High-Resolution CT of Asbestosis and Idiopathic Pulmonary Fibrosis

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OBJECTIVE. We studied high-resolution CT of asbestosis and idiopathic pulmonary fibrosis to determine whether differences—other than the frequency of associated pleural changes—could be discerned between the two diseases.

MATERIALS AND METHODS. High-resolution CT scans of 80 patients with asbestosis and 80 patients with idiopathic pulmonary fibrosis were retrospectively reviewed. Two chest radiologists assessed the type and distribution of parenchymal and pleural abnormalities on high-resolution CT.

RESULTS. Subpleural dotlike or branching opacities (65/80), subpleural curvilinear lines (55/80), mosaic perfusion (39/80), and parenchymal bands (38/80) were more common in patients with asbestosis ($p < 0.0001$). Visible intralobular bronchioles (62/80), bronchiolectasis within fibrotic consolidations (47/80), and honeycombing (61/80) were more common in patients with idiopathic pulmonary fibrosis ($p < 0.0001$). The frequencies of interlobular septal thickening, ground-glass opacities, fibrotic consolidation, and emphysema were similar in both groups. Parenchymal bands and fibrotic consolidation were more commonly seen ($p < 0.05$) in patients with asbestosis associated with pleural disease ($n = 66$) than in patients with asbestosis without pleural disease ($n = 14$). Also, statistically significant differences were noted between high-resolution CT findings of patients with asbestosis without pleural disease and those of patients with idiopathic pulmonary fibrosis, except for parenchymal bands.

CONCLUSION. Specific combinations of high-resolution CT findings strongly suggest either asbestosis or idiopathic pulmonary fibrosis. We found that CT findings that might have represented bronchiolar obstruction in the subpleural region were more prominent in patients with asbestosis than in those with idiopathic pulmonary fibrosis, whereas bronchiolar dilatation was more prominent in patients with idiopathic pulmonary fibrosis than in those with asbestosis.

Asbestosis (i.e., asbestos-induced pulmonary fibrosis) and idiopathic pulmonary fibrosis (i.e., usual interstitial pneumonia) have similar histopathologic appearances and similar radiographic manifestations [1]. Differentiating idiopathic pulmonary fibrosis from asbestosis is important because of legal and compensatory issues. However, differentiating one from the other is often difficult, particularly in patients in whom the extent of occupational asbestos exposure is not clear [2]. In addition, asbestos-exposed persons potentially are subject to the same spectrum of lung diseases as the general population [3].

The identification of these diseases is important because treatments and prognoses for such conditions may differ from those of asbestosis. The onset of asbestosis is gradual, and progression is either very slow or absent. In idiopathic pulmonary fibrosis, however, speed of onset and rate of progression are more variable and can be rapid [4]. In idiopathic pulmonary fibrosis, most patients are treated with steroids, and there is some evidence of additional benefit from cyclophosphamide or azathioprine. A committee of the American Thoracic Society and the European Respiratory Society [5] recommends a combined therapy of corticosteroid and either azathioprine or cyclophosphamide. Novel strategies using antifibrotic agents—such as colchicine, interferon-γ, pirfenidone, and the like—have been advocated, and they are expected to be valuable additions to the array of available treatments [5]. In the case of asbestosis, a cure is rarely possible, leaving the physician with limited means for helping the

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patient. For asbestos workers with more than minimal disease, complete removal from exposure remains mandatory.

High-resolution CT has an increasingly important role in the clinical diagnosis of idiopathic pulmonary fibrosis. Mathieson et al. [6] found that CT changes seen in patients with asbestosis resembled those seen in patients with idiopathic pulmonary fibrosis, although patients with asbestosis also had bilateral pleural thickening. Al-Jarad et al. [7] compared the high-resolution CT findings in patients with idiopathic pulmonary fibrosis and asbestosis and showed that there were differences other than the frequency of the associated pleural changes in patients with each of these diseases. They reported that, in addition to showing pleural disease, high-resolution CT showed that ground-glass opacities were common in idiopathic pulmonary fibrosis and rare in asbestosis, whereas thick, bandlike opacities were common in asbestosis and rare in idiopathic pulmonary fibrosis. However, parenchymal bands are usually associated with extensive pleural disease [8] and, as such, may not be a discriminating factor in patients in whom pleural diseases are not present.

Although these studies described some differences between high-resolution CT findings of asbestosis and idiopathic pulmonary fibrosis, we do not believe that a systematic review of a large number of CT signs in each condition has been previously performed. In our study, we assessed whether one or more of these CT signs can help in differentiating asbestosis from idiopathic pulmonary fibrosis.

Materials and Methods

We studied 80 patients with asbestosis and 80 patients with idiopathic pulmonary fibrosis. Informed consent was obtained from all 160 patients, and the study was approved by our internal review board.

