A Review of Electrocardiography in Pulmonary Embolism: Recognizing Pulmonary Embolus Masquerading as ST-Elevation Myocardial Infarction

Kanwal Pratap Singh Raghav, MD,[1] Premraj Makkuni, MD,[2] Vincent M. Figueredo, MD[3]

[1]Department of Hematology/Oncology, MD Anderson Cancer Center, Houston, TX; [2]Department of Cardiology, Albert Einstein Medical Center, Philadelphia, PA; [3]Einstein Institute for Heart and Vascular Health, Albert Einstein Medical Center, and Jefferson Medical College, Philadelphia, PA

A 64-year-old woman with hypertension and diabetes presented with acute shortness of breath and left-sided chest discomfort. Electrocardiogram (ECG) demonstrated Q waves, coved ST-segment elevations, and T-wave inversions in leads V_1-V_4, suggesting acute anterior ST-elevation myocardial infarction (STEMI). Catheterization revealed nonocclusive coronary artery disease with elevated pulmonary and right heart pressures, confirmed by echocardiography. Ventilation perfusion scan was deemed high probability for pulmonary embolism (PE). Treatment for a submassive PE was initiated and ECG changes resolved by discharge. This case exemplifies similarities in clinical presentation of PE and acute STEMI. The presence of Q waves in anterior leads with coved ST-elevation after PE has not been described previously. We review the differential diagnosis of ST elevation and the assorted spectrum of ECG changes seen in PE.
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Key words: Acute myocardial infarction • Electrocardiography • Pulmonary embolism • ST-segment elevation • McGinn-White sign

Pulmonary embolism (PE) and acute myocardial infarction (AMI) are among the most ominous diagnoses that can be made in the emergency department (ED). Symptoms of both can be similar, comprising acute-onset dyspnea, chest pain, cough, syncope, diaphoresis, and palpitations. Physical examination is also nonspecific and cannot reliably distinguish these two entities. Clinical features have limited diagnostic value in diagnosis of PE per se.[1] This necessitates further testing for ruling in or ruling out PE. Electrocardiogram (ECG) may

be helpful, but is limited as a diagnostic tool for PE, due to its limited sensitivity and specificity.[2-4] The McGinn-White sign (S1Q3T3), classically associated with PE, is only found in approximately 30% of cases.[5] ECG abnormalities in PE that mimic AMI are unusual. Although T-wave inversion in precordial leads can be found in 40% to 60% of cases, other signs (including Q waves and ST-segment elevations) are rare.[3,6] ST elevations are highly suggestive of AMI but can be seen in a variety of other conditions, including noncardiac conditions such as pericarditis and hyperkalemia.[7] ST elevations associated with PE mimicking AMI have infrequently been reported.[8-11] We present a similar scenario followed by a review of ECG changes in PE. The unique aspect of the case is the presence of Q waves and coved ST elevations, along with T-wave inversions in precordial leads. The McGinn-White sign is also present. The importance of interpretation of ECG changes in PE and acknowledgment of non-AMI etiologies of ST elevation are discussed.

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Case Review

A 64-year-old woman presented to the ED with sudden-onset shortness of breath and wheezing, which started 5 hours prior to arrival. The shortness of breath was accompanied by left-sided nonradiating chest discomfort, palpitation, and diaphoresis. The patient had a syncopal episode lasting a few minutes, which occurred the night before symptom onset. She denied any previous history of a similar episode. She denied any fevers, recent cough or cold, back pain, lower extremity swelling, leg pains, or confusion. She had no previous history of angina-like symptoms. She denied any recent surgery, trauma, immobilization, or travel. Past medical history included hypertension, type 2 diabetes mellitus, obesity, hyperlipidemia, spinal stenosis, and mild intermittent asthma. No previous history of coronary artery disease (CAD), peripheral vascular disease, stroke, malignancy, or venous thromboembolism was reported. The family history was positive for CAD in her father (but there was no history of premature CAD-associated death). There was no family history of any thromboembolic disease, and she did not smoke or drink.

Medications included lisinopril, simvastatin, furosemide, valsartan, tramadol, diltiazem, multivitamins, tolterodine, colchicine, metformin, lansoprazole, and fexofenadine.

