



CASE REPORT

REVISED Case Report: Diffuse T wave inversions as initial electrocardiographic evidence in acute pulmonary embolism [version 2; referees: 2 approved]

Ogechukwu Egini ¹, Alix Dufresne², Mazin Khalid¹, Chinedu Egini³, Eric Jaffe⁴

¹Department of Medicine, Interfaith Medical Center, Brooklyn, NY, 11213, USA

²Division of Cardiology, Interfaith Medical Center, Brooklyn, NY, 11213, USA

³University of Port Harcourt, Port Harcourt, Nigeria

⁴Internal Medicine Residency Program, Interfaith Medical Center, Brooklyn, NY, 11213, USA

v2 First published: 13 Jun 2018, 7:738 (doi: [10.12688/f1000research.14927.1](https://doi.org/10.12688/f1000research.14927.1))
 Latest published: 06 Sep 2018, 7:738 (doi: [10.12688/f1000research.14927.2](https://doi.org/10.12688/f1000research.14927.2))

Abstract

Acute pulmonary embolism (PE) is a life-threatening condition and is typically diagnosed by a combination of symptoms, clinical signs and imaging. Electrocardiogram may be helpful in diagnosis, and the most widely described pattern of occurrence is the so-called S₁Q₃T₃ pattern. Here, we describe the case of an African-American male who presented with typical chest pain, diffuse T wave inversions with serial troponin elevation. There was initial concern for Wellen's syndrome but was finally diagnosed as acute PE. This case underscores the necessity of vigilance and a lower threshold for PE work up even in patients presenting as acute coronary syndrome.

Keywords

PE, T-waves, inversion

Open Peer Review

Referee Status:

	Invited Referees	
	1	2
REVISED version 2 published 06 Sep 2018		 report
version 1 published 13 Jun 2018	 report	 report

- 1 **Chibundo Uchenna Nwaneli**, Nnamdi Azikiwe University, Nigeria
- 2 **Chukwudi Obiagwu**, Maimonides Medical Center, USA

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Corresponding author: Ogechukwu Egini (ogechukwu.egini@yahoo.com)

Author roles: **Egini O:** Conceptualization, Data Curation, Investigation, Supervision, Validation, Visualization, Writing – Original Draft Preparation; **Dufresne A:** Writing – Review & Editing; **Khalid M:** Data Curation, Writing – Original Draft Preparation; **Egini C:** Writing – Original Draft Preparation; **Jaffe E:** Writing – Review & Editing

Competing interests: No competing interests were disclosed.

Grant information: The author(s) declared that no grants were involved in supporting this work.

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How to cite this article: Egini O, Dufresne A, Khalid M *et al.* **Case Report: Diffuse T wave inversions as initial electrocardiographic evidence in acute pulmonary embolism [version 2; referees: 2 approved]** *F1000Research* 2018, 7:738 (doi: [10.12688/f1000research.14927.2](https://doi.org/10.12688/f1000research.14927.2))

First published: 13 Jun 2018, 7:738 (doi: [10.12688/f1000research.14927.1](https://doi.org/10.12688/f1000research.14927.1))

REVISED Amendments from Version 1

We have updated our reference to reflect recent findings on the cost imperative of treating acute PE in the United States as shown in a newer study and have also included images of the chest CT angiogram (Figure 3), as well as a video of the coronary angiogram (Supplementary File 1).

See referee reports

Introduction

Acute pulmonary embolism (PE) is responsible for 20–25% of sudden death in the United States^{1,2}. It exacts a huge economic burden both on the sufferer and the health system. In their study to assess PE and deep vein thrombosis (DVT) inpatient costs in the United States, Lamori *et al.* found that the mean cost of initial hospitalization for acute PE was approximately \$37,006 per patient³. This figure was higher for older patients, women and readmissions. Prompt diagnosis is, therefore, essential to reduce disease burden. The so-called S₁Q₃T₃ pattern is the classic electrocardiogram (EKG) presentation in acute PE⁴ but is not seen in all acute PE cases. We present the case of acute PE with initial clinical presentation that mimicked acute coronary syndrome and an initial EKG pattern that suggested Wellen's syndrome.

