Stress-Induced Pulmonary Systolic Hypertension in Patients With Scleroderma

To the Editor:

We read with great interest the study of Alkotob et al (July 2006)[1] on pulmonary hypertension (PHTN) in patients with scleroderma. They demonstrate that stress-induced pulmonary systolic hypertension in patients with scleroderma is highly prevalent, 46%, defined by an abnormal rise in pulmonary artery systolic pressure (PASP) > 40 mm Hg during exercise. The question is: are really all these patients a risk population of PHTN?

Knowledge of the risk for the development PHTN is essential. Chang et al[2] observed that among 361 patients undergoing serial echocardiography without initial evidence of PHTN, mild-to-moderate PHTN developed in 25.5%, significantly lower than 46% referred.

We have also determined the prevalence of stress-induced pulmonary artery hypertension in 49 patients with scleroderma. We excluded patients receiving treatment with sildenafil, bosentan, or prostacyclin analogues for severe Raynaud phenomenon in 3 months previous.

In our study, the prevalence was 42% (PASP > 40 mm Hg), 31.7% (PASP > 50 mm Hg), 19.5% (PASP > 60 mm Hg), and 9.8% (PASP > 65 mm Hg), respectively. In this last group, all patients had normal resting pulmonary arterial pressure; we observed a significantly higher prevalence of severe Raynaud phenomenon and diffusion capacity < 80% of predicted. Thus far, two patients (50%) with PASP > 65 mm Hg have acquired resting PHTN within the 1-year period, whereas only two patients (10%) with PASP from 50 to 60 mm Hg at exercise acquired PHTN.

The cut-off of PASP at exercise has not been established. We think that for the definition of PHTN, the value at exercise must be > 40 mm Hg, probably 50 mm Hg, and that this group would be intensely observed for the development of resting PHTN.

Jose Luis Callejas, MD
Eduardo Moreno, MD
Pilar Martin, MD
Lourdes Lopez-Perez, MD
Norberto Ortego, MD
Hospital Clinico San Cecilio
Granada, Spain

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Correspondence to: Jose Luis Callejas, MD, Hospital Clinico San Cecilio, Granada, Spain; e-mail: JLCALLEJA@telefonica.net
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References

1 Alkotob ML, Soltani P, Sheatt MA, et al. Reduced exercise capacity and stress-induced pulmonary hypertension in patients with scleroderma. Chest 2006; 130:176–181

2 Chang B, Schachna L, White B, et al. Natural history of mild-moderate pulmonary hypertension and the risk factors for severe pulmonary hypertension in scleroderma. J Rheumatol 2006; 33:269–274

Response

To the Editor:

We appreciate the remarks of Callejas et al concerning our recent article in *CHEST* (July 2006).[1] The prevalence of a pulmonary artery systolic pressure of > 40 mm Hg in our patients was similar to that in the population described. In order to qualify as a positive response, we agree that patients should produce a minimum value of 40 mm Hg + right atrial pressure during exercise; a less robust response most often indicates pulmonary venous hypertension. For now, we believe it prudent to study all patients whose resting pulmonary artery pressure is normal and whose exercise pressure exceeds 40 mm Hg + right atrial pressure. Currently, we repeat the exercise echocardiogram every 3 to 6 months in those patients.

As noted,[1] several of our patients ultimately progressed to resting pulmonary hypertension. Both of the markers for such disease progression, and the mechanism for stress-induced pulmonary hypertension remain the subject of intense interest and ongoing investigation.

David Silverman, MD
University of Connecticut
Farmington, CT

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Correspondence to: David Silverman, MD, University of Connecticut, Department of Cardiology, 263 Farmington Ave, Farmington, CT 06030; e-mail: silverman@nso1.uchc.edu
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References

1 Alkotob ML, Soltani P, Sheatt MA, et al. Reduced exercise capacity and stress-induced pulmonary hypertension in patients with scleroderma. Chest 2006; 130:176–181

Severe Sepsis and Septic Shock

Should Blood Be Transfused To Raise Mixed Venous Oxygen Saturation?

To the Editor:

We share the sentiments expressed by Otero et al[1] (November 2006) regarding the importance of reducing mortality from severe