Upon arrival in the ED, the patient was complaining of persistent chest discomfort, shortness of breath, dizziness, and nausea. She was afebrile with a blood pressure of 101/50 mm Hg, a heart rate of 87 beats/min, a respiratory rate of 26 breaths/min, and her oxygen saturation by pulse oximetry was 90% at room air and 96% with 4-L/min oxygen via nasal canula.

Physical examination revealed a profoundly diaphoretic, morbidly obese woman in moderate respiratory distress, using accessory respiratory muscles. Her chest was clear to auscultation and percussion bilaterally. Cardiac examination revealed a regular heart rate with normal S_1 and S_2 heart sounds. Extremities revealed no clubbing, cyanosis, or edema.

Complete blood cell counts showed leucocytosis (white cell count ~ 13,000/mm^3) with relative neutrophilia (segmented neutrophils ~ 83.4%). Comprehensive metabolic panel showed hyperglycemia (blood glucose ~ 333 mg/dL) and serum creatinine (SCr ~ 1.6 mg/dL). Cardiac biosite markers showed mildly elevated myoglobin at 208 ng/mL (0-170 ng/mL), followed by a troponin I elevation of 0.44 ng/mL (0-0.04 ng/mL). Portable chest radiograph revealed no acute cardiopulmonary disease and mild cardiomegaly.

ECG (Figure 1) revealed a normal sinus rhythm at a heart rate of 84 beats/min, normal axis, and a low-voltage QRS. There were Q waves in leads V_1-V_3, ST elevations (V_3-V_4), and T-wave inversions in leads V_1-V_4, suggestive of an acute anterior wall myocardial infarction (MI). Of note, there was also an S1Q3T3 pattern consistent with a McGinn-White sign. No previous ECG was available for comparison.

After treatment with aspirin, oxygen, and nitrates, the patient was rushed to the cardiac catheterization laboratory for emergency cardiac catheterization. She did not have any significant response to nitrate therapy. Coronary angiography showed the coronary arteries to be free of any occlusive disease. A left ventriculogram showed a dynamic left ventricle with ejection fraction > 70% and no regional wall motion abnormalities. Right heart catheterization showed elevated right heart pressures (right atrial ~ 22 mm Hg, right ventricle [RV] ~ 85/15 mm Hg, pulmonary artery [PA] ~ 83/13 mm Hg, with mean pressure ~ 52 mm Hg and pulmonary capillary wedge pressure ~ 39 mm Hg).