The asbestosis group consisted of 50 men and 30 women who were 41–83 years old (mean ± SD, 61 ± 9 years). Twenty-four of the patients had never smoked, and 56 patients had a history of smoking. The idiopathic pulmonary fibrosis group consisted of 56 men and 24 women who were 41–82 years old (63 ± 9 years). Eighteen of the patients had never smoked, and 62 patients had a history of smoking.

The diagnosis of asbestosis was based on the following criteria: a history of substantial exposure to asbestos; a time interval between exposure and disease detection that was consistent with asbestosis; radiographic evidence of bilateral diffuse irregular opacities (perfusion category ≥ 1/0, according to the International Labour Organization international classification [9]); absence of cardiopulmonary disease that could cause these abnormalities; and the presence of middle to late inspiratory crackles. The 80 patients with asbestosis met all these criteria.

The subjects were derived from a large cohort of textile workers from the Sennan industrial area of Osaka, Japan. Asbestosis has a relatively close association with both the magnitude and duration of exposure to inhaled asbestos. The latency period between the exposure and the discovery of manifestations of the disease is likely to be a minimum of 15 years. In our 80 patients with abestosis, the mean exposure to asbestos dust was 23.2 years (range, 15–35 years), and the mean interval since the first exposure was 29.3 years (range, 20–45 years).

According to the International Labour Organization classification of the radiographic appearances of the pneumoconioses [9], 32 of 80 patients with asbestosis had a pneumoconiosis perfusion category of 1, 35 had a perfusion category of 2, and 13 had a perfusion category of 3. Transbronchial biopsy specimens in 12 patients and autopsy specimens in 14 patients gave evidence of diffuse interstitial fibrosis with asbestos fiber bodies. In another 15 patients in whom bronchoalveolar lavage was performed, asbestos fiber bodies were detected in bronchoalveolar lavage fluid.

The diagnosis of idiopathic pulmonary fibrosis was established by open or video-assisted thoracoscopic lung biopsy in 32 patients and by autopsy in 18 patients. The histologic hallmark and chief diagnostic criterion of idiopathic pulmonary fibrosis is a heterogeneous appearance of the specimen at low magnification, with alternating areas of normal lung, interstitial infiltration, fibrosis, and honeycomb change. Interstitial inflammation is usually mild to moderate, patchy, and composed of an alveolar septal infiltrate of lymphocytes, plasma cells, and histiocytes associated with hyperplasia of type II pneumocytes. The fibrotic zones show temporal heterogeneity with dense acellular collagen and scattered fibroblastic foci. Areas of honeycomb change are composed of cystic fibrotic air spaces that frequently are lined with bronchiolar epithelium and filled with mucin. Smooth muscle hyperplasia is commonly seen in areas of fibrosis and honeycomb change [5].

In the 30 patients with no histologic confirmation of the disease, the diagnosis was based on the clinical criteria described by Turner-Warwick et al. [10] and modified by Xaubet et al. [11]. In these 30 patients, transbronchial biopsy showed morphologic changes suggestive of idiopathic pulmonary fibrosis and no evidence of asbestos fiber bodies; any granulomatous process was ruled out. Histopathologic examinations found no evidence of pneumoconiosis.

None of the patients with idiopathic pulmonary fibrosis had been exposed to asbestos or showed any symptoms or signs suggestive of connective tissue disease or malignancy. None had positive test results for avian precipitins or had received any drug known to induce lung fibrosis.

In the 80 patients with idiopathic pulmonary fibrosis, duration of symptoms before they had undergone CT ranged from 3 to 33 months (mean, 17 months), and the result of their pulmonary function tests was the predicted percentage of forced vital capacity, 77.8% ± 14.7%.

High-resolution CT was performed with a Quantex Plus CT scanner (General Electric–Yokogawa Medical Systems, Milwaukee, WI). All CT scans with 1.5-mm collimation at 20-mm intervals were obtained with the patient supine at maximal inspiration. Prone repositioning was used in patients in whom the posterior lungs were partially obscured by gravity-dependent attenuation, which included 38 of the 80 patients with asbestosis and 27 of the 80 patients with idiopathic pulmonary fibrosis. Scanning extended from the lung apices to below the costophrenic angles. Images were reconstructed with a high-spatial-frequency algorithm. The window settings used were appropriate for lung parenchyma (window width, 1500 H; level, –700 H). None of the patients received an IV administration of contrast medium. No expiratory scans were obtained.

The CT scans were reviewed independently by two chest radiologists who were unaware of the clinical and pathologic data, and the final decisions on CT findings were reached by consensus. The lungs were divided into three equal thirds that were measured from the apex to the base. The reviewers evaluated the scans for the presence of a ground-glass opacity, consolidation, honeycombing, interlobular septal thickening, intralobular interstitial thickening, a centrilobular opacity, a subpleural dotlike or branching opacity, a subpleural line, a parenchymal band, traction bronchiectasis, visible intralobular bronchioles, mosaic perfusion, and emphysema.

