CASE REPORT

Case Report: Diffuse T wave inversions as initial electrocardiographic evidence in acute pulmonary embolism [version 1; peer review: 1 approved, 1 approved with reservations]

Ogechukwu Egini1, 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

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

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1 Chibundo Uchenna Nwanell, Nnamdi Azikiwe University, Awka, Nigeria

2 Chukwudi Obiagwu, Maimonides Medical Center, New York City, USA

Any reports and responses or comments on the article can be found at the end of the article.
Introduction

Acute pulmonary embolism (PE) is responsible for 20–25% of sudden death in the United States\(^1\). It exacts a huge economic burden both on the sufferer and the health system with some estimates placing the annual cost of care between $7,594 to $16,644 per patient\(^3\). Prompt diagnosis is essential to reduce disease burden. The so-called S\(_3\)Q\(_3\)T\(_3\) pattern is the classic electrocardiogram (EKG) presentation in acute PE\(^4\) 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.

In 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 × 10\(^3\)/ul (reference 130–400 × 10\(^3\)/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. Left heart catheterization revealed normal coronaries. Oxygen saturation dropped to 91% in room air while the patient laid on catheterization table but improved with supplemental oxygen via nasal cannula. A repeat EKG at this time showed a Q\(_3\)T\(_3\) pattern in lead III (Figure 2). This was followed by a computerized tomography of the chest with angiogram (chest CTA), which revealed a saddle pulmonary embolus which extended into the right and left pulmonary arteries and involved all lobar branches of the pulmonary arteries.

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 hemoptysis\(^4\).

Figure 1. EKG showing deep T wave inversions in leads V1-V6 and the inferior limb leads.
A number of EKG findings have been described in acute PE patients but the classic EKG finding is the S\textsubscript{1}Q\textsubscript{3}T\textsubscript{3} pattern\textsuperscript{5}. The incidence of this pattern in acute PE is highly variable\textsuperscript{5}. Other EKG changes have been reported in patients diagnosed with PE\textsuperscript{6} 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 syndrome\textsuperscript{7}. The only clue to possible acute PE in our case was the transient desaturation that occurred during cardiac catheterization. This 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 criteria\textsuperscript{8}. 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 thrombolysis\textsuperscript{9}. In some cases of massive PE with contraindication to or failure of systemic fibrinolysis, surgical or catheter embolectomy can be considered\textsuperscript{10}.

**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.

**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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**References**

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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 below.

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.

**References**
2. Pasupathy S, Air T, Dreyer RP, Tavella R, Beltrame JF: Systematic review of patients presenting with

PubMed Abstract | Publisher Full Text

**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.

**Reviewer 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, Brooklyn, 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.

Reviewer Report 03 July 2018

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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.

**Reviewer 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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