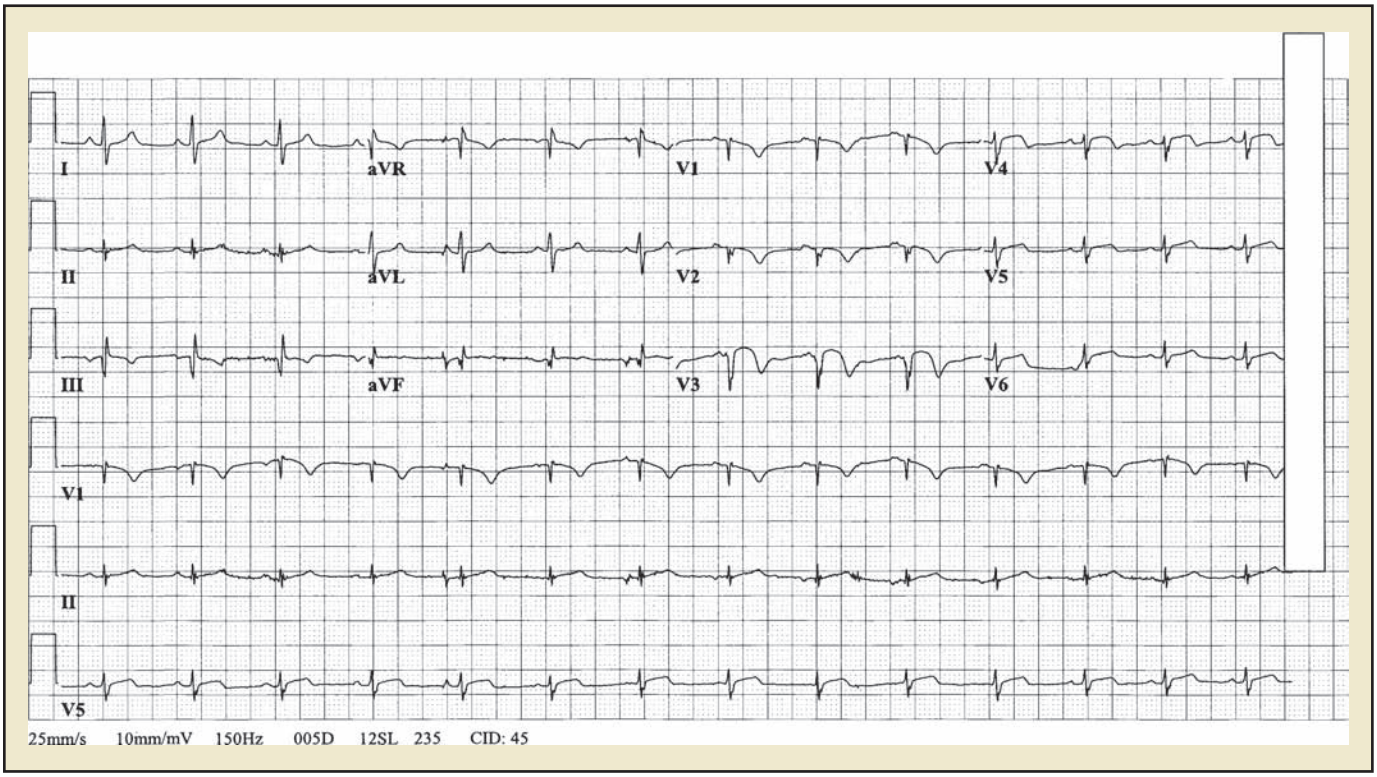
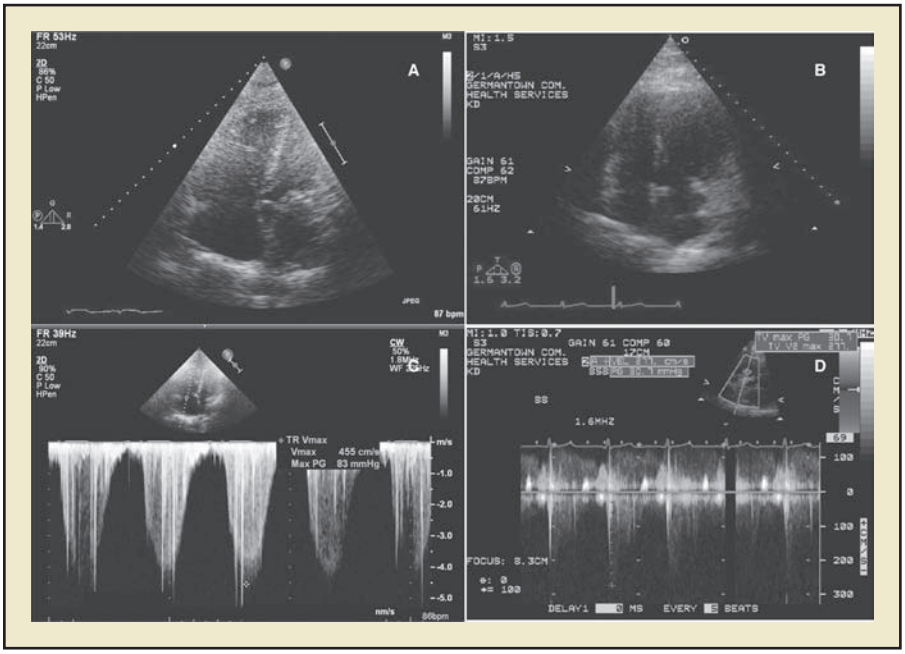


Figure 1. *Electrocardiogram at presentation.*

Following cardiac catheterization, a transthoracic echocardiogram (TTE) (Figure 2) was performed, which demonstrated severe right ventricular dilation and dysfunction, and elevated PA pressures (PA systolic pressure [PASP] ~ 95-100 mm Hg). A serpentine mass at the bifurcation of the PA, suggestive of a thrombus, was also seen.