Areas of ground-glass attenuation were defined as areas of hazy, increased attenuation that did not obscure the underlying vascular markings. Fibrotic consolidation was defined as consolidation with loss of volume. Consolidation was considered present if the opacities obscured the underlying vessels. If consolidation was present, the presence or absence of bronchiolectasis (an air bronchiologram) within the consolidation was recorded. Honeycombing was defined as an accumulation of cystic spaces with thickened walls. Coarse honeycombing was defined as honeycombing in which the diameter of the honeycomb spaces was greater than 5 mm. Interlobular septal thickening was defined as short lines contacting the pleural surface perpendicularly or as a pattern of multiple polygonal lines, representing thickened interlobular septa. Intralobular interstitial thickening was defined as thickenings of the intralobular interstitium resulting in a fine weblike or netlike appearance to the lobular parenchyma [12].

Distribution of parenchymal opacities was recorded as centrilobular if opacities were identified around peripheral pulmonary arterial branches or 3–5 mm away from the pleura, interlobular septa, or pulmonary veins [13]. Subpleural dotlike or branching opacities were defined as those that arose a few millimeters from the pleura but seldom touched it, appearing as fine branching structures sometimes connected to the most peripheral pulmonary arterial branches [14, 15]. These opacities represent centrilobular opacities in the subpleural zones. Subpleural

lines were defined as linear areas of increased attenuation seen within 1 cm of the pleura and parallel to the inner chest wall [16]. Parenchymal bands were defined as linear, nontapering densities that are 2–5 cm long and extend through the lung to contact the pleural surface [15]. Traction bronchiectasis was defined as bronchial dilatation within areas of a parenchymal abnormality. Bronchial dilatation was considered present if the internal diameter of a bronchus was greater than that of the adjacent pulmonary artery. Visible intralobular bronchioles were considered present if an airway was visualized within 1 cm of the costal pleura. Mosaic perfusion was defined as areas of decreased attenuation with a lobular or multilobular distribution, adjacent to areas of high attenuation [17]. Emphysema was characterized by areas of decreased attenuation, disruption of the vascular pattern, and absence of a well-defined wall.

The observers also noted the extent of the abnormal opacities present in the lungs. The extent of involvement of parenchymal abnormalities was assessed independently for each of the three zones of each lung. The upper, middle, and lower lung zones were scored by visually estimating the extent of disease in each zone. The score was based on the percentage of lung parenchyma that showed evidence of abnormality and was estimated to the nearest 10% of parenchymal involvement. The overall percentage of involvement was calculated by averaging the scores from each of the six lung zones. Zonal predominance was assessed as being upper, middle, lower, or random. The left–right symmetry was also assessed as being predominantly right- or left-sided or equal.

The interpreters separately recorded the presence or absence of pleural plaques and diffuse pleural thickening, using scans obtained at mediastinal window settings (level, 0 H; width, 300 H). Diffuse pleural thickening was defined as a contiguous sheet of pleural thickening that was wider than 5 cm, longer than 8 cm in the craniocaudal plane, and more than 3 mm thick [18].

Statistical analyses were performed with a statistical software package (Statistical Package for the Social Sciences [SPSS]-PC 7.5, version 4.0.5J, SPSS, Chicago, IL). We compared the frequencies of disease and the type of abnormalities seen in the two conditions using either Fisher's exact test or the chi-square test, as appropriate. Results were considered significant when p was less than 0.05.

Results

No significant differences were found between the patients with asbestosis and the patients with idiopathic pulmonary fibrosis in age, sex, or smoking history. Differences between the groups for extent of disease as seen on CT were also not significant. The extent of parenchymal abnormalities revealed on CT scans ranged from 3% to 64% (18% ± 11%) of the lung parenchyma in patients with asbestosis and from 4% to 75% (22% ± 10%) in patients with idiopathic pulmonary fibrosis. All patients with asbestosis and all those with idiopathic pulmonary fibrosis had bilateral parenchymal abnormalities. In the group with asbestosis, vertical distribution of the parenchymal abnormalities led to observation of predominant involvement of the lower lung zones in 78 patients and predominant involvement of the upper lung zones in two patients. In the group with idiopathic pulmonary fibrosis, the vertical distribution led to an assessment of predominant involvement of the lower zone of the lung in 77 patients. No patient was found to have predominant involvement of the upper zone of the lung. In three patients, the upper, middle, and lower lung zones were judged to be equally involved.

