Pulmonary Vasculopathy and Recurrent Pneumothoraces

To the Editor:

I offer the following comments on the vascular pathology in the case reported by Schnader et al in CHEST (November 1996). It is well established that in lung tissue resected from patients with idiopathic spontaneous pneumothorax, the muscular pulmonary arteries frequently show severe intimal fibrosis, as illustrated in Figure 1A, 1C, and 1D. Hemosiderin-laden macrophages are also commonly present. The thick-walled pleural vessels illustrated in Figure 1F and 1G are modified bronchial arteries ("sperrarterien") which are commonly encountered in normal lungs. Figure 1E shows a cholesterol-ester granuloma. It was previously thought that pulmonary hypertension was important in the pathogenesis of pulmonary cholesterol-ester granulomas, but for some years it has been known that they are nonspecific lesions encountered in a wide variety of conditions.

In summary, all the vascular lesions described and illustrated in this case are incidental and are known to occur in idiopathic spontaneous pneumothorax. There is no reason to investigate this patient for pulmonary hypertension. It is important for pathologists to understand that the presence of medial hypertrophy and intimal fibrosis in muscular pulmonary arteries does not necessarily indicate pulmonary hypertension. These intimal and medial lesions may develop as a reaction to chronic inflammation and fibrosis in the adjacent lung tissue. They have no hemodynamic significance.

J. Michael Kay, MD
Department of Laboratory Medicine
St. Joseph's Hospital
Department of Pathology
McMaster University
Hamilton, Ontario, Canada

REFERENCES


To the Editor:

We appreciate Dr. Kay’s comments. We agree that many of the pathologic changes reported in the case presentation are nonspecific, as we point out clearly in our article. We do not disagree with the original report, which documents a “nonspecific” endarteritis. But along the lines of what is most interesting about this case, and as we discuss, it is unclear whether the vascular changes precede the pneumothoraces or the pneumothoraces precede the vascular changes, and whether or not there is a cause-and-effect relationship.

Finally, the scenario in which these pulmonary vasculopathologic changes are uncovered fortuitously is not a common one for clinicians. Although Dr. Kay is of the opinion that such changes are “ incidental” and that “there is no reason to investigate,” it is clear that clinicians are not always comfortable or confident with this approach. In our case, the impetus for an investigation was strengthened by the abnormal diffusing capacity.

Jeff Schnader, MD, CM, FCCP
Dayton VA Medical Center
Wright State University School of Medicine
Dayton, Ohio

Peter B. Terry, MD
The Johns Hopkins Hospital
Baltimore

Adam S. Katz, MD
North Shore University Hospital
Cornell University Medical College
Manhasset, New York

Stephen K. Field, MD, CM
University of Calgary
Calgary, Alberta, Canada

Kenneth M. Moser, MD, FCCP
University of California at San Diego

REFERENCES


