The Importance of Trans-Thoracic Echocardiographic Suprasternal View in the Diagnosis and Treatment Follow-Up of Pulmonary Emboli

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KEY WORDS: pulmonary emboli, echocardiography, diagnosis, thrombolysis

IMAJ 2017; 19: 61–62

The diagnosis of pulmonary emboli (PE) relies on clinical probability and multiple imaging modalities. The role of trans-thoracic echocardiography (TTE) in the diagnostic evaluation of PE is well known and usually focuses on the presence or absence of signs suggesting right ventricular (RV) pressure overload. Nevertheless, TTE may sometimes reveal the PE itself in specific less "popular" views, which could be the first clue to its diagnosis. This is reflected in the patient presented here.

PATIENT DESCRIPTION

A 66 year old female with no cardiac or respiratory history was admitted for a pre-syncope episode upon walking. The event occurred a few days prior to her admission. The patient denied any chest pain or respiratory symptoms before or after the pre-syncope. Upon admission to the emergency room (ER), the patient was hemodynamically stable with no signs of respiratory distress; transcutaneous O_2 room air saturation was 90% and normalized during the stay in the ER. Physical examination was unrevealing and a chest X-ray showed normal cardiac silhouette and clear lungs. A 12 lead electrocardiogram (ECG) showed diffuse mild ST elevation as well as T wave conversions in leads V1-V4**.** Cardiac troponin T was 0.2 ng/ml (normal < 0.04 ng/ml).

A cardiac TTE study revealed normal left ventricular (LV) function, mild RV hypokinesia and no significant valvular disease or any pericardial effusion. A suprasternal view revealed a large mobile mass in the orifice of the right pulmonary artery (PA) [Figure 1A], although this was not seen in the parasternal short axis aortic root view. To confirm the diagnosis of PE, chest computed tomography (CT) was performed revealing a large saddle-shaped PE located within the main PA [Figure 1C] as well as significant RV dilatation (RV/LV diameter ratio 1.3 on four-chamber view). A diagnosis of sub-massive PE according to the 2011 American Heart Association (AHA) scientific statement [3] or intermediate-high risk PE according to the 2014 European Society of Cardiologists (ESC) guidelines [1] was made based on the combination of

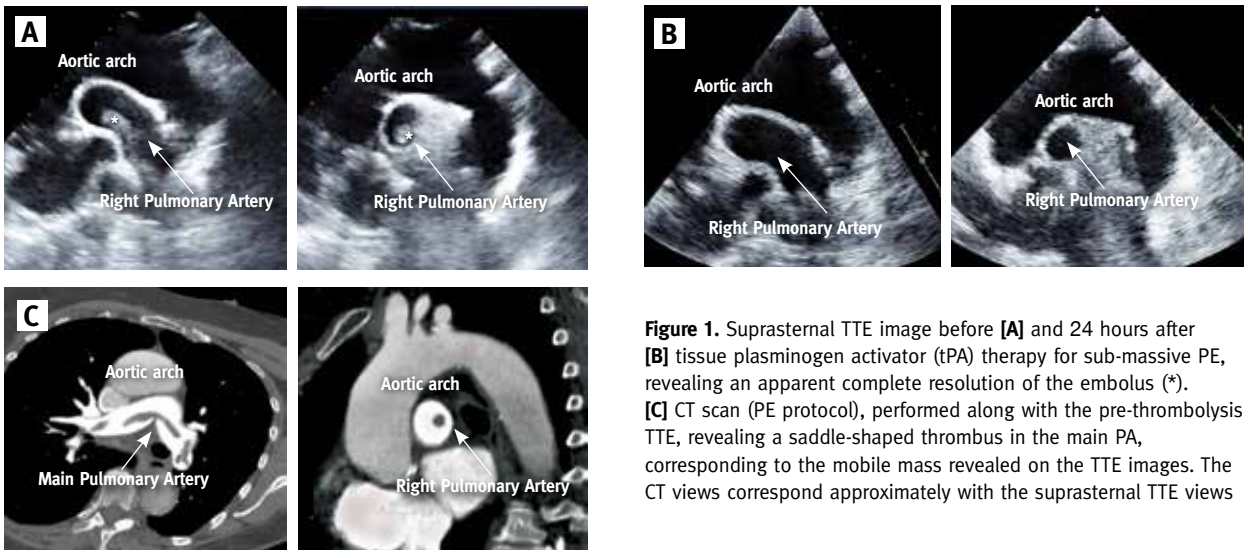

Figure 1. Suprasternal TTE image before **[A]** and 24 hours after **[B]** tissue plasminogen activator (tPA) therapy for sub-massive PE, revealing an apparent complete resolution of the embolus (*). **[C]** CT scan (PE protocol), performed along with the pre-thrombolysis TTE, revealing a saddle-shaped thrombus in the main PA, corresponding to the mobile mass revealed on the TTE images. The CT views correspond approximately with the suprasternal TTE views

the definitive CT imaging results along with the dilated RV and elevated troponin [1-3]. Accordingly, thrombolysis therapy was given using an infusion of 100 mg tissue plasminogen activator (tPA) within 1 hour. Upon treatment, there was no change in the patient's stable clinical status, and no clinical evidence of bleeding, peripheral emboli or hemodynamic instability. On repeat TTE the next morning, there was no sign of the PA mobile mass [Figure 1B], suggesting a rapid and efficient lysis of the clot.

