Asbestos-Related Pleural Disease and Asbestosis: A Comparison of CT and Chest Radiography

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High-resolution CT (HRCT) has the ability to demonstrate both asbestos-related pleural disease and parenchymal abnormalities consistent with asbestosis. The role of CT in the diagnosis of asbestosis can be defined by comparing it with radiography. We evaluated 60 men who had a history of occupational exposure to asbestos and whose outside chest radiographs were considered abnormal. Chest radiographs (inside films) and HRCT were performed in all patients at our institution and were interpreted independently by experienced radiologists. Outside film results were compiled from the submitted reports. The final conclusion regarding the interpretation of the radiologic examinations was determined by consensus when disagreements existed. Positive predictive values (the likelihood that a positive report is correct) for pleural disease were: outside films 56%, inside films 79%, HRCT 100%. The positive predictive values for parenchymal disease were: outside films 51%, inside films 83%, HRCT 100%.

The addition of HRCT to chest radiography is most useful in eliminating false-positive diagnoses of asbestos-related pleural disease caused by subpleural fat and false-positive diagnoses of parenchymal asbestosis in patients with extensive plaques or emphysema obscuring lung detail. The interpretation of chest radiographs in patients exposed to asbestos is often extremely difficult and subjective, and we recommend that positive findings (except calcified plaques) be confirmed with HRCT.

The ability of CT to reliably differentiate subpleural fat from asbestos-related pleural disease is well known [1, 2]. Two recent studies have documented the characteristic HRCT findings in parenchymal asbestosis [3, 4]. The exact role of HRCT vs chest radiography in the clinical evaluation of patients exposed to asbestos has not been extensively assessed. We report the results of a prospective study of the relative efficacy of chest radiography and HRCT in evaluating patients with suspected asbestos-related disease.

Materials and Methods

Sixty men (average age, 58 years) suspected of having an occupational lung disease made up the study group. Fifty-five patients were referred because of a history of occupational asbestos exposure of at least 1-year duration and outside chest films interpreted as showing asbestos-related pleural disease with or without parenchymal asbestosis. The remaining five all had a similar history of occupational asbestos exposure and were referred because of outside films showing pleural disease and suspected mesothelioma (two), interstitial lung disease with a history of either exposure to chlorine gas (one) or occupational asthma (one), and interstitial lung disease with suspected lung cancer (one).

Chest films were obtained at our institution (inside films) in all cases and consisted of posteroanterior, lateral, and oblique views in 50 patients and posteroanterior and lateral views in the remaining 10.

HRCT was performed in all patients with either a Siemens DR3 or a GE 9800. With the Siemens, a 2-mm scan thickness was used at 30-mm intervals from apex to left atrium, followed by 12-mm intervals to the diaphragm; other parameters were a 7-sec scan time, 720 projections, and a strong edge-enhancement algorithm. With the GE, a 1.5-mm scan

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thickness was used with a 2-sec scan time, 360 projections, and a bone algorithm. The initial 40 patients were scanned supine with prone repositioning when the lower lungs were partially obscured by gravity-dependent density. The last 20 were scanned in the prone position only, since it became apparent that the latter technique always significantly improved posterior lower lung detail because of less respiratory motion and anterior shift of dependent density.

Outside film results were compiled from the reports submitted with the films. None of the outside interpreters were B-readers (physicians certified by the National Institute of Occupational Safety and Health as expert in the interpretation of chest radiographs of patients exposed to dusts); 75% were pulmonary physicians, and 25% were either radiologists or occupational medicine physicians. Outside films were interpreted as showing asbestos-related pleural disease in 57 patients and as positive for interstitial fibrosis (asbestosis) in 38 patients. Inside films were all blindly interpreted and were scored (according to International Labour Office criteria) by an experienced chest radiologist B-reader. The CT scans were all blindly interpreted by one of the authors who was experienced in chest CT. All cases with disagreements between outside film, inside film, and/or HRCT interpretations were resolved by consensus of the aforementioned radiologists assisted by a pulmonary physician B-reader, and a final radiologic diagnosis was reached. HRCT was not considered infallible in this process (avoiding artificial designation of HRCT as the gold standard), since all radiologic data were evaluated side by side as in routine clinical consultation. Agreement as to the likely radiologic diagnosis and the cause for the diagnostic errors was reached in all but one case (inside films were subtly positive for interstitial disease, while the HRCT was normal). The final radiologic diagnosis was the same as the diagnoses made with outside film, inside film, and HRCT when there was agreement, but when there was disagreement, it was the consensus diagnosis.