The ventilation perfusion (V/Q) performed for the evaluation of a PE showed multiple perfusion defects and was deemed as high probability for PE. Because the patient had a low clinical probability of PE but a high-probability V/Q scan, a Doppler ultrasound of the lower extremities was performed, which showed deep venous thrombosis of right popliteal vein. The patient was heparinized and an intravenous inferior vena cava (IVC) filter was placed to prevent further pulmonary emboli. The

Figure 2. *Transthoracic echocardiogram (TTE) study.* **(A)** *TTE in four-chamber view at presentation showed a severely dilated right ventricle (RV). Severe right ventricular dysfunction was present.* **(B)** *Five months later, with appropriate treatment, the RV size has normalized. The right ventricular function had returned to normal.* **(C)** *Doppler interrogation showed severe regurgitation and severe pulmonary hypertension with a pressure gradient of 83 mm Hg across the tricuspid valve.* **(D)** *Five months later, the tricuspid regurgitation had become mild and the pulmonary pressure decreased significantly with a gradient across the tricuspid valve measuring 31 mm Hg.*



temporary IVC filter was placed in this setting of a massive PE because it was thought that another embolic event would be poorly tolerated, due to an already compromised pulmonary vascular bed. The patient recovered well, and was discharged on chronic warfarin anticoagulation. Repeat ECG performed prior to discharge showed resolution of ST elevations and Q waves and improved T-wave changes. A repeat echocardiogram 1 month later revealed a normal-sized RV, trace tricuspid regurgitation, and a PASP of 55-60 mm Hg (Figure 2).

Discussion and Review of Literature

Developing a differential diagnosis of an acute presentation and a final accurate diagnosis in the ED is largely based on clinical picture (symptoms and signs) and easily available rapid diagnostic tests. The precept is to quickly identify and treat conditions that warrant immediate treatment or may be life threatening. PE and AMI are two such diagnoses. They often present in similar ways; the situation becomes more unnerving if the diagnostics used also start mimicking one other. An acute sense of reasoning and awareness of this possibility are vital to patient outcome.

Revisiting ST Elevation: Not Just MI

The ST segment is the electrical equivalent of the period between the end of ventricular depolarization and the beginning of ventricular repolarization. ST segment is usually near isoelectric because myocardial cells attain similar potentials during early repolarization. ST segment is a low-amplitude wave, which steadily leads to the T wave. The J point, which heralds the onset of the ST segment, is usually isoelectric. A normal ST segment has a duration of 80 to 120 ms and is slightly concave upward.

Table 1
ST Elevation in Causes Other Than Acute Myocardial Infarction

Differential Diagnosis	Pattern
Normal ST-segment elevation	Usually V_1-V_4 1-3 mm: male pattern, < 1 mm female pattern ST segment concave
Early repolarization	ST elevation marked in V_4, II > III J-point notched ST segment concave Reciprocal ST depression in aVR Mild PR-segment depression
Early repolarization with persistent juvenile T wave	Young black men Coved ST elevation and TWI in midprecordial leads
Left bundle branch block	ST-T and QRS discordant ST elevation concave
Acute myopericarditis	Diffuse ST elevation with PR depression ST elevation II > III and without reciprocal depression in aVL
Hyperkalemia	ST-segment downsloping
Brugada syndrome and arrhythmogenic RV cardiomyopathy	Loss of action potential in RV epicardium only Complete/partial right bundle branch block ST downsloping and saddleback shape Usually V_1 and V_2
Pulmonary embolism	Anteroseptal leads and associated with TWI
Prinzmetal angina	Transient ST elevations only
Post direct cardioversion	Striking transient ST (often > 10 mm) elevations only

RV, right ventricular; TWI, T-wave inversion.
Data from Wang K et al.[7]

