資料 5

PLEURAL DISEASE

Edited by

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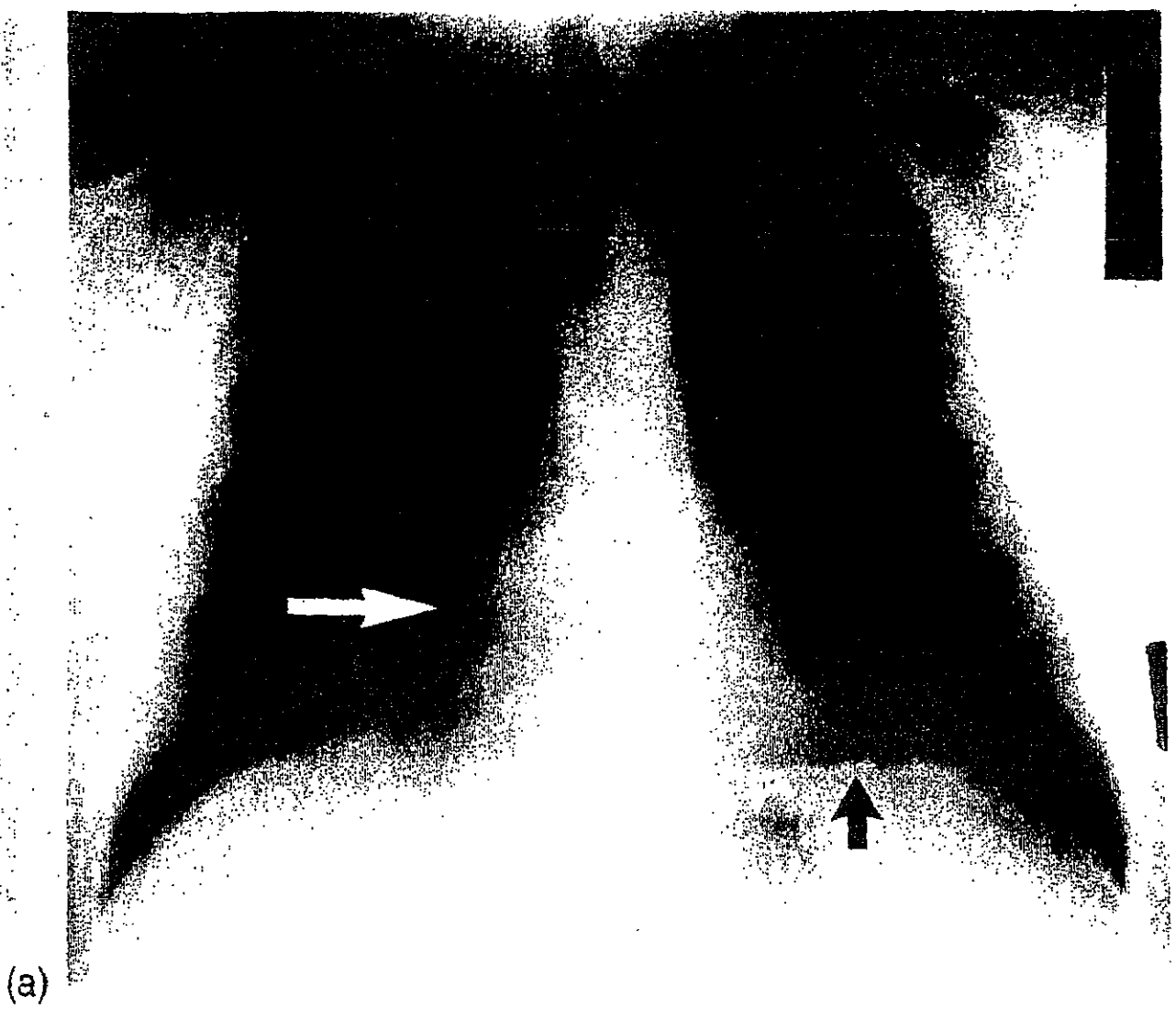

Figure 2 A former insulator with pleural plaques in multiple locations. (a) The chest radiograph shows a thin calcified plaque along the right heart border (white arrow) and overlying the dome of the left diaphragm (black arrow). (b) The chest CT also shows the delicate pleural plaque in the mediastinal pleura overlying the heart (small white arrow), but the plaque in the right paraspinous region on chest CT (large white arrow) was not visible on chest x-ray.