Inside films were considered positive for interstitial disease when they scored 1/1 or higher. Similarly, we considered our HRCT findings to be positive only when they were judged unequivocally to be abnormal. There is evidence that 1/0 is found too often on the radiographs of patients who have not been exposed to asbestos (especially smokers) to be a useful discriminator [5, 6]. The profusion score is less important in this clinical context than is the qualitative judgment as to whether interstitial disease compatible with asbestosis is present [6].

Interstitial disease was diagnosed on HRCT when one or more of the following were present: nondependent lines or dense bands not representing vessels extending to the pleural surface (usually, but not always, in the region of a plaque); subpleural curvilinear densities and/or honeycombing, most prominent in the posterior lower lobes [3, 4, 7–11]. These findings are most often found in the posterior lower lobes and correspond to the distribution of abnormalities at pathologic examination [12]. The lines and dense bands extending to the pleural surface most likely represent fibrotic, thickened interlobular septa [4, 12]. The subpleural curvilinear densities seem to represent peribronchiolar fibrosis and alveolar collapse with fibrosis [3]. They are characteristic of parenchymal asbestosis but may be seen in other interstitial diseases.

Alterations in normal density gradients, inhomogeneity of density, and parenchymal distortion were considered nonspecific since they are present in patients with chronic obstructive pulmonary disease [13]. Questionable findings (corresponding to 1/0 or 0/1) were considered negative.

Unfortunately, no pathologic confirmation of diagnosis was obtained in this series, but the above radiologic criteria have been established by previous pathologic correlation [3, 7–10). HRCT findings described in usual interstitial pneumonia (fibrosing alveolitis) can be transposed to parenchymal asbestosis because the chief pathologic distinction between the former and the latter is the histologic demonstration of asbestos bodies and/or fibers in the latter [3, 12, 14].

The final diagnosis of asbestos-related pleural disease was primarily based on the final radiologic diagnosis. The final diagnosis of parenchymal asbestosis was based on the following criteria: (1) evidence of occupational exposure to asbestos based on the patient's work history and/or the presence of radiologic pleural disease; (2) bilateral late inspiratory or paninspiratory basal crepitations; (3) relevant lung function abnormalities; (4) bilateral basal interstitial fibrosis as the final radiologic diagnosis; and (5) rigorous exclusion of simulative disease and its causes. If at least numbers 1, 4, and 5 were present, a diagnosis of parenchymal asbestosis was reached. In the absence of 4, a combination of 1, 2, 3, and 5 were considered sufficient [6, 14–16]. Note that the diagnosis of parenchymal asbestosis almost always depends primarily on a radiologic appearance compatible with interstitial fibrosis, and measurable impairment of lung function or physical disability need not be present [6, 14].

Radiologic studies were considered true or false positive or true or false negative for pleural disease and parenchymal disease separately. This assignment was based on agreement or disagreement of the blinded interpretation with the final diagnoses, which were all "clinical" since there was no pathologic material.

Results

Thirteen patients (22%) had a final diagnosis of asbestos-related pleural disease without parenchymal involvement, and two patients (3%) had a final diagnosis of parenchymal asbestosis without pleural disease. Nineteen patients (32%) had a final diagnosis of both asbestos-related pleural disease and parenchymal asbestosis. Six of the 21 patients with parenchymal asbestosis had restrictive pulmonary functions (four of the six had crepitations); five of 21 had obstructive changes only; and in 10 patients pulmonary function tests were normal. Of the 15 patients with parenchymal asbestosis and no restrictive pulmonary functions, eight had crepitations. The remaining seven had both inside film and HRCT diagnoses of interstitial fibrosis. Twenty-four patients (40%) had no evidence of asbestos-related pleural disease or interstitial lung disease, and two patients (3%) had other interstitial lung diseases (sarcoid and coal-worker's pneumoconiosis). Thirty-one of the 52 patients with a history of cigarette smoking had clinical evidence of obstructive airways disease.