Case report

A 66 year old African-American male presented to the Emergency Room (ER) complaining of a 2-hour history of chest pain. Chest pain was described as left-sided, non-pleuritic, non-radiating, retrosternal, squeezing in character and persistent. Pain was reported as 9 on a 10-point pain scale and relieved by taking 0.4mg tablet of nitroglycerin sublingually. It was associated with shortness of breath, dizziness and sweating, but the patient denied loss of consciousness, cough, palpitation or

swelling of the extremities. He denied any use of illicit substances. A week prior to this hospitalization he presented to the hospital with a similar complaint. At that time, chest pain was relieved by 325mg dose Aspirin taken orally; troponin was normal and EKG did not show any significant change from baseline. His echocardiogram was also normal and he was discharged with scheduled outpatient stress test. Medical history was significant for poorly-controlled diabetes type 2, hypertension, dyslipidemia and obesity.

On this visit, his pulse rate was 84 beats per minute; BP 119/66 mm/Hg; respiration rate 16 breaths per minute and his oxygen saturation was 98% on room air. Initial troponin was elevated at 0.19ng/ml (reference 0.00 – 0.05ng/ml); hemoglobin of 14.4g/dl (reference 13–17g/dl) and platelet count of 210 × 10³/ul (reference 130–400 × 10³/ul).

EKG showed deep T wave inversions in leads V1–V6 and the inferior limb leads (Figure 1). We assumed an assessment of non-ST elevation myocardial infarction and a loading dose of Aspirin (325 mg) and Plavix (300 mg) were given orally in the ER along with Atorvastatin (80 mg) and a weight-based dose of Enoxaparin. Repeat troponin 6 hours later was 1.05. Cardiac catheterization revealed normal coronaries (Supplementary file 1). While the patient was still lying on the cardiac cath table, his oxygen saturation dropped to 91%. There was no chest pain, tachypnea or tachycardia at this time. Supplemental oxygen at 2l/min via nasal cannula improved saturation to 97%. A repeat EKG showed a Q₃T₃ pattern in lead III (Figure 2). In view of these new findings (low oxygen saturation and a change in the EKG pattern), a computerized tomography of the chest with angiogram (chest CTA) was ordered. This revealed a saddle pulmonary embolus which extended into the right and left pulmonary arteries and involved all lobar branches of the pulmonary arteries (Figure 3).

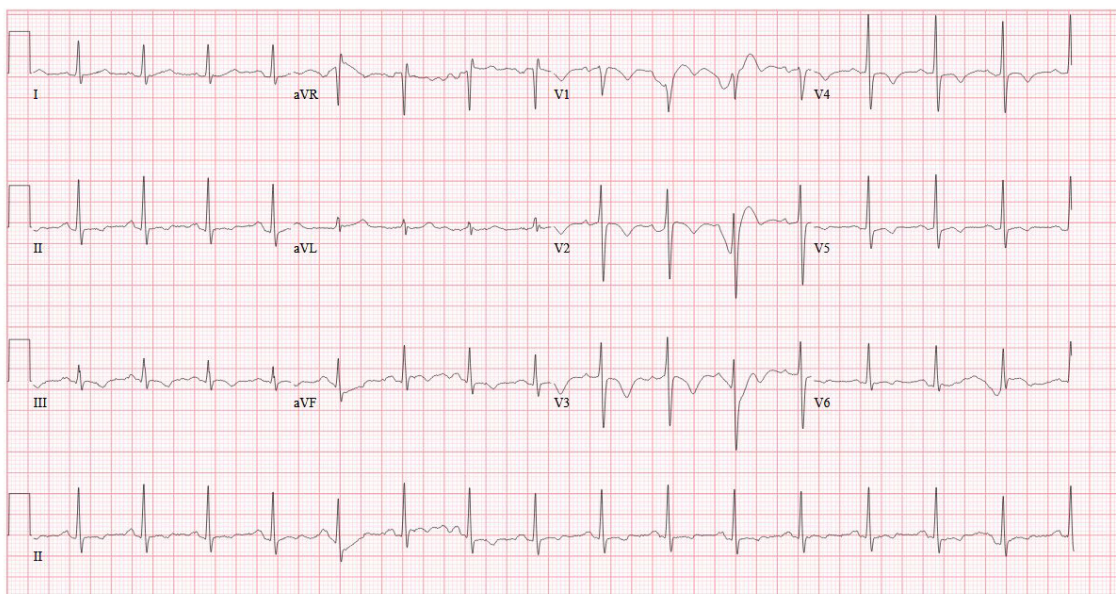


Figure 1. EKG showing deep T wave inversions in leads V1–V6 and the inferior limb leads.

