as leads V₁, III, and/or V₂-V₄. When the RV is dilated, the leads V₂-V₃ and even V₄ will be adjacent to the RV. Second, patency of the coronary arteries was confirmed in part of the patients showing STE in these leads. In our cases, coronary angiography showed only slight atherosclerosis without embolism. Therefore, STE resulting from paradoxical coronary artery embolism most probably accounts for a very small part of the patients. Another, seemingly more relevant explanation for STE, is transmural myocardial ischemia of the RV.^{3,4,17} Hypotension, hypoxemia, RV strain, and catecholamine surge in APE can cause subendocardial ischemia of the LV and transmural ischemia of the RV.^{9,17} We speculate that the STE in leads V₁ or V₁-V₃/V₄ is the consequence of transmural ischemia mainly resulting from RVS, hypoxemia and/or hypotension associated with APE.

Ischemic ECG patterns of APE are dependent on the following factors: the degree and the ratio of the RV transmural ischemia and the LV subendocardial ischemia, the modification of the ratio of the RV/LV diameter, modifications of the positional orientation of the heart in the chest (cardiac clockwise rotation around the longitudinal axis), the marked localized dilatation of the right ventricular outflow tract (RVOT), basal infundibular region, or crista supraventricularis, acute coronary insufficiency secondary to a fall in cardiac output, and the coexistence of cardiopulmonary disease. Probably, also other factors are involved. If the RV transmural ischemia is dominant, the ECG will present STE in the precordial leads mimicking anteroseptal myocardial infarction (as in Figs. 1A and B). If the LV subendocardial ischemia is dominant, the ECG will present STE in leads aVR and ST-segment depression in leads I and V₄-V₆^{17,20} (as in Fig. 3B), which in acute coronary syndrome has been defined as circumferential

Figure 4. (A) The apical 4-chamber view at the apex of the heart revealed enlarged right ventricle, which was greater than the left ventricle. (B) Color Doppler examination showed severe tricuspid regurgitation and 35 mmHg of the regurgitation pressure gradient. (C) The apical 4-chamber view at the apex of the heart after treatment revealed normal right ventricle and left ventricle morphology. (D) Color Doppler examination showed mild tricuspid regurgitation and 25 mmHg of the regurgitation pressure gradient.

subendocardial ischemia, and is associated with left main coronary artery occlusion or multivessel coronary artery disease.^{21,22} If RV transmural ischemia and LV subendocardial ischemia are codominant, the ECG will present STE in leads aVR, V₁/V₂, and/or III, and ST-segment depression in leads I and V₄/V₅-V₆^{16,23,24} (as in Fig. 3C).

In acute coronary syndrome, negative T waves are "postischemic" ECG changes typically following STE caused by transmural ischemia in the corresponding leads.²⁵ In takotsubo cardiomyopathy, negative T waves are also considered "postischemic" ECG changes, typically following STE in the corresponding leads, caused by transmural ischemia at the cellular level.²⁶ It is reasonable to speculate that also NTW in the precordial leads are "postischemic" ECG changes and typically secondary to transmural ischemia in the RV. Transmural myocardial edema has been demonstrated to underlie the NTW associated with acute coronary syndrome or takotsubo cardiomyopathy.²⁷ When

the edema disappears, the NTW is normalized.²⁸ In addition, NTW in STE myocardial infarction is regarded as a sign of an open infarct-related artery with restored microcirculation.²⁹ We can speculate that transmural myocardial ischemia persisting for a period of time will cause myocardial edema after reperfusion, which will result in NTW. In case of a new episode of transmural myocardial ischemia, the T-wave negativity will decrease and pseudo-normalization and STE may develop. Therefore, NTW is possibly a "postischemic" ECG pattern and reflects former transmural myocardial ischemia. We speculate that these mechanisms also apply to patients with APE. Sreeram et al³⁰ found that NTW in the inferior leads III and aVF or in leads V₁-V₄ occurred more often in patients with symptoms for more than 7 days. This finding is consistent with our hypothesis. Regarding our Case 1, the ECG on admission showed NTW in leads V₁-V₄ when the patient was hypotensive and the transthoracic echocardiogram revealed RV dilatation, free wall

hypokinesia, D-shaped LV, and severe tricuspid regurgitation. The ECG during cardiogenic shock showed pseudo-normalization of the NTW and higher STE in leads V₁-V₃. However, the ECG 2 hours later after the thrombolytic therapy, during hemodynamic stability, showed reappearance of the NTW. Also, the T waves were deeper than that at admission, without ST deviation in any lead. Four days after admission, the ECG showed NTW in leads II, III, aVF, V₁-V₅, and V₃R-V₅R. However, the transthoracic echocardiogram showed normal RV morphology and function. Regarding our Case 2, the ECG on admission showed the SI-QIII pattern, minor STD in leads I, II, aVF, V₄-V₆ and minor STE in leads aVR and V₁. During cardiogenic shock, both the STD and the STE were accentuated and the echocardiogram revealed RV dilatation, free wall hypokinesia, and severe tricuspid regurgitation. After successful thrombolytic therapy, NTW appeared in the leads V₁-V₄ without ST-segment deviation. Eight days after admission, the ECG showed NTW in leads III and V₁-V₂. However, repeated echocardiogram showed mild tricuspid regurgitation without other abnormalities. These two cases indicate that STE in the precordial leads reflects more severe RVD or RVS than NTW, and also that NTW in the precordial leads possibly represents a "postischemic" ECG pattern following transmural myocardial ischemia. We also clearly found the persistence of NTW beyond recovery of the RVD and the deepest NTW developing later after the acute event. The discordance between NTW and RVD or RVS is an important element of novelty that disproves the traditional perspective that NTW in precordial leads in APE results only from RVD and RVS. The possible explanation is that NTW is a "postischemic" ECG pattern reflecting previous transmural myocardial ischemia, mainly resulting from severe RVS. When RVD and RVS disappear, myocardial ischemia will be resolved and NTW will develop. In clinical practice, we can find some patients with NTW with concomitant ST-segment deviation in the precordial leads (as in Fig. 1A); this presentation indicates RVD and RVS.

Although ischemic ST-segment deviation in APE can mimic acute coronary syndrome, ECG changes indicating RVS are helpful to aid in the diagnosis of APE. The ECG parameters of RVS include SI-QIII/SI-QIII-TIII,¹⁴ notched S wave in lead V₁,^{31,32} RBBB,¹⁴ or Qr sign in lead V₁.³³

CONCLUSIONS

Based on follow-up ECGs during hemodynamic changes in two patients with APE, we have described the correlation between STE and NTW in the precordial leads. We speculate that NTW in APE may represent an evolutionary "postischemic" stage following STE, which is a sign of transmural myocardial ischemia of the RV. Obviously, STE reflects more severe RVD or RVS than NTW in patients with APE.

LIMITATIONS

Our conclusion of NTW in APE representing an evolutionary "postischemic" stage following STE is speculative. Another limitation is the absence of cardiac magnetic resonance imaging to evaluate the correlation between myocardial edema and NTW in patients with APE.

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