Inside films were interpreted as showing asbestos-related pleural disease in 38 patients (16 had calcified pleural plaques, all true positive) and as showing parenchymal asbestosis in 23 patients. Four patients who were thought to have parenchymal asbestosis on inside films had a profusion score higher than 1/1. One of these was false positive.

HRCT showed asbestos-related pleural disease in 31 patients (18 with calcification) and parenchymal asbestosis in 21 patients.

All 17 false-positive outside film diagnoses of pleural disease for which both inside films and HRCT had true-negative diagnoses were caused by subpleural fat. The eight false-positive diagnoses of asbestos-related pleural disease on both outside and inside films were caused by subpleural fat in seven and intercostal muscle in one (Fig. 1). The 14 false-positive outside film diagnoses of parenchymal asbestosis for

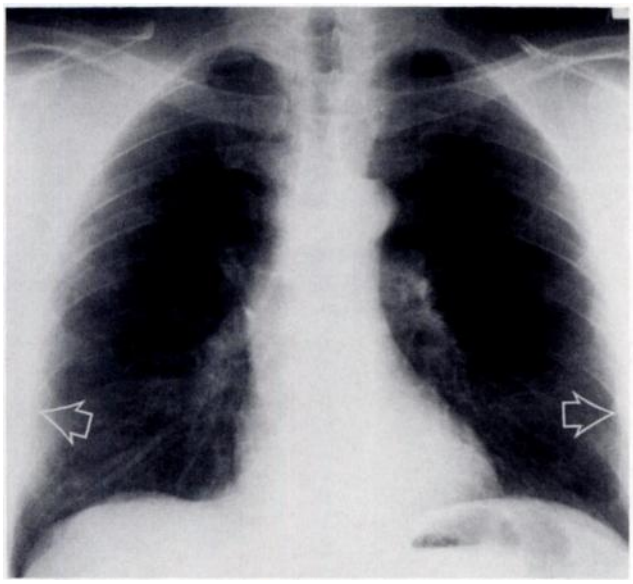
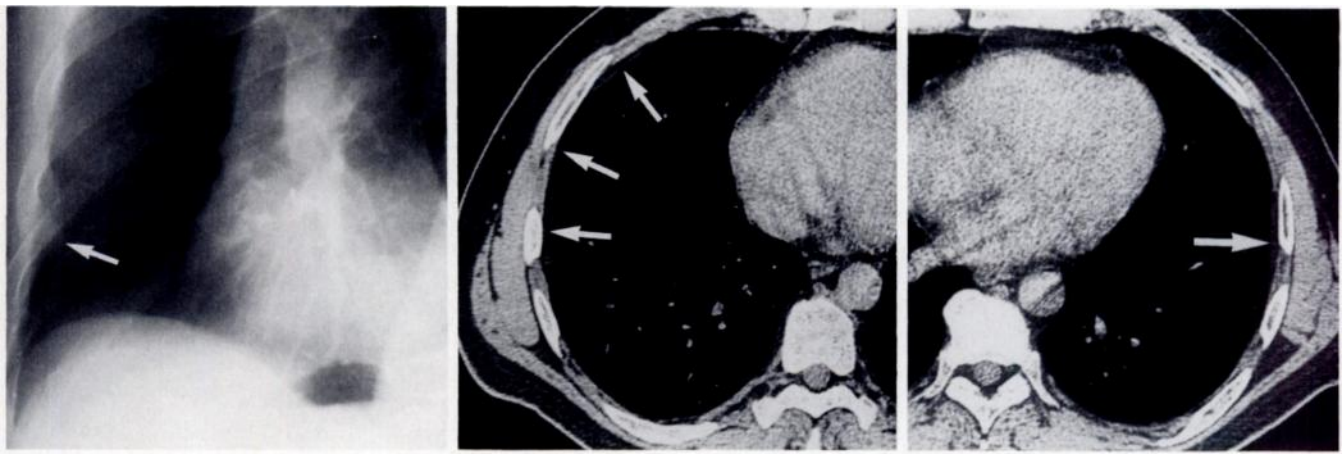


Fig. 1.—Pleural thickening noted on outside and inside films proved to be subpleural fat on high-resolution CT.