The frequencies of parenchymal abnormalities seen in patients with asbestosis and idiopathic pulmonary fibrosis are shown in Table 1. Subpleural dotlike or branching opacities were present in 65 (81%) of the 80 patients with asbestosis and 20 (25%) of the 80 patients with idiopathic pulmonary fibrosis ($p < 0.0001$) (Fig. 1). Subpleural curvilinear lines and parenchymal bands were more common in patients with asbestosis: 55 (69%) of the 80 patients with asbestosis and 22 (28%) of the 80 patients with idiopathic pulmonary fibrosis had subpleural lines (Fig. 2), and 38 (48%) of the 80 patients with asbestosis and three (4%) of the 80 patients with idiopathic pulmonary fibrosis had parenchymal bands ($p < 0.0001$). Visible intralobular bronchioles were more common in patients with idiopathic pulmonary fibrosis (Fig. 3). Mosaic perfusion was present in 39 (49%) of the 80 patients with asbestosis (Fig. 4) but in only nine (11%) of the 80 patients with idiopathic pulmonary fibrosis ($p < 0.0001$). In patients with asbestosis, the size of the pulmonary vessels in the lobular areas with low attenuation decreased. Fibrotic consolidation was seen in 35 patients with asbestosis and in 47 patients with idiopathic pulmonary fibrosis. Bronchiolectasis was seen within the consolidation in all 47 patients with idiopathic pulmonary fibrosis (Fig. 5) but was often not seen within the consolidation in patients with asbestosis ($p < 0.0001$) (Fig. 6).

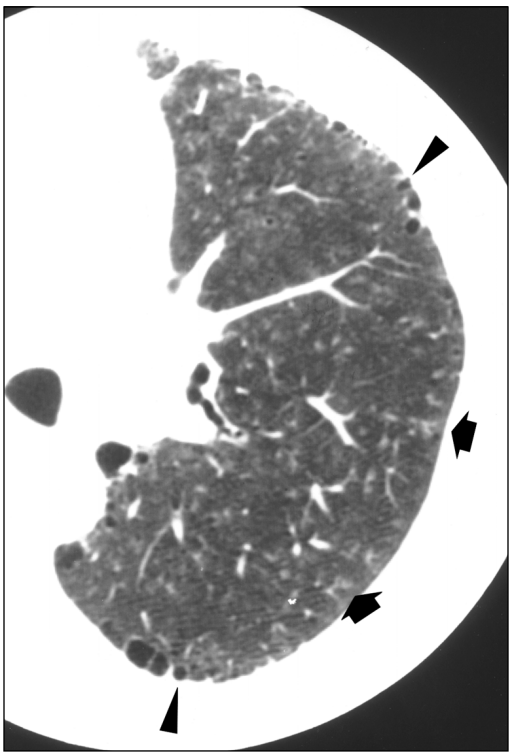
Asymmetric parenchymal abnormalities were found in 20 patients with asbestosis and in 17 patients with idiopathic pulmonary fibrosis. Left-sided predominance was seen in 19 of these 20 patients with asbestosis, and right-sided predominance was seen in one patient with asbestosis. Among the patients with idiopathic pulmonary fibrosis, 10 patients had left-sided predominance, and seven patients had right-sided predominance.

The frequency of high-resolution CT findings for mild parenchymal abnormalities (disease extent visible on CT < 10%) and more extensive abnormalities (disease extent visible on CT ≥ 10%) was examined. Sixteen patients with asbestosis and 10 patients with idiopathic pulmonary fibrosis had mild pa-

TABLE 1 **Comparison of Parenchymal Abnormalities Seen on CT Scans of Patients with Asbestosis and Patients with Idiopathic Pulmonary Fibrosis**

CT Finding	Patients with Asbestosis (n = 80)		Patients with Idiopathic Pulmonary Fibrosis (n = 80)		p
	No.	%	No.	%	
Irregular interlobular septal thickening	70	88	69	86	NS
Intralobular interstitial thickening	55	69	78	98	< 0.0001
Subpleural dotlike or branching opacity	65	81	20	25	< 0.0001
Centrilobular opacity	11	14	2	3	< 0.02
Ground-glass opacity	76	95	79	99	NS
Honeycombing	27	34	61	76	< 0.0001
Coarse honeycombing (> 5-mm diameter)	7	9	28	35	< 0.0001
Traction bronchiectasis	55	69	76	95	< 0.0001
Fibrotic consolidation	35	44	47	59	NS
Bronchiolectasis within consolidation	11	14	47	59	< 0.0001
Visible intralobular bronchioles	16	20	62	78	< 0.0001
Subpleural line	55	69	22	28	< 0.0001
Parenchymal band	38	48	3	4	< 0.0001
Mosaic perfusion	39	49	9	11	< 0.0001
Emphysema	8	10	15	19	NS

Note.—NS = not significant.