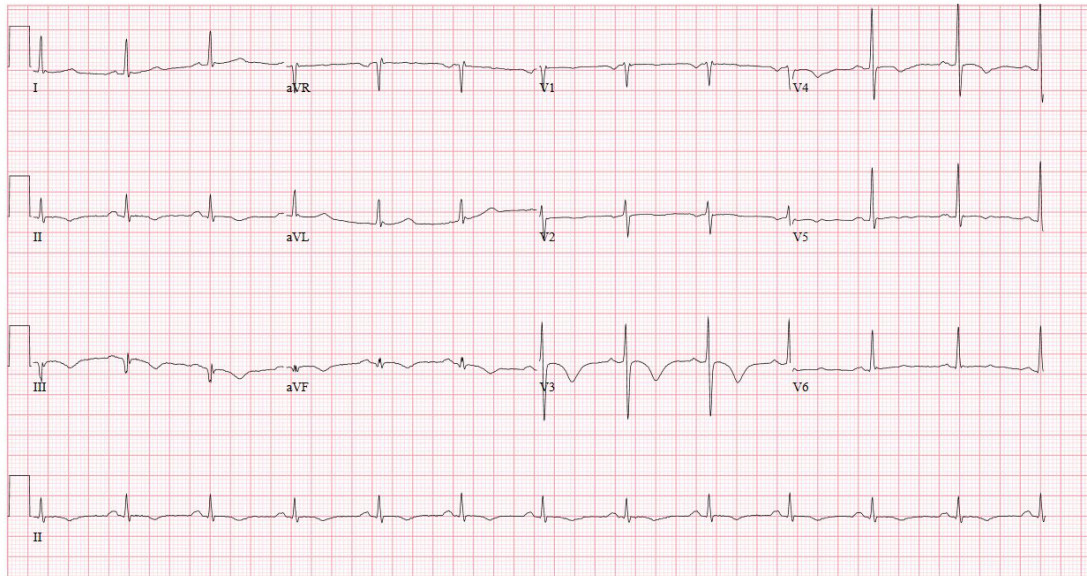


Figure 2. Repeat EKG now showing a Q_3T_3 pattern in lead III.

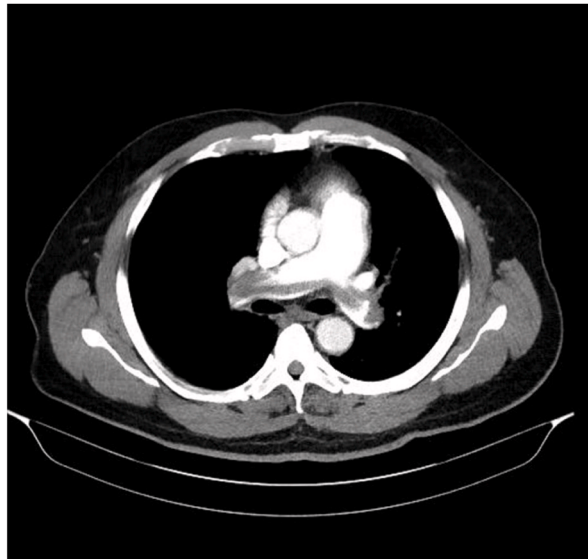


Figure 3. Axial CTA of the chest showing a saddle embolus with extension into the branches of the pulmonary artery. CTA was performed using Siemens SOMATOM Perspective 128 slices. Images were obtained in a cranio-caudal direction following contrast injection at 3ml/s. Contrast optimization was based on bolus tracking at the level of the main pulmonary artery using a trigger level of 100 HU.