***A*, Bilateral slightly asymmetric "pleural thickening" (*arrows*).**

***B*, Oblique film shows region of maximal "plaque" thickness (*arrow*).**

***C* and *D*, High-resolution CT at level of maximal "pleural thickening" shows bilateral asymmetric subpleural fat deposits (*arrows*). Subpleural fat has same radiolucency as subcutaneous fat. Pleural thickening would have same density as muscle.**



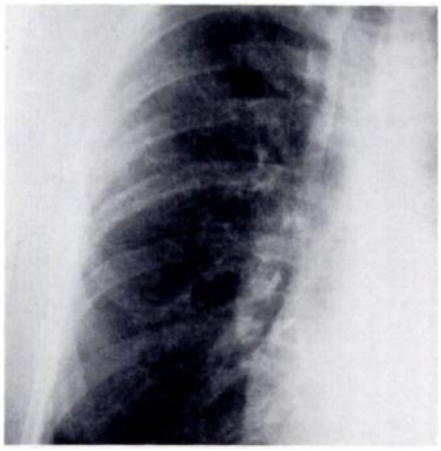
B C D

which both inside films and HRCT were true-negative were caused by prominent vessels, obscuration of the lung by plaques en face, chronic obstructive pulmonary disease, bronchiectasis, or scarring from surgery or old tuberculosis. The four false-positive diagnoses of parenchymal asbestosis on both outside and inside films were caused by walls of bullae (emphysema) in two (Fig. 2) and focal parenchymal scarring, possibly from previous infection, in two.

There were no false-positive HRCT findings for either pleural or parenchymal disease (in all cases in which HRCT was positive and inside films negative, the chest radiologist agreed that the HRCT was correct).

In only two cases did HRCT depict parenchymal asbestosis not suspected on either outside or inside films. The findings were subtle linear densities adjacent to plaques (Fig. 3). There were two false-negative inside films for pleural disease caused by subtle diaphragmatic plaques (Fig. 4) and one false-negative HRCT scan due to a solitary calcified plaque that was missed because it was in between the slices. There was one case in which outside and inside films were positive for parenchymal asbestosis and HRCT was negative, and consensus could not be reached.

Additional diagnoses made on HRCT that were not made on either outside or inside films were emphysema in seven patients, bronchiectasis in two patients, and benign effusion in one patient thought to have mesothelioma on the basis of outside films. The results including sensitivity, specificity, accuracy, and positive and negative predictive values [17] are summarized in Table 1. Because positive outside films were required for entry into the study, the positive predictive values are the most significant parameter, and a percentage t-test of significance was applied to them. The differences between outside and inside films, outside films and HRCT, and inside films and HRCT for both pleural and parenchymal disease were significant with $p < .05$, $p < .01$, and $p < .025$, respectively.



