McConnel sign, a simple highly specific sign for identification of a frequent, under diagnosed association: pulmonary embolism and syncope

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Abstract

Syncope is a common reason for emergency room visits and hospital admission and, in about one patient out of six it can be a manifestation of pulmonary embolism (PE). The prognosis of this condition is largely dependent on the timing of diagnosis and treatment since early anticoagulation may modify the clinical consequences of PE and further loss of consciousness. In this article, we describe the case of a woman presenting with syncope, who showed at the admission a cardiac ultrasound highly specific cluster of signs of pulmonary embolism. This rapid non-invasive evaluation permitted to identify causal diagnosis and to speed up the clinical process.

Case Report

An 89-year-old female patient was admitted to the emergency room for a recent syncope nearly one hour before admission. The patient had temporarily lost consciousness and had fallen on the floor with retrograde amnesia. On physical examination, she was awake, hemodynamically stable (TA 175/85 mm Hg), rhythmic heart rate (79 per minute), with mild hypoxia (89% on pulse oximeter), and without fever.

A 12-lead electrocardiogram showed normal sinus rhythm with S1QT3 pattern and an incomplete right bundle branch block (Figure 1). Arterial blood gas analysis revealed a hypoxic and hypocapnic profile (pO_2 53 mmHg; pCO_2 31 mmHg). The laboratory results revealed increase in CRP (63 mg/dL), leukocytosis (20.32×10^9/L), and increased serum troponin (52 ng/L).

The thoracic X-ray evidenced bilateral interstitial effusion and enlarged heart, suggesting initial heart failure. The low back X-ray showed a depression of the upper border of L3, therefore a corset was placed.

Focused cardiac ultrasound showed an enlarged right ventricle, greater than the left ventricle, hypokinetic lateral wall, and hyperkinetic apex of the right ventricle (McConnell sign). This typical ultrasound sign is quite specific for diagnosing acute pulmonary embolism (specificity 94%).

Because of a high probability of pulmonary embolism, the anticoagulant therapy was started with enoxaparin 4000 UI b.i.d. The dose of enoxaparin was calculated according to renal function (creatinine clearance 32 mL/min).

The patient underwent computed tomography angiography of the thorax that revealed filling defects on both main pulmonary arteries, especially in the middle lobe's area (Figure 2), and showed an enlarged right ventricle, which was greater than the left ventricle (Figure 3).

The echo-color Doppler of the lower limbs resulted negative for deep venous thrombosis, while the abdomen ultrasound showed a hypo-hyperechoic lesion of 4.5 cm in the left hepatic lobe. Computed tomography of the abdomen with contrast demonstrated a hepatic angioma.

The clinician decided to shift from heparin therapy to a direct oral anti-coagulant, so edoxaban 30 mg was started based on the creatinine clearance calculated with Cockcroft-Gault formula.

During the hospital stay, the patient underwent oxygen therapy with 2 liters of oxygen, maintaining normal pulse oximetry.

She was discharged after eleven days of hospitalization and admitted to an elderly care institution because of numerous comorbidity and cognitive impairment.

Discussion

Syncope, defined as a self-limited transient loss of consciousness and postural tone due to global cerebral hypoperfusion, is a common reason for emergency room visits, with a third of these visits leading to inpatient admission. Syncope carries an estimated 2-year mortality rate of over 25% and is strongly associated with falls, a potentially devastating contributor to morbidity and mortality among the elderly.[1] Syncope accounts for up to 6% of all hospitalizations and 3% of all emergency room visits.[2,3]

The prevalence of syncope increases with age, exceeding 20% in those aged ≥75 years,[4] with an annual incidence that approaches 2% in persons over 80 years. In addition to multi-morbidity and polypharmacy, several age-related changes in cardiovascular structure and function contribute to the higher incidence and prevalence of syncope in the elderly. These most important include attenuated baroreceptor and autonomic reflexes, diastolic dysfunction, impaired adrenergic responsiveness, and impaired maintenance of intravascular volume related to decreased salt/water handling and reduced renin-aldosterone levels.[5] So, it is important to know the physiologic changes that occur with normal aging to better understand their interaction with disease processes and facilitate better evaluation and treatment of syncope when it happens. Attention to situational stresses, such as posture changes, meals, or medications, is also likely to increase the diagnostic yield and improve therapeutic strategies that can reduce morbidity and potential mortality of recurrent episodes. Therapy should be directed toward minimizing multiple factors that contribute to syncope, avoiding iatrogenic medication effects, and treating specific contributory diseases.[5]

Etiologies of syncope may be grouped according to underlying pathophysiology-neutrally-mediated, orthostatic/dysautonomic, and cardiac, which include arrhythmic and structural etiologies.[6] Neurally-mediated etiologies account for almost two-thirds of syncope, whereas arrhythmic and structural cardiovascular etiologies account for the minority. Although identifying a predominant etiology is important for management, syncope is often multi-factorial in the elderly, highlighting the importance of

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maintaining a broad differential during the evaluation period.[1]

Despite the exponentially increasing incidence of syncope in people aged 70 and older, data gathered in this population are limited. In particular, little is known about the relative frequency of causes of loss of consciousness in older persons, essentially because a standardized diagnostic protocol is infrequently used at older ages.[7] The Italian Group for the study of syncope in elderly (GIS) designed a standardized algorithm derived from European Society of Cardiology guidelines published in 2001.[8] The first study reporting the use of this protocol was published in The American Geriatrics Society in 2006 and showed that the neurally-mediated syncope, which had previously been reported to occur infrequently in older people,[9] was the most frequent type of syncope in this population.[7]