The transmural gradients determine ST patterns.[12] Normal variants with raised ST elevations are common[13] (Table 1). Ischemia or infarction leads to ST deviations by causing a disturbance in the electrical properties of myocardial cells (ischemic cells are partially depolarized in resting state as compared with normal cells), thereby generating a current of injury between ischemic and normal tissue. Hence, whenever transmural infarction occurs, the ST vector is directed toward the epicardium and the overlying lead records an ST elevation. A variety of insults other than ischemic injury to the myocardium can cause disruption of the electrical properties of cardiac muscle cell and can therefore potentially create ST elevations (Table 1, Figure 3). Table 1 and Figures 1 to 3 illustrate clues in the ECG that may differentiate one etiology from another. These clues, though helpful, are not sufficient to rule out or rule in MI, and therefore the ECG can only act as a supplement to clinical data. Nonacute MI causes of ST elevations are seen with a frequency of greater than 50% in all cases of chest pain.[14]

With regard to the differential diagnosis of ST elevations, the following pearls can be very useful:[7]

1. Normal ST elevations: these are seen in leads V_1-V_4. They are about 1-3 mm (male pattern) and

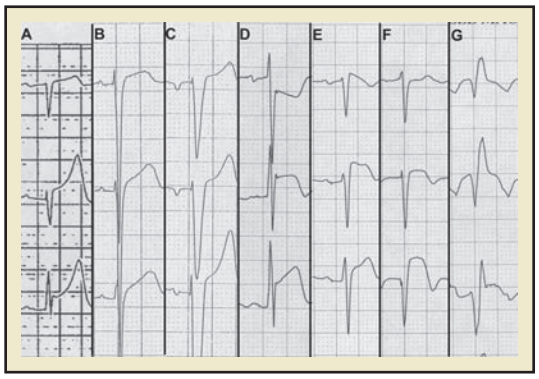


Figure 3. *Various causes of ST elevation in the early precordial leads.* **(A)** *Early repolarization.* **(B)** *Left ventricular hypertrophy repolarization abnormality.* **(C)** *Left bundle branch block.* **(D)** *Acute pericarditis.* **(E)** *Brugada pattern.* **(F)** *Acute anteroseptal myocardial infarction.* **(G)** *Right bundle branch block with acute anteroseptal myocardial infarction.*

< 1 mm (female pattern). The ST segment is concave upward.

2. Early repolarization: the ST elevations are marked in V_4, II > III and are associated with reciprocal ST depression in aVR. ST segment is concave upward. Mild PR depression and a notched J point may also be seen. Early repolarization with persistent juvenile T waves may be seen in young black men; in such cases, the ST segment is coved and T-wave inversions are seen in midprecordial leads.
3. PE: a wide variety of ECG changes can be appreciated in PE (Table 1). ST elevations occur in anteroseptal leads and are associated with T-wave inversions.
4. Acute myopericarditis: diffuse ST elevations with PR depressions are seen.
5. Brugada syndrome: ST segments are downsloping and saddle back in shape. These are seen in V_1 and V_2 and are associated with complete or partial right bundle branch block.

Hyperkalemia, Prinzmetal angina, and left bundle branch block can also have ST elevations. Enormously prominent ST elevations can also be seen in the setting of post direct cardioversion patients.

A variety of clues are available to distinguish between ST elevations of AMI and other causes (Table 1, Figure 3). Bedside echocardiography can do much not only to rule out AMI but also to rule in some of the differential diagnoses, such as submassive PE, as was the case in our patient. TTE is a sensitive tool for evaluating suspected AMI in the ED.[15] In a prospective study of 180 patients with possible AMI in the ED, Sabia and colleagues[15] demonstrated that systolic wall motion abnormalities on a two-dimensional echocardiography had a sensitivity of 93%, a specificity of 60%, and a negative predictive value of 98% for AMI in patients with chest pain. Bearing in mind the nonspecificity of ECG, its use in the ED could be encouraged. There may be some value in obtaining a right-sided ECG showing corroborative changes. Akula and associates[16] showed that 20% of patients with PE who have normal left-sided ECGs had abnormal right-sided ECGs with ST-segment elevations and a qr or qs pattern (prominent q waves) in one or more of leads V_{4R}, V_{5R}, and V_{6R}.