Chest CT scan has proven to be more sensitive than chest radiography for detecting pleural plaques and for discriminating between pleural fibrosis and extrapleural fat (23–25,59). Gevenois et al. (25) performed a conventional and high-resolution CT scan (HRCT) on 159 asbestos-exposed workers with normal chest radiographs. Of these workers, 37.1% demonstrated pleural thickening on CT scan. Conventional CT proved superior to HRCT in detecting these plaques.

Simple circumscribed asbestos-related pleural plaques usually do not produce clinical symptoms. Often they are discovered incidentally during the clinical evaluation of unrelated health problems or during participation in a screening program. Despite their subclinical presentation, pleural plaques are associated with statistically significant pulmonary function abnormalities. Most

(b)

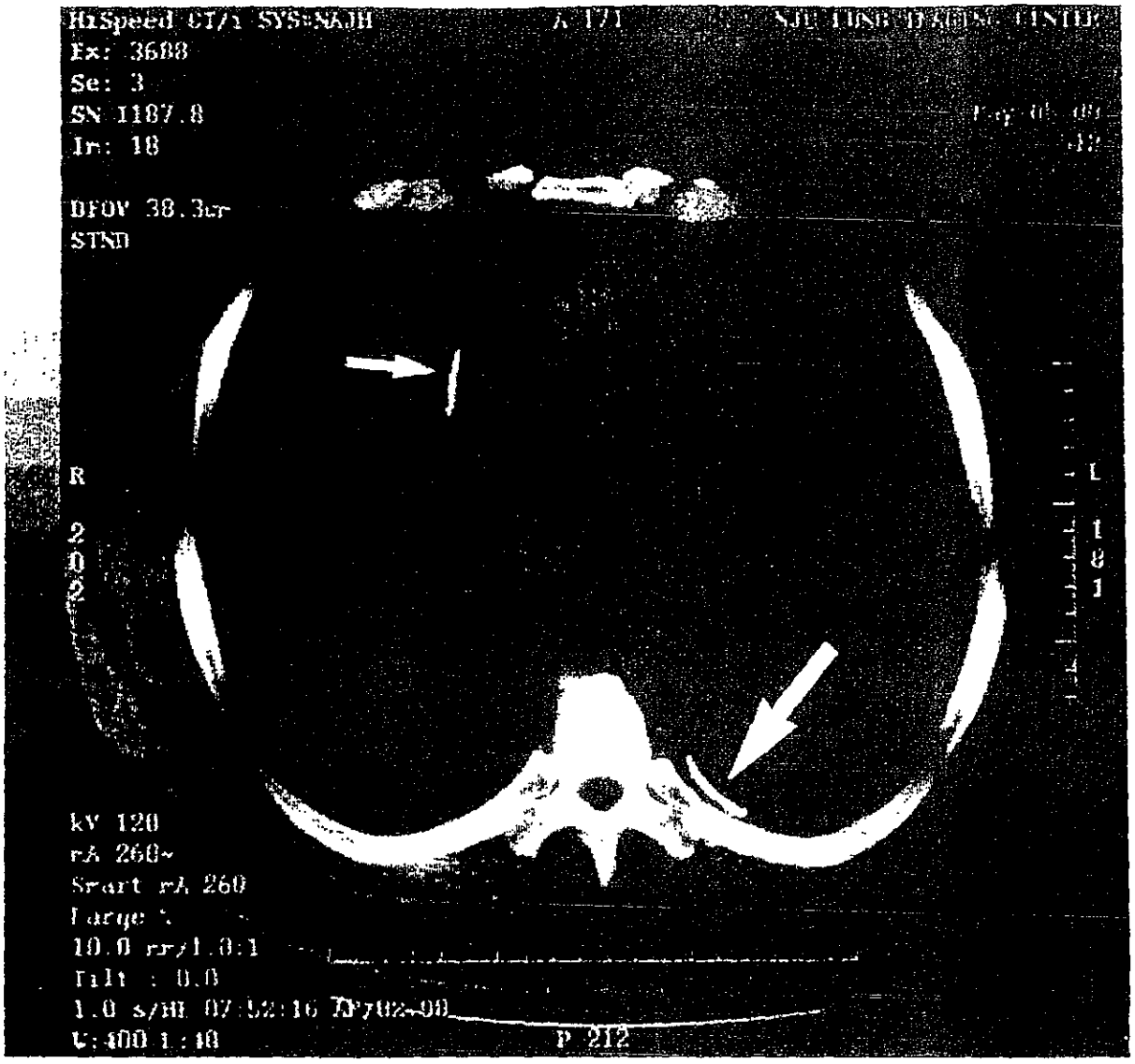

Figure 2 Continued.