Treatment was continued with Enoxaparin (100mg subcutaneously every 12 hours) for 6 days, at which time he became stable and maintained oxygen saturation above 96% even when supine. He was discharged on Apixaban (10mg po bid for 7 days followed by 5mg po bid) with plan to complete 3 months of therapy. Follow up visits were scheduled with the Cardiology and Hematology clinics.

Discussion

Acute pulmonary embolism (PE) is caused by blockage of a pulmonary artery by blood clot. In one study, investigators

found that the commonest clinical symptoms in acute PE patients were dyspnea, chest pain, syncope and hemoptysis⁴. A number of EKG findings have been described in acute PE patients but the classic EKG finding is the $S_1Q_3T_3$ pattern⁵. The incidence of this pattern in acute PE is highly variable⁵. Other EKG changes have been reported in patients diagnosed with PE⁶ but there were initial supporting clinical evidence to warrant suspicion and further diagnostic testing for PE. On the contrary, our patient presented with features suggestive of acute coronary syndrome - typical chest pain, diffuse T wave inversions and elevated cardiac enzymes. Pulse rate, respiration rate and

oxygen saturation were normal essentially making an acute PE assessment difficult at time of presentation. Given a background of significant cardiovascular risk factors, a coronary event was thought more likely. Deep T wave inversions on the precordial leads were concerning for Wellen's syndrome⁷. The clues to possible acute PE in our case was the transient desaturation that occurred during cardiac catheterization and the observed change on repeat EKG. These dictated the urgency of getting a chest CTA. The chest CTA is the gold standard for diagnosis of PE and was shown in the Prospective Investigation of Pulmonary Embolism Diagnosis II (PIOPED II) to have a high sensitivity and specificity for acute PE diagnosis and was also concordant with the pretest Well's criteria⁸. A ventilation-perfusion (V/Q) scan may also effectively diagnose acute PE and is useful in renal insufficiency or contrast allergy. Treatment of acute PE is based on risk stratification. Anticoagulation is the mainstay of therapy and the duration of treatment is determined by a number of factors including provoked vs unprovoked PE and/or recurrence of acute PE. Those with acute PE and hypotension without significant bleeding risk require thrombolysis⁹. In some cases of massive PE with contraindication to or failure of

systemic fibrinolysis, surgical or catheter embolectomy can be considered¹⁰.

Conclusion

Acute pulmonary embolism should be considered as a differential in patients with deep T wave inversions on EKG who do not have typical PE presentation.

Consent

Written informed consent for the publication of the patient's clinical details and clinical images was obtained from the patient.

Data availability

All data underlying the results are available as part of the article and no additional source data are required.

Grant information

The author(s) declared that no grants were involved in supporting this work.

Supplementary material

Supplementary File 1. Video file of angiogram showing patent coronaries

[Click here to access the data](#)

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Open Peer Review

Current Referee Status:  

Version 2

Referee Report 05 October 2018

doi:[10.5256/f1000research.17710.r38032](https://doi.org/10.5256/f1000research.17710.r38032)



Chukwudi Obiagwu

Department of Cardiology, Maimonides Medical Center, New York City, NJ, USA

I thank the authors for reviewing the manuscript. Interesting report.

Competing Interests: No competing interests were disclosed.

Referee Expertise: Cardiology, heart failure, interventional cardiology

I have read this submission. I believe that I have an appropriate level of expertise to confirm that it is of an acceptable scientific standard.

Version 1

Referee Report 10 July 2018

doi:[10.5256/f1000research.16252.r35031](https://doi.org/10.5256/f1000research.16252.r35031)



Chukwudi Obiagwu

Department of Cardiology, Maimonides Medical Center, New York City, NJ, USA

The author describe a case of acute PE initially masquerading as acute MI. The authors do a good job presenting a manuscript that is easy to read. However, there are a few points to be noted:

1. Introduction:

The authors on citing the cost of care for PE rely on an old article whereas there are more recent articles providing more current cost estimates. See citation added below¹.

2. The authors use of punctuation marks while describing the characteristics of chest pain needs to be reviewed.

3. Is "in this visit" the right phrase to use or "on this visit" the proper one?

4. A review of this sentence "Oxygen saturation dropped to 91% in room air while the patient laid on catheterization table but improved with supplemental oxygen via nasal cannula" is needed.