Syncope is a well-known and quite common manifestation of pulmonary embolism (PE), and its prognosis is largely dependent on the timing of diagnosis and treatment.[8] Syncope is a severe manifestation of PE. Firstly, it places patients at risk of death because of the unpredictable consequences of the loss of consciousness. Secondly, it generally represents the manifestation of either anatomical or clinically severe PE. Whether implementing or not aggressive (*i.e.*, thrombolytic) therapy in patients with PE and high risk of adverse events in spite of stable clinical condition is a matter of debate, as a recent major randomized clinical trial showed the benefit/risk of such strategy might not be favorable, especially in patients older than 75.[10] However, physicians should be alerted and consider this option whenever there are no contraindications other than advanced age to thrombolysis. Patients may be offered adjusted-dose intravenous unfractionated heparin instead of low-molecular-weight heparin in order to achieve rapid anticoagulation. They should be carefully monitored so that the decision whether continuing anticoagulation alone or associate it with a course of thrombolytic therapy is timely taken.[11]

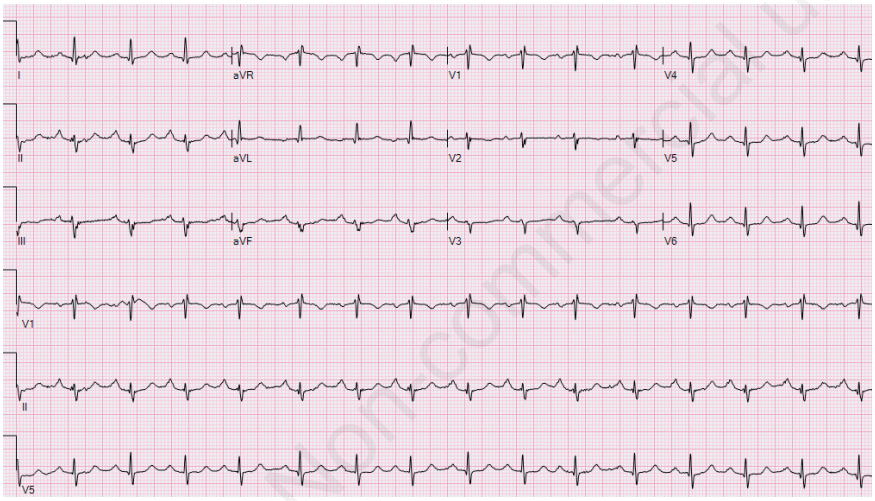

Figure 1. A 12-lead electrocardiogram at the time of admission.

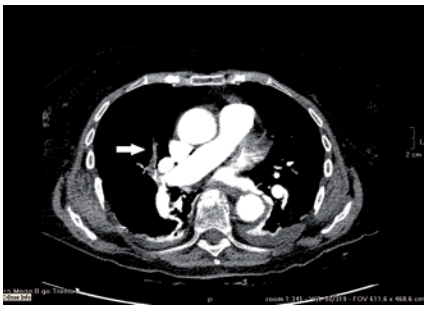

Figure 2. Filling defects on both main pulmonary arteries, especially in the middle lobe's area.

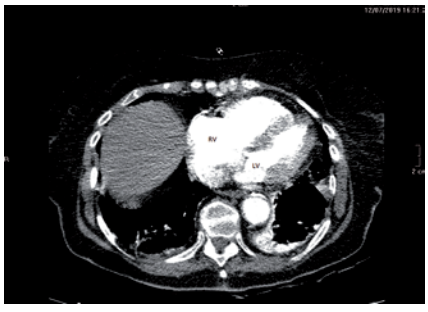

Figure 3. Enlarged right ventricle (RV), which was greater than the left ventricle (LV).

Prandoni *et al*. demonstrated that among patients who were hospitalized for a first episode of syncope in eleven different centers and who were not receiving anticoagulation therapy, pulmonary embolism was confirmed in 17.3% (approximately one of every six patients). The rate of pulmonary embolism was highest among those who did not have an alternative explanation for syncope.[12]

The case report shown above underlines the importance of a full syncope diagnostic algorithm, which should include assessment for pulmonary embolism, especially in elderly patients.

The recent guidelines of the European Society of Cardiology do not recommend echocardiography as routine imaging modality in patients with suspected not high-risk pulmonary embolism.[13] However, because of its availability and noninvasive character, it can be a proper imaging method in the differential diagnosis of syncope. Moreover, echocardiographic signs of RV pressure overload and/or dysfunction, especially with more specific echocardiographic findings such as the McConnel sign, are suggestive for the diagnosis of acute pulmonary embolism and useful for its risk stratification. Three mechanisms have been proposed to explain the McConnell sign. First, in acute pulmonary embolism, the tethering of the right ventricular apex to a contracting and often hyperdynamic left ventricle may account for the preserved wall motion at the apex.[14,15] Second, the right ventricle may be assuming a more spherical shape to equalize regional wall stress when subjected to an abrupt increase in afterload.[16,17] Third, there may be localized ischemia of the right ventricular free wall due to increased wall stress.[15] Noteworthy, these proposed mechanisms become evident when emboli already obstruct a large proportion of the pulmonary vascular bed.

The 60/60 sign has also been described in addition to the McConnell sign; the 60/60 sign combines pulmonary ejection acceleration time <60 milliseconds with a tricuspid regurgitation peak systolic gradient <60 mm Hg is highly suggestive for PE.[18] Mobile right-heart thrombus is another specific but rare feature associated with higher-rate mortality.[19]

Conclusions

Echocardiography may be useful in cases of high-risk pulmonary embolism in which a rapid presumptive diagnosis is required to justify the use of thrombolytic

therapy.[20,21] Those measures might allow a quick diagnosis of pulmonary embolism followed by prompt treatment with anticoagulant, thus improving the patient's short and long-term prognosis.

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