consistently they lead to a reduction of the forced vital capacity (FVC) (14,60). Pleural plaques have also been associated with airflow limitation (61–65) While this can be partially attributed to the prevalence of tobacco use in asbestos-exposed cohorts, reports in nonsmokers (62) suggest an independent asbestos-related mechanism responsible for airflow limitation. The most plausible scenario is that physiological airflow limitation is a reflection of pathologically apparent, but radiographically occult peribronchiolar fibrosing alveolitis, the early tissue response to inhalation of asbestos fibers (66).

D. Treatment

No specific treatment is needed for circumscribed pleural plaques. However, since they are a marker of exposure and as such are associated with the risk of developing asbestosis and malignancy, regular follow-up of affected individuals is prudent. Recently much attention has focused on low-dose spiral CT as a lung

cancer–screening tool (67–69) motivated by persistently poor 5-year survival rates (70). Asbestos-exposed workers are a well-defined high-risk group in which this screening tool has great potential. An expert panel recently met to review the advances in radiology and screening of asbestos-related disease (71). They concluded that data available do not justify broad-based lung cancer screening in asbestos-exposed cohorts. For now, the decision to screen with low-dose spiral CT must be made on a case-by-case basis.

Regular follow-up visits also offer opportunities to emphasize the importance of smoking cessation and to assist with achieving this, if necessary, and to assure updated immunization records especially for the influenza vaccine and Pneumovax.

V. Diffuse Pleural Thickening

A. Epidemiology

Diffuse pleural thickening has been recognized only recently as a distinct asbestos-related entity. For many years it was considered to be part of the spectrum of parenchymal asbestosis (48). On the other hand, it was often not clearly distinguished from circumscribed parietal pleural plaques. It is often touted as the sequel of an asbestos-related benign pleural effusion (72–75). Diffuse pleural thickening is not as specific for asbestos exposure as bilateral partially calcified pleural plaques are, since it has also been associated with other disorders, including parapneumonic exudative effusions, hemothorax, collagen vascular disease, drug exposure, especially bromocriptine (76), and Dressler's syndrome. The incidence of diffuse pleural thickening is thought to be significantly lower than that of pleural plaques. This is supported by a study conducted by Hillerdal et al. (77), who followed 891 cases with pleural thickening due to asbestos exposure and observed that 84 individuals (approximately 10%) developed diffuse pleural thickening over time. Schwartz et al. examined the chest radiographs of 1211 sheet metal workers and concluded that 260 (21.5%) had developed circumscribed pleural plaques, while again a smaller proportion, only 74 (6.1%), suffered from visceral pleural fibrosis. McLoud et al. (74) could not confirm a significantly lower incidence of diffuse pleural thickening compared to circumscribed pleural thickening when they reviewed chest radiographs of 1373 asbestos-exposed individuals. They found that 10% of the cohort had diffuse pleural thickening and only 16.5% had circumscribed pleural thickening. Some of these differences likely relate to how diffuse pleural thickening is defined by the different investigators or related to the type of asbestos fiber. De Klerk et al. studied workers exposed to crocidolite and found more diffuse pleural thickening than plaques (78).

Diffuse pleural thickening incidence increases with time since first exposure. It is associated with asbestos fiber burden levels that are intermediate between those of levels associated with pleural plaques and those of asbestosis (79–82).

B. Pathogenesis

In contrast to circumscribed pleural plaques, diffuse pleural thickening affects the visceral pleura and typically covers a much larger surface area (Fig. 3). It initially forms in the posterior and posterolateral portions of the lower visceral pleura. With time, it evolves and extends into the costophrenic angles and apices. Diffuse pleural thickening is usually bilateral, but can occur unilaterally. Adhesions between the two pleural sheaths are common. Microscopically, the visceral pleura is replaced by a layer of dense collagenous tissue with a basket-weave pattern reminiscent of that found in parietal pleural plaques. Asbestos fibers and bodies can be recovered from the pleuroparenchymal tissue, especially in the vicinity of the pleural thickening (31).