5. The authors would do well to provide an image of the coronary angiogram and CTA PE protocol.
6. Post coronary angiogram, while in the cath lab, did the patient still complain of chest pain, was he tachypneic, or in distress that led to desaturation to 91%? A lot health care providers will not get a CTA chest for O2 saturation of 91% without other symptoms.

On seeing normal coronary arteries, prior symptoms could have been ascribed to MINOCA. See citation 2².

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Is the background of the case's history and progression described in sufficient detail?

Partly

Are enough details provided of any physical examination and diagnostic tests, treatment given and outcomes?

Yes

Is sufficient discussion included of the importance of the findings and their relevance to future understanding of disease processes, diagnosis or treatment?

Yes

Is the case presented with sufficient detail to be useful for other practitioners?

Yes

Competing Interests: No competing interests were disclosed.

Referee Expertise: Cardiology, heart failure, interventional cardiology

I have read this submission. I believe that I have an appropriate level of expertise to confirm that it is of an acceptable scientific standard, however I have significant reservations, as outlined above.

Author Response 04 Sep 2018

Ogechukwu Egini, Interfaith Medical Center, USA

Thank you for the responses.

We have updated Version 2 to reflect more recent analysis of the cost imperative of treating acute PE in a US hospital and have also included images of both coronary angiogram and an axial cut of the CTA PE. MINOCA is a consideration in this situation. However, given the 2 new findings of a

drop in oxygen saturation and the change in EKG pattern, we thought it was best to rule out acute PE at that time.

Competing Interests: No competing interests were disclosed.

Referee Report 03 July 2018

doi:10.5256/f1000research.16252.r35034



Chibundo Uchenna Nwaneli

Department of Medicine, Faculty of Medicine, Nnamdi Azikiwe University, Awka, Nigeria

The article I reviewed is a case report of 66 year old African American who presented to the hospital with recurrent chest pain whose initial evaluation was thought to be consistent with coronary artery disease. He was subsequently found to have Acute pulmonary embolism on Chest CT angiogram and normal coronary vessels. This case is quite intriguing because of the presentation. It throws up some question I would like the authors to try to answer and offer explanation.

1. The initial presentation with chest pain which resolved with administration of 325mg aspirin: did the team think it was pulmonary embolism. Is it usual for embolism to respond with single low dose aspirin.
2. The relief of patient's pain with Nitroglycerin, is it typical with chest pain from embolism.
3. Is it possible that we are dealing with Non ST segment myocardial infarction (NSTEMI) coexisting with pulmonary embolism.
4. Myocardial infarction occurring in the setting of normal coronary arteries have been reported. Could this be the case here knowing fully well that this patient had multiple risk factors for coronary artery disease.

The EKG findings observed in the index patient have been described in pulmonary embolism as well as the elevated troponin. They could as well be found in NSTEMI.

Treatment for submassive pulmonale embolism and NSTEMI both require anticoagulation.

I dont know how many pictorials that are allowed for case report by the journal but I would like the authors to include the baseline EKG of the patient on his first presentation to the hospital. If also permissible the Chest CT and Coronary angiogram images. This I think will enable readers agree with their conclusions.

Overall the case report was well written.

The take home message is that diagnosis of pulmonary embolism can be difficult especially when the features mimic other conditions such as acute coronary syndrome. EKG changes may give a clue to the diagnosis. As clinicians we know the inexactness of scientific data and we should have an open mind to other differential diagnosis.

Is the background of the case's history and progression described in sufficient detail?

Yes

Are enough details provided of any physical examination and diagnostic tests, treatment given and outcomes?

Yes

Is sufficient discussion included of the importance of the findings and their relevance to future understanding of disease processes, diagnosis or treatment?

Partly

Is the case presented with sufficient detail to be useful for other practitioners?

Yes

Competing Interests: I know the lead author in person. He was a final year medical undergraduate in the hospital when i was doing junior residency in internal medicine. However we have not collaborated in any study and we have not seen in close to 5 years.

Referee Expertise: Heart failure, Hypertension, Echocardiography, Cardiovascular Risk factors

I have read this submission. I believe that I have an appropriate level of expertise to confirm that it is of an acceptable scientific standard.

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