
THE AUTHORS REPLY: Arias et al. correctly note that diabetes is an independent risk factor for congestive heart failure. Data from the Cardiovascular Health Study have also shown a significant independent relationship between diabetes and the development of congestive heart failure; most cases of heart failure in that cohort were associated with a normal or near-normal ejection fraction — that is, they were cases of diastolic heart failure. We agree that aggressive control of diabetes, as well as of hypertension, should be considered an important component of the management of heart failure.

Gerard P. Aurigemma, M.D.
William H. Gaasch, M.D.
University of Massachusetts Medical School
Worcester, MA 01655
aurigemg@ummhc.org


TO THE EDITOR: In their review of treatment for pulmonary arterial hypertension, Humbert et al. (Sept. 30 issue) discuss therapeutic strategies with the use of prostacyclin and endothelin-receptor antagonists. Potential future therapies focus on other vasodilators and combination therapy. However, in addition, on the basis of pathophysiological mechanisms, there might be a role for immunosuppressive therapy. Several case reports show that in patients with systemic lupus erythematosus, the use of immunosuppressive drugs in the absence of disease activity results in an improvement of severe pulmonary arterial hypertension. Recently, the role of inflammation in pulmonary arterial hypertension has been described. Moreover, endothelin-receptor antagonists work not only because of their vasodilating effects, but also because of their blockade of the proliferation of smooth-muscle cells. In that respect, they are disease-modifying drugs. The combination of vasodilating, antiproliferative, and immunosuppressive therapies might be the most effective approach. Further studies are warranted to elucidate the role of immunosuppressive therapy in pulmonary arterial hypertension.

Anne-Margriett Huisman, M.D.
Sint Franciscus Gasthuis
3045 PM Rotterdam, the Netherlands
m.huisman@sfg.nl

Simone A. Vreugdenhil, M.D.
Erasmus Medical Center Rotterdam
3000 DR Rotterdam, the Netherlands

Henk C. van Paassen, M.D.
Sint Franciscus Gasthuis
3045 PM Rotterdam, the Netherlands


THE AUTHORS REPLY: We agree with Dr. Huisman and colleagues that inflammatory mechanisms may play an important role in pulmonary arterial hypertension of various origins. Considerable improvements have been reported with the use of antiinflammatory drugs in cases of pulmonary arterial hypertension associated with systemic lupus erythematosus. In addition, patients with idiopathic pulmonary arterial hypertension also have immunologic disturbances, suggesting a possible role of...
inflammation in the pathophysiology of the condition. However, most, if not all, of such patients have no clinical response to antiinflammatory agents, and current therapeutic guidelines therefore do not consider these drugs. Nevertheless, it is widely accepted that antiinflammatory agents should be proposed for patients with pulmonary arterial hypertension and active inflammatory diseases such as systemic lupus erythematosus. Currently approved therapies (endothelin-receptor antagonists and prostacyclin derivatives) have antiinflammatory effects, possibly contributing to the efficacy of these drugs, which do not act as pure vasodilators. Further developments in the management of pulmonary arterial hypertension may indeed involve the use of a combination of drugs with different targets and effects in order to maximize their action on the vasoconstrictive, proliferative, and inflammatory components of the disease process.

Marc Humbert, M.D., Ph.D.
Olivier Sitbon, M.D.
Gérald Simonneau, M.D.
Hôpital Antoine Béclère
92140 Clamart, France
marc.humbert@abc.aphp.fr


Case 29-2004: A Woman with Acute Onset of Chest Pain and Fever

TO THE EDITOR: With reference to the recent Case Record (Sept. 16 issue) about a 75-year-old woman with acute onset of chest pain followed by fever, I would question the decision to initiate empirical levofloxacin treatment after specimens had been obtained for culture. The presentation included fever, systemic symptoms, cardiac symptoms, a murmur, and abnormal results of urinalysis in a patient with known (albeit mild) valve disease. Therefore, I would have thought that endocarditis would be at the top (or near the top) of the differential diagnosis, although the murmur was not typical of mitral regurgitation and no aortic regurgitant murmur was heard. There were no frank urinary symptoms, and the concentration of nitrites in the urine was not reported. With all this in mind, would there not have been an argument for either holding off on antibiotics and obtaining more serial blood cultures or immediately starting empirical treatment for endocarditis, depending on the patient’s clinical state? The antibiotic therapy could then have been rationalized, if necessary, after the appropriate culture results and echocardiographic studies were available.

Andrew R.L. Medford, M.B., Ch.B.
Southmead Hospital
Bristol BS10 5NB, United Kingdom
andrew.medford@bris.ac.uk


DR. CHAE REPLIES: I agree with Dr. Medford that a rigorous approach to the care of a patient with suspected endocarditis should include either serial blood cultures without antibiotic therapy or, if the clinical suspicion is high, initiation of empirical antibiotic treatment after samples for cultures have been obtained. However, as detailed extensively in the discussion of the case presentation, this patient’s visit to the emergency department was precipitated by acute chest pain associated with electrocardiographic changes. The patient was initially afebrile and had no cardinal signs or symptoms to suggest endocarditis. Levofloxacin was started in the emergency department because of the positive results of urinalysis and the potential need for an invasive procedure such as coronary angiography, which made it important to minimize the risk of infection associated with instrumentation. The implications of the patient’s prodromal systemic symptoms were not fully appreciated until after she was admitted, when a fever developed and further clinical assessment suggested that endocarditis was a likely cause.

Claudia Chae, M.D., M.P.H.
Massachusetts General Hospital
Boston, MA 02114