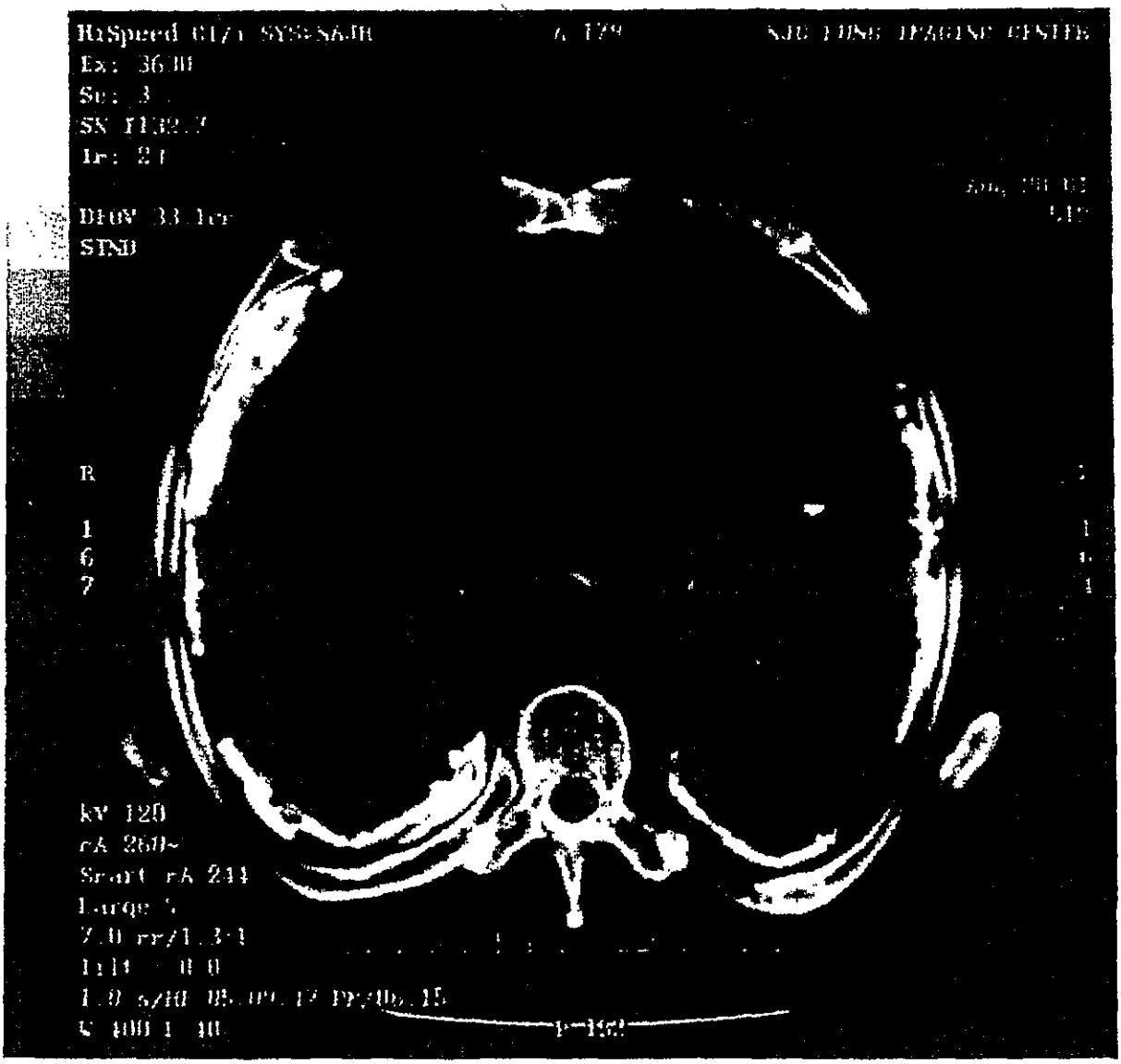

Figure 3 Chest CT depicting extensively calcified thick pleural rind extending around most of the circumference of the lung typical of diffuse pleural thickening.

The pathogenesis of diffuse pleural thickening is not precisely known. However, passage of asbestos fibers into the pleural space via lymphatics with a subsequent inflammatory response is also thought to play a role in diffuse pleural thickening. It is not known why some individuals develop circumscribed plaque while others develop diffuse pleural thickening.

C. Clinical Presentation

In contrast to simple parietal plaques, diffuse pleural thickening is often associated with respiratory symptoms. Dyspnea on exertion represents the most common complaint. In a study by Yates et al. 61 out of 64 asbestos-exposed workers with diffuse pleural thickening complained of breathlessness with exertion (83). Occasionally pleuritic chest pain occurs, most likely due to pleural adhesions in those with diffuse pleural thickening (83,84). Sometimes the pain mimics angina (85).