COMMENT

Our case illustrates the importance of a complete TTE examination including both a suprasternal view and a parasternal short axis aortic root view, which are complementary images of the main PA branches and at times may be the only views revealing a large embolus in the right PA and main PA bifurcation, respectively. Our case emphasizes the importance of the suprasternal view for PA clot diagnosis even when such a clot is not seen in all other views. This might be especially true in cases where a potential diagnosis of PE is present but the initial medical history, physical examination and basic laboratory results are all non-diagnostic. In our case specifically, the patient was initially suspected of having acute coronary syndrome (ACS) due to the ECG changes and elevated troponin. The diagnosis of PE was remote given the absence of any predisposing risk factors, the vague episode of presyncope without any chest pain, respiratory symptoms or other signs of hemodynamic instability, and ambiguous ECG changes showing T wave inversion in leads V1-V4 on one hand and a diffuse ST elevation on the other. A D-dimer test might have raised the attention to such a diagnosis, but since the working diagnosis was ACS this test was not done. On the whole, the pre-test probability for PE was low according to both the Wells and revised Geneva scores [1]. Indeed, the TTE upon admission was basically performed to assist in the evaluation of a patient with suspected ACS. A TTE examination without the suprasternal view would not have been informative in our case, whereas this important view enabled a clear diagnosis. Notably, the standard TTE views in our case were suboptimal and did not reveal any significant signs of RV pressure overload. Acknowledging that the CT study was performed due to PE suspicion on the suprasternal TTE view, one might have missed the diagnosis of PE without this view. The discrepancy between the RV dimensions in the TTE and CT imaging studies might be explained by the suboptimal TTE views.

Notably, the PE guidelines recommend TTE for patients with hemodynamic instability where signs of RV pressure overload might justify thrombolysis, while absence of these signs would exclude PE as a cause of the hemodynamic deterioration [1]. Nevertheless, in our case the TTE study helped to diagnose PE in a hemodynamically stable patient with low probability of PE. We stress the importance of a suprasternal view in any patient suspected of PE diagnosis even with a low probability.

The other important aspect of this report relates to thrombolytic therapy in sub-massive or intermediate-high risk PE, which is still controversial. The few clinical trials involving thrombolysis in normotensive PE patients showed a reduction in pulmonary pressure or development of chronic thrombo-embolic pulmonary hypertension, reduced need for escalation to emergency treatment, and reduced short-term hemodynamic collapse, but none has shown mortality benefit and most revealed increased bleeding complications [1-4]. Accordingly, in contrast with the class I recommendations for thrombolysis in massive PE, the guidelines for thrombolysis in sub-massive PE are more restrained. Notably, both ESC [1] and AHA [3] scientific statements suggest using thrombolysis in sub-massive PE patients upon signs of hemodynamic or respiratory decompensation (class IIa), while its routine use is discouraged (class III recommendation). The AHA statement, however, extends their recommendation to sub-massive PE patients who are hemodynamically stable but have imaging and/or laboratory evidence of significant RV strain or myocardial necrosis [3].

We use this case to suggest the efficacy of thrombolytic treatment in a hemodynamically stable patient with moderate RV dysfunction and troponin elevation, revealing a rapid resolution of the clot, as seen by a 24 hour follow-up suprasternal TTE view. Although we show the acute success of thrombolytic therapy, we still do not know its long-term impact on RV function or the patient's long-term clinical status.

Interestingly, both the European and American guidelines do not discuss the importance of syncope as a sign of hemodynamic instability, even if the latter resolved. Many patients with PE present with syncope, which obviously is a symptom of temporary hemodynamic instability. We believe that this should be regarded as an ominous sign for high risk PE even if hemodynamic stability is regained, and should be treated as such.

In summary, this case illustrates the importance of a high degree of suspicion in the diagnosis of PE, even when more common diseases are expected (i.e., ACS). The use of TTE suprasternal view can help with the imaging of a thrombus in the main PA and at times may be the only diagnostic view. The use of thrombolytic therapy in patients with high-intermediate risk or submassive PE is to be considered.

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