A Frog in the Well's Score – Shortcomings of the Well's Score for Diagnosis of Acute Pulmonary Embolism

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ABSTRACT

Pulmonary embolism (PE) is one of the leading causes of undiagnosed deaths in patients worldwide due to its unpredictable clinical course and mimicry of various other diseases. The often over-used simplified Well's score and D-dimer test must be utilized with prudence to stratify the probability of PE. Astute interpretation of electrocardiography (ECG) signs and bedside echocardiography findings has helped Emergency Physicians narrow down a diagnosis of PE. PE remains a diagnostic challenge; nevertheless, with high index of suspicion, appropriate understanding of clinical probability scores and use of bedside screening tests like ECG and bedside echocardiography, PE can be rapidly diagnosed and managed in the Emergency Department.

Keywords: Emergency Department, pulmonary embolism, Well's score, bedside echocardiography, empirical heparin, CT pulmonary angiogram, McConnell's sign

Pulmonary embolism (PE) is one of the leading causes of undiagnosed deaths in patients worldwide due to its unpredictable clinical course, highly variable symptomatology, mimicry of various other diseases and difficulty in obtaining reliable diagnostic tests. The triad of cough, dyspnea and hemoptysis is found in abysmally low number of patients with PE. The often over-used simplified Well's score and D-dimer test must be utilized with prudence to stratify the probability of PE prior to confirmation with a computed tomography pulmonary angiography (CTPA), which is the gold standard diagnostic test for PE. However, high cost, unavailability in all medical centers, reliance on a normal renal function and high radiation output often make it unfeasible for all patients. Astute interpretation of pathognomonic electrocardiography (ECG) signs and characteristic bedside echocardiography findings have shown promise in helping Emergency Physicians narrow down a diagnosis of PE, as well as rapidly confirming the presence or absence of other conditions, which present in similar fashion. Studies have also shown that initiation of 'empirical heparin' in the Emergency Department (ED) greatly reduced morbidity and mortality in such patients for whom PE is suspected, but a CTPA is either delayed or not feasible. We present the case of a 76-year-old male with a different presentation from the usual described triad of PE. This shows how a broad suspicion and bedside echocardiography help in rapid diagnosis and treatment.

CASE REPORT

Mr KV, a 76-year-old man was brought to the ED, with a laceration measuring 7 cm, over the right parietal area of his scalp, associated with history of collapse in the bathroom early that morning. There were no witnessed seizures and he regained consciousness while in the ambulance towards the hospital, but vomited 4 times in quick succession. Over the last 1 month, he had suffered from recurrent fainting spells, which resolved spontaneously over half an hour and with no associated seizures or vomiting. He was not a smoker or consumer of alcohol, neither had he ever had any previous surgeries. There was no prior history of diabetes, hypertension, chronic kidney disease, ischemic heart disease, asthma or chronic obstructive pulmonary disease. On arrival, he was conscious and oriented (Glasgow Coma Scale [GCS] 15/15), with bilateral equally reactive pupils. He was

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afebrile with regular pulse rate of 116/min and blood pressure of 140/90 mmHg. Although he had no respiratory discomfort, his respiratory rate was 26/min with pulse oximetry of 88% on room air. There was a 7 cm horizontal laceration over his right parietal region and the rest of systemic examination was unremarkable.

Arterial blood gas analysis demonstrated hypoxia (PaO_2 = 68% with an SaO_2 of 86%), but no other significant findings. His chest X-ray was unremarkable. Plain computed tomography (CT) brain was undertaken on account of sudden collapse in the toilet with multiple episodes of vomiting as well as similar episodes of collapse, in the past. However, it was a normal study. Baseline blood investigations indicated leukocytosis of 16,100/mm^3, with serum creatinine of 1.7 mg/dL. Due to his "comfortable hypoxia", the absence of clinical chest findings and persistent tachycardia, a diagnosis of PE was considered. Well's score was calculated to be 4.5 and bedside D-dimer assessment was found to be 4,910 ng/mL.

The ECG showed sinus tachycardia (Fig. 1). Bedside echocardiography showed dilated right ventricle and right atrium with positive McConnell's sign, i.e., regional right ventricular (RV) dysfunction, akinesia of the mid free wall, but normal motion at the apex. CTPA was deferred due to elevated creatinine. Unfractionated heparin therapy was empirically initiated with bolus dose of 5,000 units IV, and continued thereafter. The patient was admitted for further care and 3 days later, after normalizing the serum creatinine, CTPA was performed, confirming the diagnosis of PE - multiple filling defects involving the right pulmonary artery extending into the segmental branches. A representative image from the scan series is enclosed (Fig. 2). The patient was treated with 5 days of IV unfractionated heparin, after which he was discharged in a stable condition, on warfarin.

