Communications to the Editor

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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

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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 predate 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
Manhattan, New York
Stephen K. Field, MD, CM
University of Calgary
Calgary, Alberta, Canada
Kenneth M. Moser, MD, FCCP
University of California at San Diego

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1 Schnader J, Terry PB, Field SK, et al. Clinical conference on management dilemmas: pulmonary vasculopathy and
Therapeutic Thoracoscopy Under Local Anesthesia

To the Editor:

We read with great interest the report by Nezu et al on thoracoscopic bullectomy performed under local anesthesia for the treatment of spontaneous pneumothorax (January 1997).1 The authors selected patients with no pleural adhesions (by air insufflation and then x-ray), and they tried to identify their bullae preoperatively by using CT scans. They compared their results with those of a historical group in which the same procedure was performed under general anesthesia; they then concluded that the approach using local anesthesia is safe and is associated with a shorter hospital stay.

We would like to raise four points. First, in our experience, it is relatively unusual for recurrent primary spontaneous pneumothorax not to have any adhesions, as the natural history of a ruptured bulla is eventual adhesion to the chest wall. Therefore, if this selection criteria is strictly followed, it seems that the described procedure will be applicable only to a relatively small group of patients. Second, although a CT scan can confirm the presence of apical bullae, it fails to identify ruptured bullae or blebs, which can coexist with unruptured bullae. The former requires careful thoracoscopic examination,2 which is more difficult to carry out in a spontaneously ventilating lung. Third, the authors did not compare subjective or objective pain measurements (by visual analogue scale or analgesic requirements) between the two groups. The reported average postoperative hospital stay of 4.5 days in their local anesthesia group, although less than the 5.8 days reported in their general anesthesia group, is still longer than our result of 3.0 days in over 200 consecutive patients, whose procedures were all performed under general anesthesia.3 Fourth, mechanical pleurodesis and pleurectomy, which are believed to be important components of surgical treatment,4 are difficult to perform under local anesthesia. Besides, this leaves virtually no safety margin if major intraoperative complications do occur.

Although bullectomy under local anesthesia is technically feasible, the claim that this approach is superior to its counterpart performed under general anesthesia with selected one-lung ventilation may be a little premature.

Anthony P. C. Yim, MD, FCCP
M. Bashir Izzat, MD, MCh
Prince of Wales Hospital
Hong Kong

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To the Editor:

We thank Drs. Yim and Izzat for their very pertinent comments on our article (January 1997).1 First, they claimed that recurrent primary spontaneous pneumothorax without any pleural adhesions is relatively unusual. However, we disagree; 56% of our patients with recurrent pneumothorax were preoperatively diagnosed by the air-inflation test to be practically free from adhesions. We have to admit that there might have been minimal adhesions present in such patients, and that our description may have been a little extreme. However, there were many patients with recurrent pneumothorax who were practically adhesion-free. Furthermore, we operated on the patients within a relatively short period after the onset of recurrent spontaneous pneumothorax, which may have influenced the higher ratio of the adhesion-free patients in our series.

As for their second claim, we agree that CT scans fail to identify ruptured bullae or blebs coexisting with unruptured bullae. Although they mentioned the difficulty in locating ruptured bullae in the lung of a patient receiving mechanical ventilation, we can control the expansion and collapse of the lung using a valved port with an insufflation stopcock, with the patient under local anesthesia and sedated, as described in our paper.

In response to their third question, we did not perform subjective or objective pain measurement between the two groups during or after the operation. However, there are no apparent clinical differences between the two groups in terms of complaints of pain. As for the average duration of postoperative hospital stay, our results (4.5 and 5.8 days for the local and general anesthesia groups, respectively) were longer than those obtained by Yim.2 However, there may be many differences in his circumstances and ours, such as social medical insurance systems. Under such conditions, we do not think it is appropriate to simply compare the durations of hospital stay.

We agree with their fourth comment about the importance of mechanical pleurodesis or pleurectomy in the management of spontaneous pneumothorax. We do only fibrin glue pleurodesis in our procedure, and other pleurodesis procedures are hardly required because of the low recurrence rate of 3.1%. Fibrin glue pleurodesis can be easily performed with the patient under local anesthesia and sedated; this was the method we used for simple cases.

Certainly, our procedure is not applicable to all of the patients with spontaneous pneumothorax at this time. However, considering several advantages, such as its minimal invasiveness, this procedure can be used as an alternative for selected patients with uncomplicated spontaneous pneumothorax.

Kunimoto Nezu, MD
Sotchiro Kitamura, MD, FCCP
Department of Surgery III
Nara Medical University
Nara, Japan

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