statements deserve further comment.

Although the noncaseating granuloma has traditionally been regarded as the *sine qua non* pathologic finding for the diagnosis of sarcoidosis, the presence of interstitial pneumonitis has been well described in the literature. Huang et al. studied the open lung biopsy specimens of 81 patients with sarcoidosis and observed both granulomata and interstitial pneumonitis in all cases. Assessment of the intensity of alveolitis (i.e., the accumulation of mononuclear inflammatory and immune effector cells within the interstitium) is now routinely accomplished by the technique of bronchoalveolar lavage. In addition, it has been well established that alveolitis or an interstitial pneumonitis most likely represent the initial lesion of sarcoidosis, preceding granuloma formation. This conclusion is based primarily upon the work of Rosen et al., who demonstrated that interstitial pneumonitis—predominant in 62 percent of 128 open lung biopsy specimens—was inversely related to the density of granuloma.

It is also a known fact that different lesions of sarcoidosis can coexist in the same lung. Crystal et al. have observed that the overall picture of sarcoidosis is a "montage of alveolar-capillary units in various stages of inflammation and derangement." At different times and at different rates, small foci of interstitial pneumonitis may either resolve or go on to granuloma formation. Similarly, granulomata may either resolve or progress to structural derangement and eventual fibrosis.

It is therefore not surprising that Aisner and Albin observed some degree of fibrosis as well as interstitial pneumonitis in their case. Rosen et al. noted some degree of fibrosis in 10 percent of cases in which interstitial pneumonitis was predominant. Huang et al. observed focal or diffuse fibrosis in more than half of their cases, including some with Type I radiographs. Aisner and Albin's contention that their case is unusual is perhaps unjustified.

Finally, the authors' point that the diagnosis of sarcoidosis cannot be excluded by finding only interstitial pneumonitis on transbronchial biopsy is not new. Rosen et al. also concluded that the sampling of small amounts of tissue by transbronchial biopsy may reveal only interstitial pneumonitis if this is the predominant finding and granulomata are scarce.

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REFERENCES


To the Editor:

We appreciate the comments of Drs. Davis and Berkmen, but we disagree with their conclusion that our case is not unusual. What makes our case novel is the presence of diffuse interstitial inflamm-

ation with fibrosis in an advanced case of sarcoidosis. We concur that the earliest lung lesion in sarcoidosis is an alveolitis, and that these inflammatory cells may serve as substrate for the formation of granulomas. However, this alveolitis represents an early (the earliest?) lesion in sarcoidosis and is typically sparse in individuals with advanced disease. Indeed, Rosen postulated that interstitial pneumonitis represented "a very early lesion in the evolution of pulmonary sarcoidosis." These authors further state that in their series, "interstitial pneumonitis was usually focal, with intervening areas of normal-appearing pulmonary parenchyma." Far from being commonplace, only 24 percent in Rosen's study had interstitial pneumonitis predominating; only 32 percent of these showed fibrosis; and less than 20 percent of their group with the radiographic sarcoid stage of our patient had interstitial pneumonitis.

We agree with the assertion that interstitial pneumonitis is seen in sarcoidosis. Our case is unique in that it demonstrates that interstitial pneumonitis with fibrosis can predominate the pathology even in advanced disease, which is not what is reported in the literature. This finding serves to reinforce our messages; namely: 1) that pneumonitis and fibrosis can occur distinct from granuloma formation; 2) that these findings occur in end-stage sarcoidosis; 3) that transbronchial biopsies demonstrating inflammation and fibrosis may be compatible with advanced sarcoidosis; and 4) that this diagnosis should be pursued when clinically suspected despite finding interstitial inflammation and fibrosis.

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To the Editor:

Your attention to detail and accuracy in medical literature is well known. I would like to suggest that the article entitled "The Treatment of Endobronchial Stenosis Using Balloon Catheter Dilatation" (Chest 1986; 90:1145-51) is somewhat misleading in that the casual reader might feel that this is the definitive treatment for such a disease process. However, the abstract and the article detail that use of the YAG laser for definitive treatment of these lesions is an integral part of the procedure and probably should have been reflected in the title.

I am concerned, as you must be, that the noncritical practitioner may simply peruse the journals and take from them that which he feels would enhance his or her practice. I feel that the title of an article should accurately reflect its content and wonder whether or not it might have been more accurate to reflect the use of balloon dilatation as an adjunct to definitive care with the laser for these patients.

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Balloon Dilatation and the Laser

Communications to the Editor

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