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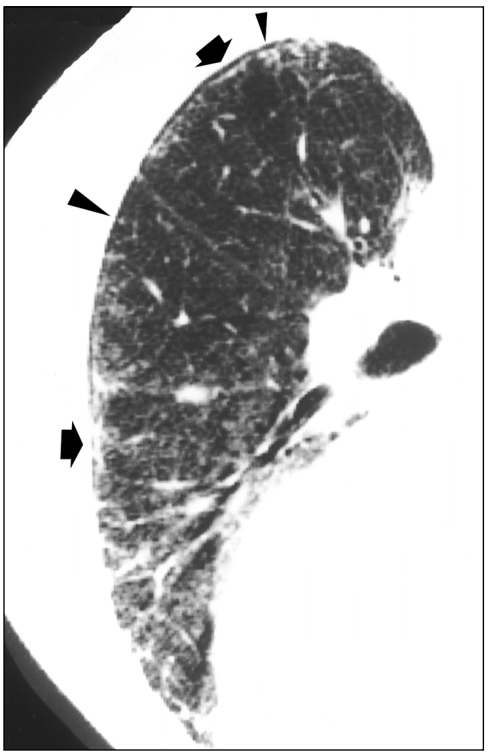

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Fig. 1.—65-year-old man with asbestosis. High-resolution CT scan shows subpleural dotlike or branching opacities (*arrows*) located a few millimeters away from pleura. Note paraseptal emphysema (*arrowheads*).

Fig. 2.—54-year-old man with asbestosis. High-resolution CT scan obtained with patient prone shows subpleural lines (*arrows*) parallel to inner chest wall. Note subpleural dotlike opacities (*arrowheads*).

renchymal abnormalities. No significant difference between asbestosis patients with mild parenchymal abnormalities and asbestosis patients with more extensive abnormalities was found except for intralobular interstitial thickening and mosaic perfusion. In idiopathic pulmonary fibrosis, no significant difference was evident between patients with mild parenchymal abnormalities and patients with more extensive parenchymal abnormalities except for interlobular and intralobular interstitial thickening, honeycombing, and traction bronchiectasis. Patients with extensive asbestosis were significantly more likely than patients with milder cases of the disease to have intralobular interstitial thickening (49/64 vs 6/16) and mosaic perfusion (36/64 vs 3/16). Patients with extensive idiopathic pulmonary fibrosis were more likely than patients with milder cases of the disease to have honeycombing (58/70 vs 3/10), traction bronchiectasis (70/70 vs 6/10), and interlobular (63/70 vs 6/10) and intralobular (70/70 vs 8/10) interstitial thickening.

The combinations of frequent findings seen on high-resolution CT scans were selected for each patient. A combination of subpleural dots and subpleural lines was found in 49 (61%) of the 80 patients with asbestosis and in 10 (13%) of the 80 patients with idiopathic pulmonary fibrosis. A combination of subpleural dots, subpleural lines, and parenchymal bands was found in 28 (35%) of the 80 patients with asbestosis; however, this combination was found in only one (1%) of the 80 patients with idiopathic pulmonary fibrosis. A combination of subpleural dots, subpleural lines, parenchymal bands, and mosaic perfusion was found in 17 (21%) of the 80 patients with asbestosis and in none of the 80 patients with idiopathic pulmonary fibrosis. A combination of visible bronchioles and honeycombing was found in 50 (63%) of the 80 patients with idiopathic pulmonary fibrosis, and in nine (11%) of the 80 patients with asbestosis. A combination of visible bronchioles, bronchiolectasis within consolidation, and honeycombing was found in 28 (35%) of the 80 patients with idiopathic pulmonary fibrosis and in only two (3%) of the 80 patients with asbestosis.

Pleural disease was found in 66 (83%) of 80 patients with asbestosis. Forty-six patients with asbestosis had pleural plaques, and 43 patients with asbestosis had diffuse pleural thickening. Twenty-three patients with asbestosis had both pleural plaques and pleural thickening. There was no significant difference between the patients with asbestosis but no pleural disease and patients with asbestosis with pleural disease, except for parenchymal band and fibrotic consolidation. Parenchymal bands were found in three (21%) of 14 patients with asbestosis without pleural disease and 35 (53%) of 66 patients with asbestosis with pleural disease. Parenchymal bands were found in 33 (77%) of 43 patients with diffuse pleural thickening. Fibrotic consolidation was found in 26 (60%) of 43 patients with diffuse pleural thickening. Parenchymal bands and fibrotic consolidation were significantly more common in patients with diffuse pleural thickening.