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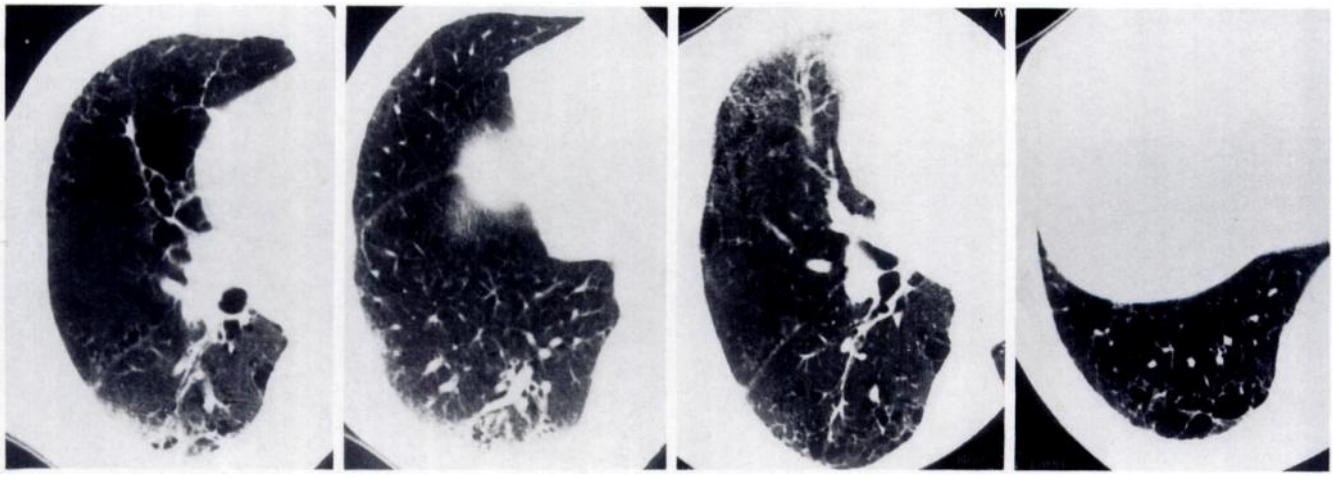


B C D E

Fig. 2.—Misdiagnosis of asbestosis on outside and inside films caused by emphysema (right side only shown; abnormalities were bilateral).

***A*, Increased lung markings.**

***B* and *C*, Supine high-resolution CT shows extensive parenchymal destruction and bullae anteriorly (*B*). Parenchymal destruction at posterior bases was continuous with cephalad bullae. Posterior bases are somewhat obscured by gravity-dependent density (*B* and *C*).**

***D* and *E*, Prone high-resolution CT at nearly corresponding levels shows bullae and extensive parenchymal destruction consistent with emphysema and no interstitial disease. A detailed work history revealed no definite asbestos exposure and 40 pack-years of cigarette smoking.**

A more confident diagnosis was frequently possible on HRCT as compared with inside films, even when all three studies were tabulated as being in agreement. This was especially true in cases with prominent subpleural fat, nonspecific increased parenchymal markings, or extensive plaques obscuring the lungs. The confidence factor was difficult to quantitate.

Discussion

The specificity of outside films was low, indicating that normal subjects would not be properly identified; the positive predictive values of outside films indicated that a positive diagnosis of pleural or parenchymal disease would be correct in only approximately 50% of patients. Needless anxiety is apt to be created if a patient exposed to asbestos has his or her radiographs interpreted by an inexperienced reader. Because of selection bias, we cannot establish the true frequency of false-negative diagnoses on outside film interpretation, and outside film sensitivity may be artificially high. Similarly, outside film specificity for pleural disease is artificially low because only a few of the final diagnoses were negative.

Chest radiographs interpreted by an expert in evaluating asbestos-related disease (inside films) have high sensitivity, specificity, and negative predictive value. It therefore appears that most normal subjects can be correctly identified at relatively low cost and that most existing lesions will be detected. However, a comparison of outside and inside films and HRCT in a group of exposed patients with negative outside films would best assess negative predictive value, sensitivity, and the screening value of HRCT. Since the positive predictive value for both pleural and parenchymal disease is about 80%, confirmation by HRCT of any abnormalities other than calcified plaques seen on radiographs is recommended. Our results indicate that HRCT will be especially useful in eliminating false-positive diagnoses of noncalcified plaques caused by subpleural fat and prominent intercostal muscles and false-positive diagnoses of parenchymal disease in patients with emphysema or extensive plaques obscuring parenchymal detail. HRCT is not needed merely to confirm the presence of calcified pleural plaques. HRCT is marginally more sensitive than inside films for both pleural and parenchymal disease and does not create additional false-positive findings.