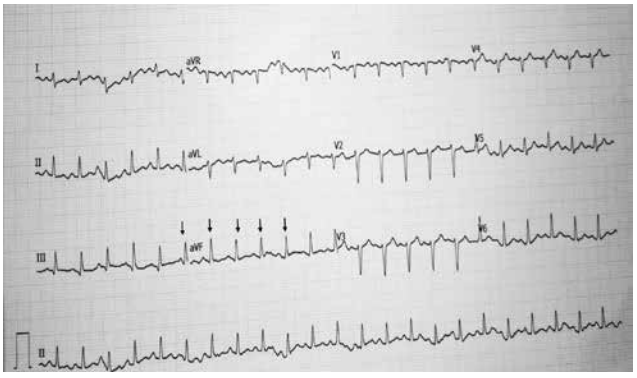

Figure 1. ECG of patient. Black arrows indicate sinus tachycardia.

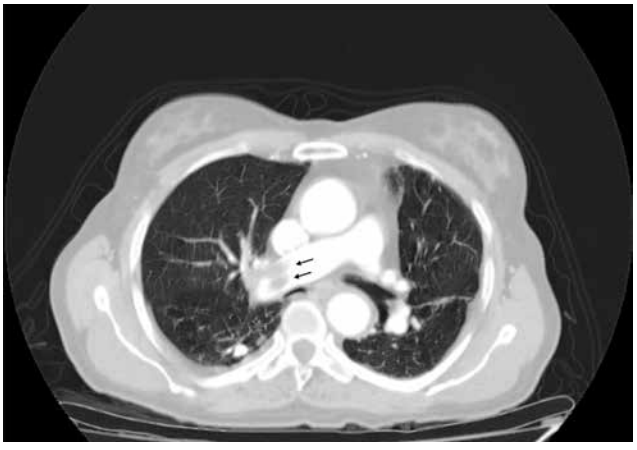

Figure 2. CTPA (*black arrows*) indicating multiple filling defects involving the right pulmonary artery extending into the segmental branches.

DISCUSSION

Acute PE is a major harbinger of death, a major cause of undiagnosed mortality worldwide. The diagnosis of PE has always been challenging, as it is the second leading cause of sudden death without a discernible cause, due to wide variety of presenting symptoms, quick progression and paucity of rapid diagnostic modalities with high sensitivity and specificity. The typical triad of 'Chest Pain, Dyspnea and Hemoptysis' is found in absurdly few number of patients, and presentation of PE can vary greatly. The presentation could be extremely subtle with rare occurrences such as seizures, as part of the symptom complex. In fact, PE was identified in nearly 1 of every 6 patients hospitalized for a first episode of syncope. The commonly employed Well's score, though a useful clinical screening tool, has limited diagnostic capacity in PE. Conditions such as pneumonia, pneumothorax, sepsis or even panic attacks would sufficiently satisfy the Well's score criteria and lead to over-investigation for PE. Further, the challenge for diagnosis is frequently compounded by significant delay in organizing CTPA, due to multiple factors including unstable hemodynamic status of patient, deranged renal function and delay in obtaining CT in centers, which do not have the privilege of 24 × 7 imaging services.

Role of ECG

The role of ECG in prognosticating PE is increasingly recognized. The ECG in addition to clinical acumen, steers the Emergency Physician towards the diagnosis. McGinn and White described the first association between acute PE and specific ECG changes when they noted the familiar 'S1Q3T3' pattern in 7 patients

with acute cor pulmonale. However, it is reported that patients with normal ECGs at admission revealed diagnostic features of embolism in serial ECGs carried out subsequently. ECG findings in PE range from sinus tachycardia (44% of patients), right axis deviation (16% of patients), right bundle branch block (18% of patients), inverted T waves in leads V1-V4 (right ventricular strain pattern - 18% of patients), and the "S1Q3T3" pattern (20% of patients), and any one of these findings doubles the probability of PE. The most common ECG changes when compared with previous ECG in the setting of PE are T-wave inversion and flattening, most commonly in the inferior leads, and occurring in approximately one-third of cases. It has been observed that approximately one-quarter of patients will have new-onset sinus tachycardia.