In patients with asbestosis without pleural disease, subpleural dots, subpleural lines, and mosaic perfusion were more common and bronchiolectasis within consolidation, visible intralobular bronchioles, and honeycombing were less common. A combination of subpleural dots and subpleural lines was found in eight (57%) of 14 patients with asbestosis without pleural disease. A combination of subpleural dots and mosaic perfusion was found in eight (57%) of the 14 patients with asbestosis without pleural disease. A combination of subpleural dots, subpleural lines, and mosaic perfusion was found in five (36%) of 14 patients with asbestosis without pleural disease. A combination of visible bronchioles and honeycombing was found in two (14%) of the 14 patients with asbestosis without pleural disease, and a combination of visible bronchioles and bronchi-

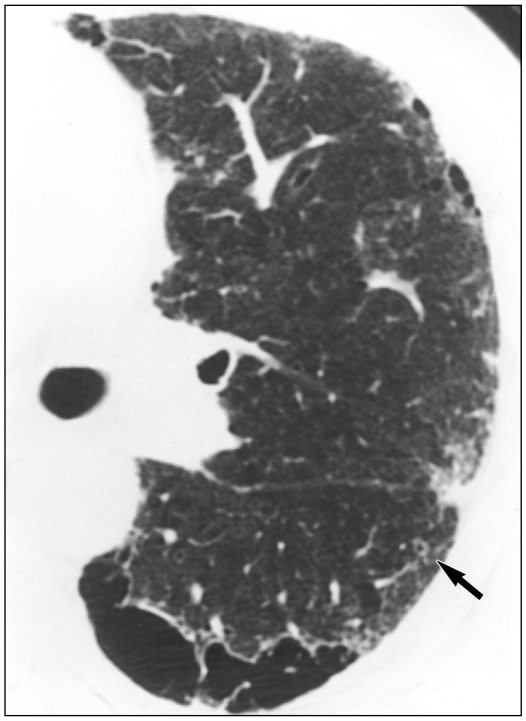


Fig. 3.—58-year-old man with idiopathic pulmonary fibrosis. High-resolution CT scan shows intralobular bronchiole. Note dilated bronchiole (*arrow*) in subpleural region.

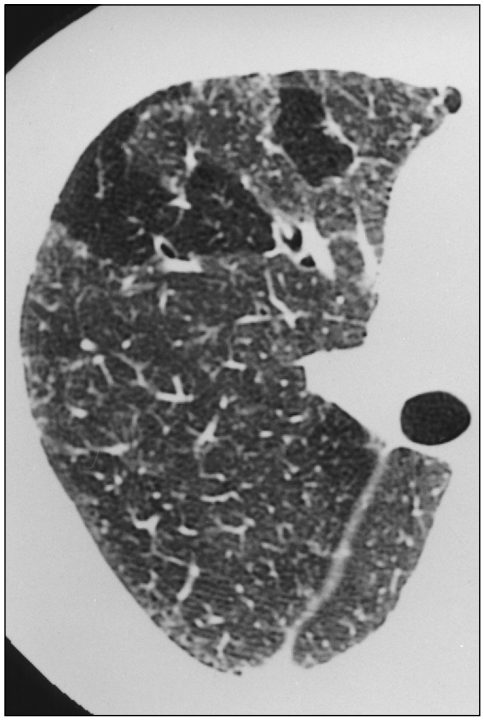


Fig. 4.—High-resolution CT scan obtained in 81-year-old man with asbestosis shows mosaic perfusion.

olectasis within consolidation was not found in any of the 14 patients.

Pleural disease was found in three (4%) of the 80 patients with idiopathic pulmonary fibrosis. These three patients had diffuse pleural thickening and no pleural plaques. In these three patients, parenchymal bands were found.

Discussion

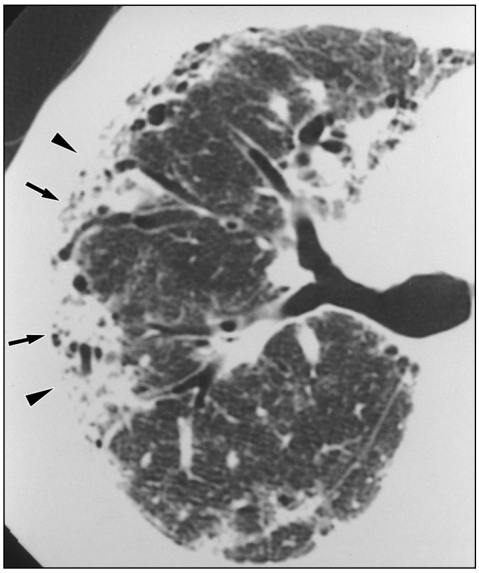
Physicians must frequently diagnose pulmonary asbestosis in the absence of a histopathologic assessment of lung tissue. High-resolution CT can assist in the assessment of patients with equivocal findings and is gaining favor as a screening tool. Similarly, in diagnosing idiopathic pulmonary fibrosis, the trend is toward avoiding invasive diagnostic biopsy procedures in patients with a clinical presentation and CT findings typical of idiopathic pulmonary fibrosis [19]. High-resolution CT has an increasingly important role in the clinical diagnosis of asbestosis and idiopathic pulmonary fibrosis.

