

Pulmonary arterial hypertension

Massimo Miniati

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In *Internal and Emergency Medicine*, Wu and coworkers [1] review the topic of pulmonary arterial hypertension. Overall, their article is sound and pertinent; however, some issues deserve comments.

First, the value of chest radiography in the diagnostic process is somewhat overlooked. As pointed out by the authors, a prominent pulmonary artery trunk, or enlargement of the right heart cavities are strongly suggestive of pulmonary arterial hypertension. However, chest radiography also provides valuable information as regards chronic left heart failure, which is often associated with elevated pulmonary artery pressure.

Chronic left heart failure is manifested on the chest radiograph by enlargement of the left heart cavities, dilated upper lobe vessels, and interstitial edema [2]. Therefore, chest radiography should be firmly incorporated in the diagnostic workup of suspected pulmonary arterial hypertension for it offers important clues to differentiate pre-capillary from post-capillary pulmonary hypertension.

Transthoracic echocardiography is often inaccurate in estimating pulmonary artery systolic pressure (PASP). This is clearly borne out by a recent prospective study of 65 patients, suspected of having pulmonary arterial hypertension, who underwent Doppler echocardiography (DE) and right heart catheterization (RHC) within 1 h of the other test [3].

In that study, estimates of PASP by DE are considered “accurate” if they fall within 10 mmHg of the value measured by RHC. In the study, echocardiographic

estimates of PASP could be rated accurately in only 48% of the patients, underestimation occurring in 25%, and overestimation in 27% [3].

There was a significant, positive correlation between DE estimates and actual measurements of PASP at RHC, with a correlation coefficient of 0.66. Nevertheless, the correlation coefficient is simply a measure of an association between two variables, and not of the agreement between two methods of measuring a given variable [4]. Failure to understand this, may lead to serious misconception of the value of any diagnostic method in clinical practice [4].

The inaccuracy of DE in estimating PASP is often due to a poor visualization of the tricuspid regurgitation jet, or because of an unreliable estimation of the right atrial pressure from the compressibility of the inferior vena cava [3].

Thus, even though echocardiography is a useful noninvasive screening tool, physicians should not be overly reliant on DE estimates of PASP in their approach to patients with suspected pulmonary arterial hypertension, or in assessing changes of PASP in response to therapy.

Escalation therapy with dual or multimodal medication regimens is considered standard of care by current treatment guidelines for pulmonary hypertension [1]. In a recent cohort study, 821 patients with established pulmonary arterial hypertension received bosentan as the initial treatment, and were followed up to 3 years [5]. Of the 190 patients who died during follow-up, only 21 (11%) had been escalated to prostanoid therapy.

The fact that 89% of the patients who expired did so without being escalated to prostanoids is of concern. Possible explanations of such an unexpected finding are: (a) inadequate characterization of patients at inclusion; (b) insufficient knowledge of treatment options by the prescribing physicians; (c) reluctance to adhere to treatment guidelines

M. Miniati (✉)
Dipartimento di Area Critica Medico Chirurgica,
Università degli Studi di Firenze, Viale Morgagni 85,
50134 Florence, Italy
e-mail: Massimo.Miniati@unifi.it

due to the perceived complexity or invasive nature of prostanoid delivery.

No matter what the reason might be, it appears that the behavior of the practicing physician is at variance with the recommendations of the clinical investigators. More effective strategies are, therefore, needed to optimize the management of pulmonary arterial hypertension.

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