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PE and ECG Changes: What to Watch For

Clinical features are unreliable in the diagnosis of PE[1]; therefore, it often goes unnoticed on presentation in many patients. A high degree of clinical suspicion in combination with imaging studies is needed for diagnosing this condition. ECG is a useful initial investigation because results are normal in only approximately 29% of all cases of PE (6% massive and 23% submassive).[6] Although some ECG changes are more common in PE, the ECG alone is inadequate to decide or deny the diagnosis.[17] Despite the low sensitivity and specificity of ECG for diagnosing PE, it can still direct clinicians. ECG findings from various studies are shown in Table 2. The classic McGinn-White sign (S1Q3T3) described by McGinn and White in 1935 is seen in only one-third of cases. It is a result of pressure and volume overload in the RV and can be seen in other disorders causing cor pulmonale. The S wave in lead I indicates a right bundle branch block. The Q wave, along with T-wave inversion, in lead III points to right ventricular strain.

Table 2 delineates the variety of ECG changes seen in PE. These case series show the innumerable ECG changes that can occur with varying frequencies during episodes of PE. Sinha and associates[18] showed that even though standard ECG findings have relatively low clinical use, they could increase the pretest probability of PE before performing computed tomography (CT) pulmonary angiography.

ST elevation is not among the usual findings associated with PE. It probably occurs due to acute right ventricular strain and elevated pressures resulting from a submassive or massive PE. This strain can cause

Table 2
Electrocardiographic Changes in Pulmonary Embolism

ECG Changes	Stein PD et al.[6] (n = 90) (%)	Nielsen TT et al.[19] (n = 87) (%)	Ferrari E et al.[3] (n = 80) (%)	Cutforth and Oram[22] (n = 50) (%)	Lenègre J et al.[20] (n = 50) (%)	Sreeram N et al.[21] (n = 49) (%)
Sinus tachycardia	69	NR	36	68	90	NR
TWI (V_1-V_4)	42	NR	68	46	89	NR
ST-T wave changes	26	40	NR	NR	NR	59
Axis deviation	21	9	NR	NR	NR	NR
Conduction defects (RBBB/LBBB/1°AVB)	19	34	22	40	24	67
Normal ECG	13	18	9	24	10	18
S1Q3T3	12	44	50	28	52	NR
Q wave V_1, III, aVF	11	NR	NR	NR	NR	49
Low-voltage ECG	6	NR	29	29	NR	NR
Pulmonary P wave	6	NR	5	12	NR	NR
Rhythm anomaly (APB/VPB/AFl/AFib)	6	6	NR	NR	NR	22
Right atrial hypertrophy	5	NR	NR	NR	NR	8
Right ventricle hypertrophy	5	NR	NR	NR	NR	NR

AFib, atrial fibrillation; AFl, atrial flutter; APB, atrial premature beats; AVB, atrioventricular block; ECG, electrocardiogram; LBBB, left bundle branch block; NR, not reported; RBBB, right bundle branch block; TWI, T-wave inversion; VPB, ventricular premature beats.

current of injury resulting in ST deviations.

Conclusions

We present a unique case of submassive PE that presented to the ED with ECG findings of ST elevations and Q waves in precordial leads, which was treated as a provisional diagnosis of STEMI. The elevated right heart pressures during catheterization and absence of CAD gave a clue to the presence of a PE, which was later confirmed by imaging. Our patient survived and is doing well on long-term anticoagulation.

We believe that in situations in which the clinical picture and ECG may cause a diagnostic dilemma, the use of cardiac markers and bedside TTE would add an element of certainty to the diagnosis and can be used as an adjunct to the ECG and clinical judgment. Needless to say, this will only come into practice if this presentation is common knowledge among cardiologists and emergency physicians. The ECG should be interpreted carefully in such cases for other evidence of changes

Main Points

- Electrocardiography alone is not sufficiently sensitive or specific to rule out or rule in the diagnosis of pulmonary embolism (PE).
- Sinus tachycardia and T-wave inversions are the most common electrocardiographic changes seen with PE.
- The McGinn-White sign (S1Q3T3), classically associated with PE, is seen in only 30% of cases.
- ST-segment elevations are highly suggestive of acute myocardial infarction but can also be seen in a variety of other cardiac and noncardiac conditions.
- Electrocardiographic changes should be interpreted cautiously in light of overall clinical presentation and, when required, supplemented with additional testing.

associated with PE. Whenever necessary, additional tests such as CT angiography, V/Q scans, and Doppler imaging should be used to differentiate these two etiologies. ■

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