The presence of pleural disease in asbestosis has been used as the prime differentiating radiographic finding. In a study of chest radiographic findings, 25% of South African miners with pulmonary fibrosis had no associated pleural plaques on chest radiographs. Approximately 20% of the patients with parenchymal fibrosis had no accompanying pleural thickening [20]. However, pleural plaques are not always present in asbestosis, and in their absence, asbestosis and pulmonary fibrosis may be indistinguishable on chest radiography [7].

In our study, the most important difference between idiopathic pulmonary fibrosis and asbestosis (apart from pleural disease) was the finding of centrilobular opacities in the subpleural region. The subpleural dotlike or branching opacities correspond to centrilobular opacities in the subpleural region. Subpleural dotlike or branching opacities were found not only in the mild cases of asbestosis but also in the advanced cases. In the advanced cases of asbestosis, these opacities were found in the less severely diseased regions of the lung. They were rare in idiopathic pulmonary fibrosis, and instead of dotlike opacities, dilated bronchioles (visible intralobular bronchioles) were often found in the involved subpleural centrilobular region in patients in the idiopathic pulmonary fibrosis group (Fig. 7). These findings could be identified only on high-resolution CT using thin-section collimation. A previous study has shown that in patients with asbestosis, the subpleural dotlike or branching opacities correspond histopathologically to peribronchiolar nodular fibrosis with subsequent involvement of the alveolar ducts [14].

Moreover, dilated bronchioles (i.e., bronchiolectasis) within consolidation were often found in patients with idiopathic pulmonary fibrosis, whereas in patients with asbestosis, fibrotic consolidation was often present but usually unaccompanied by bronchiolectasis. In idiopathic pulmonary fibrosis, bronchiolectasis within consolidation has been shown histologically to be caused by 1- to 2-mm-diameter dilated bronchioles surrounded by airless fibrotic lung tissue [21]. In asbestosis, fibrotic consolidation was more common in asbestosis with diffuse pleural thickening than in asbestosis without pleural disease. These may represent areas of trapped lung that can appear to be classic rounded atelectasis or lenticular opacities.

We found that mosaic perfusion—presumably due to air trapping—was more common in asbestosis than in idiopathic pulmonary fibrosis. Mosaic perfusion is seen in patients with extensive bronchiolar obstruction and is caused by shunting of blood away from poorly ventilated regions of lung [17, 22]. In our patients, the size of the pulmonary vessels in the lobular low-attenuation areas decreased. Diffuse ground-glass attenuation with lobular sparing (relatively low attenuation) can be differentiated from lobular low attenuation resulting from air trapping or vascular lung disease on the basis of the size of the centrilobular core structures: no difference is seen between the size of these structures and those of the surrounding lung in cases of diffuse ground-glass attenuation with lobular sparing,



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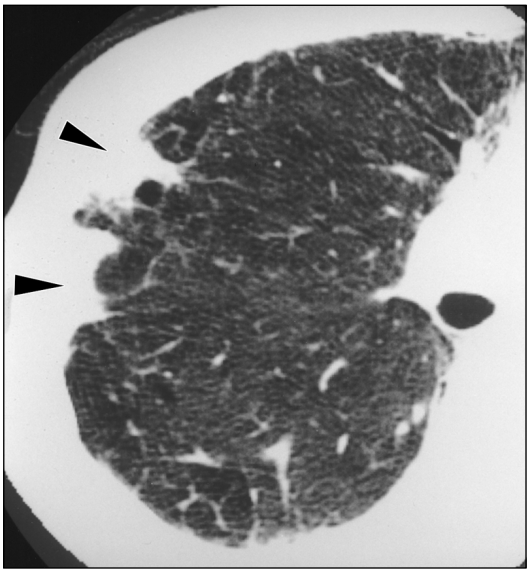

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Fig. 5.—64-year-old man with idiopathic pulmonary fibrosis. High-resolution CT scan shows bronchiolectasis within consolidation. Note fibrotic consolidation (*arrowheads*) in subpleural region. Also note dilated bronchioles, indicating bronchiolectasis (*arrows*) within consolidation.

Fig. 6.—69-year-old woman with asbestosis. High-resolution CT scan of asbestosis shows fibrotic consolidation (*arrowheads*) seen in the subpleural region. Air bronchiogram and air bronchiologram are not seen within consolidation.

whereas the centrilobular core structures are smaller than those of the surrounding lung in cases of lobular low attenuation resulting from air trapping or vascular lung disease 16, 21]. Air trapping is best seen on expiratory CT scans; however, in our study, expiratory CT scans were not used. If the expiratory scans were used, detection of air trapping may be enhanced.