The fact that ECG provides invaluable information for PE prognostication was reaffirmed by a meta-analysis of 39 studies (9,198 patients). ECG signs that were good predictors of a negative outcome included S1Q3T3, complete right bundle branch block, T-wave inversion, right axis deviation and atrial fibrillation for in-hospital mortality. It was concluded that ECG is potentially valuable in prognostication of acute PE. Moreover, changes in right-sided chest leads occur frequently in PE. Routinely recorded right-sided ECG appears to possess the greatest potential for diagnosing acute PE in patients who have not manifested typical changes in their standard 12-lead ECGs. However, it needs to be emphasized that approximately one-quarter of patients with PE may have no changes in their ECG.

Fragmented QRS (fQRS) is a convenient marker of myocardial scar evaluated by 12-lead ECG and is defined as additional spikes within the QRS complex. The presence of fragmented QRS complex (fQRS), as a simple and feasible ECG marker, seems to be a novel predictor of in-hospital adverse events and long-term all-cause mortality in PE patient population. There is scarcity of data on the prognostic importance of fQRS on short- and long-term outcomes in patients with PE.

Bedside Echocardiography

Bedside echocardiography demonstrates right ventricular dilation, reduced contractility with apical sparing, which is known as McConnell's sign and is very specific in acute PE.

Both these tests are useful for patients who are too unstable to be shifted out of the ED for CTPA, or who have deranged renal parameters, which make the use of IV contrast risky. Thrombolytic therapy, based on bedside echocardiography, has also been shown to produce successful outcome in low-resource settings.

D-dimer

D-dimer is a fibrin breakdown product which is elevated whenever the fibrinolytic system of the body is activated. The usage of D-dimer as a diagnostic marker for PE is to be discouraged, since the differential diagnoses for elevated D-dimer values in the setting of dyspnea are numerous, including sepsis, hemothorax, myocardial infarction, congestive cardiac failure, etc. D-dimer has very high sensitivity (up to 95%), but low specificity (approximately 45%) for PE, and must only be used as a 'rule out' rather than diagnostic test for patients with a low pre-test probability of PE. In a recent study on 614 patients, it has been reported that ECG signs of right ventricular strain are strongly related to elevated cardiac biomarkers and echocardiographic signs of right ventricular overload.

However, rapid point-of-care testing for D-dimer in patients who have simplified Well's score of <4, would be beneficial to indicate the clinical pathway to investigate for PE. A triad of circulatory collapse, right ventricular dilatation and large alveolar dead space is proposed for the rapid diagnosis and treatment of massive PE.

Thrombolytic Therapy

The role of thrombolytic therapy in acute PE patients is still controversial and early-onset thrombolytic therapy in the ED for high-risk and hemodynamically worsening patients appears safe and life-saving. Short-term effects of thrombolytics are well-known, whereas long-term effects on cardiac electrophysiology have not been reported before. In the absence of contraindications, it is reasonable to administer a patient with suspected PE 'empirical heparin' before confirmatory imaging, as early anticoagulation therapy is vital for management of acute PE and delay of which increases mortality significantly. Low-molecular weight heparin (LMWH) has several advantages over unfractionated heparin; however, in cases of deranged renal function, as seen in this case, unfractionated heparin is preferred.

CONCLUSION

Rapid diagnosis of PE in the ED greatly improves survival rates, but requires a systematic, multivariate approach involving high clinical suspicion, good understanding of pre-test probability with appropriate use of D-dimer, use of easily available bedside modalities such as ECG and bedside echocardiography and ideally, CTPA. Treatment can be started in the

ED on an empirical basis with unfractionated heparin, which can be continued or stopped depending on the angiography report. High Well's score is not diagnostic of PE in itself, neither is elevated D-dimer level. Appropriate clinical judgment must be used along with pre-test probability scoring and appropriate testing of D-dimer levels for all low-risk patients only. ECG and bedside echocardiography can be used for patients with elevated D-dimer levels to assess the right ventricular function and guide the Emergency Physician to start empirical heparin before shifting the patient for a CTPA.

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Formula of 10

- Start CPR within 10 minutes.
- Continue CPR for at least 10 minutes.
- Give at least 10 x 10, 100 compressions per minute.
- Assume the patient is in cardiac arrest if there is no breathing or abnormal breathing (e.g., gasping) or if a pulse cannot be readily palpated within 10 seconds.