The physical exam in patients with diffuse pleural thickening can reveal reduced chest expansion, dullness to percussion when the pleural peel has reached significant thickness, and sometimes crackles on auscultation due to concomitant parenchymal fibrosis. Al Jarad and colleagues noted that crackles could be heard in the absence of CT evidence of asbestosis in up to 40% of subjects with diffuse pleural thickening (86).

The radiological features of asbestos-related diffuse pleural thickening have recently been reviewed (82,87). The diagnosis of diffuse pleural thickening on chest radiograph relies on the obliteration of one or both costophrenic angles (74). Based on CT scan, Lynch et al. defined diffuse pleural thickening as a "continuous sheet of pleural thickening more than 5 cm wide, more than 8 cm in craniocaudal extent, and more than 3 mm thick" (88). CT scan often detects fibrous strands, or "crow's feet," extending from the thickened pleura into the lung parenchyma. High-resolution CT scan is superior to chest radiograph in demonstrating the extent of the pleural process. It allows much better visualization of the often-involved paraspinous regions of the pleura that are otherwise obscured by mediastinal structures on chest radiographs. CT scan is also superior in distinguishing between pleural thickening and extrapleural fat.

Several studies have shown that diffuse pleural thickening impairs lung function. The most consistent findings are a decrease in FVC, total lung capacity (TLC), diffusing capacity (DLCO), and exercise tolerance (83,89). In the study by Yates et al. (83) of 64 patients with diffuse pleural thickening, FEV_1 was reduced to 62% and FVC to 77% of predicted. TLC was 71% and DLCO 74% of predicted. Similar results were reported by Kee et al. who studied 53 asbestos-exposed individuals exposed in shipyards or in the construction trades. In this study, the FVC was reduced to 68% of predicted, with a mean DLCO of 72% of predicted (90). Neither study reported DLCO corrected for lung volume, which in the absence of concomitant asbestosis would be expected to be normal.

Interestingly, Al Jarad et al. showed in 20 patients that severity of disease by CT and chest radiograph scores correlated well with the extent of their pulmonary impairment (91). Schwartz et al. also demonstrated in 60 sheet metal workers with asbestos-related pleural fibrosis that the greater the volume of pleural fibrosis derived from a three-dimensional reconstructed thoracic HRCT image, the lower the total lung capacity (92).

D. Treatment

Treatment options for those with diffuse pleural thickening and pulmonary impairment are very limited. Attempts have been made at freeing the lung with decortication, but results have been disappointing (77,93). Supportive treatment is often the best option available. Intercurrent respiratory infections should be treated aggressively. Oxygen therapy is necessary for those with hypoxemia at rest or with exertion. The importance of smoking cessation should be stressed. Immunizations are warranted.

VI. Benign Asbestos Pleural Effusion

A. Epidemiology

The epidemiology of asbestos-related benign pleural effusions mirrors that of asbestosis and the other forms of asbestos-related lung and pleural disease. Risk is associated with the same forms of inhaled asbestos that have been linked to asbestosis, asbestos-related lung cancer, mesothelioma, and other pleural disorders. Described in the 1960s (94–100), the so-called benign pleural effusion may in fact portend development of other forms of asbestos-related disease, including pleural fibrosis and possibly mesothelioma (73). In one of the largest studies of prevalence and incidence, Epler and colleagues observed 34 effusions among 1135 workers exposed in a variety of industries, including shipyards, fireproofing product manufacture, and paper mills. The prevalence was dose-related, ranging from 0.2% for peripherally exposed individuals to 7% among those with most severe exposure. It is the most common asbestos-related condition in the first 20 years after exposure, with incidence of 9.2 effusions per 1000 person-years for those exposed at the highest levels and 0.7 effusions per 1000 person-years for those with the least exposure. The effusions can occur as soon as 5 years after first exposure and have almost always occurred within the first 20 years, although studies differ in their estimates of latency, ranging from a mean of 12 to 30 years. In the Epler study, pleural effusions were five times more likely to occur in asbestos-exposed individuals compared with a nonexposed control group, including effusions related to mesothelioma and lung cancer (73). In light of the rarity of benign pleural effusions in the general population, asbestos exposure should be considered whenever an unexplained exudative effusion is detected. The studies of prevalence and incidence may, in fact, have underestimated the true frequency of asbestos effusions, since many may remain subclinical, preceding development of pleural fibrosis (77).