Subpleural lines were more frequently found in patients with asbestosis than in patients with idiopathic pulmonary fibrosis. Subpleual lines may be caused by different pathophysiologic mechanisms. In asbestosis, the subpleural lines have been shown histologically to be caused by peribronchiolar fibrotic thickening combined with the flattening and collapse of the alveoli due to fibrosis [16]. A confluence of honeycomb cysts can also result in an irregular subpleural line. Kubota et al. [23] proved that the subpleural lines represent platelike atelectasis in the corticomedullary junction of the lung.

Parenchymal bands are often accompanied by diffuse pleural thickening [8, 24]. Gevenois et al. [8] reported that the presence of parenchymal bands and diffuse pleural thickening in the same cluster suggests that these bands are related to visceral pleural fibrosis rather than to interstitial fibrosis. In our study, parenchymal bands were more common in patients who had asbestosis with diffuse pleural thickening. Parenchymal bands are usually associated with extensive pleural disease and, as such, may not be a discriminating factor in patients without pleural disease.

We found statistically significant differences between findings on high-resolution CT scans of patients with asbestosis and those on the scans of patients with idiopathic pulmonary fibrosis in intralobular interstitial thickening, subpleural dotlike or branching opacities, subpleural lines, parenchymal bands, bronchiolectasis within consolidation, isolated dilated bronchioles, traction bronchiectasis, mosaic perfusion, and honeycombing. A combination of subpleural dots, subpleural lines, and parenchymal bands or mosaic perfusion was more frequent in patients with asbestosis. A combination of visible bronchioles, bronchiolectasis within consolidation, and honeycombing was more frequent in patients with idiopathic pulmonary fibrosis. These observations suggest that asbestosis and idiopathic pulmonary fibrosis have different high-resolution CT findings in the subpleural secondary lobules of the lungs, especially in the bronchiolar region. Bronchiolar obstruction in the subpleural region is more prominent in asbestosis than in idiopathic pulmonary fibrosis, whereas bronchiolar dilatation is more prominent in idiopathic pulmonary fibrosis than in asbestosis. It has been shown that exposure to asbestos may cause airway disease, giving rise to the development of airflow obstruction [24–26].

Our study has some limitations. Thirty-nine of the patients in the asbestosis group evidently fit the criteria for clinical diagnosis of asbestosis. The patients in the asbestosis group may have had other diseases, even though clinical examinations were carefully evaluated. Some of the patients with clinically diagnosed asbestosis may have actually had idiopathic pulmonary fibrosis. We obtained clinical and radiologic follow-up data that had been collected for more than 5 years for all 39 patients without pathologically proven asbestosis. The clinical progression was slow or had stabilized over time in all 39 patients, which is compatible with asbestosis. However, some patients without histologic confirmation of asbestosis

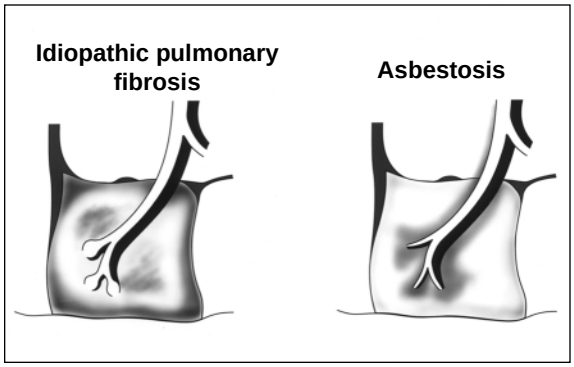

Fig. 7.—Diagram of high-resolution CT findings in subpleural secondary lobules of lungs in idiopathic pulmonary fibrosis and asbestosis. Shaded areas represent prominent fibrosis.

may have a small amount of idiopathic pulmonary fibrosis. Not all patients underwent prone scanning. Some degree of dependent opacity in the lungs can mimic or obscure disease, and it may influence subjective measurement of the extent of disease by the interpreters. Scanning was performed with the patient prone if the posterior lungs were partially obscured by gravity-dependent attenuation. Gravity-dependent opacity was not seen on many CT scans obtained at maximal inspiration with the patient supine.

In conclusion, subpleural dotlike or branching opacities, curvilinear lines, and bandlike opacities are significantly more common in patients with asbestosis, whereas honeycombing, visible bronchioles, and bronchiolectasis within consolidation are significantly more common in patients with idiopathic pulmonary fibrosis. Ground-glass opacities and interlobular septal thickening are common in both diseases. Mosaic perfusion resulting from air trapping is more common in asbestosis than in idiopathic pulmonary fibrosis. These differences are true of asbestosis without pleural disease. These CT findings suggest that bronchiolar obstruction in the subpleural region was more prominent in asbestosis than in idiopathic pulmonary fibrosis, whereas bronchiolar dilatation was more prominent in idiopathic pulmonary fibrosis than in asbestosis. These differences may be related to the underlying morphology of each process. High-resolution CT may be helpful in differentiating asbestosis from idiopathic pulmonary fibrosis.

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