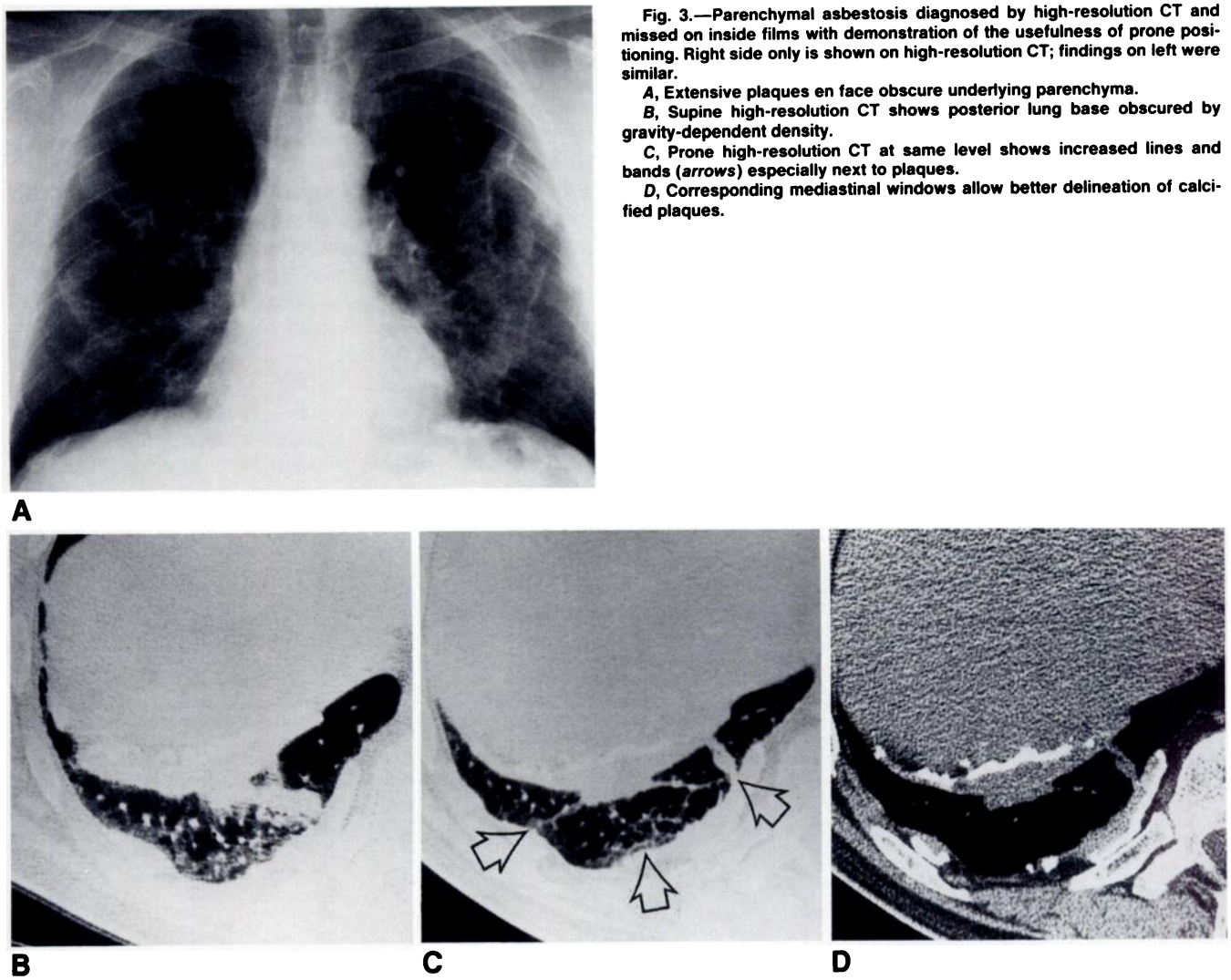


Fig. 3.—Parenchymal asbestosis diagnosed by high-resolution CT and missed on inside films with demonstration of the usefulness of prone positioning. Right side only is shown on high-resolution CT; findings on left were similar.

A, Extensive plaques en face obscure underlying parenchyma.

B, Supine high-resolution CT shows posterior lung base obscured by gravity-dependent density.

C, Prone high-resolution CT at same level shows increased lines and bands (*arrows*) especially next to plaques.

D, Corresponding mediastinal windows allow better delineation of calcified plaques.

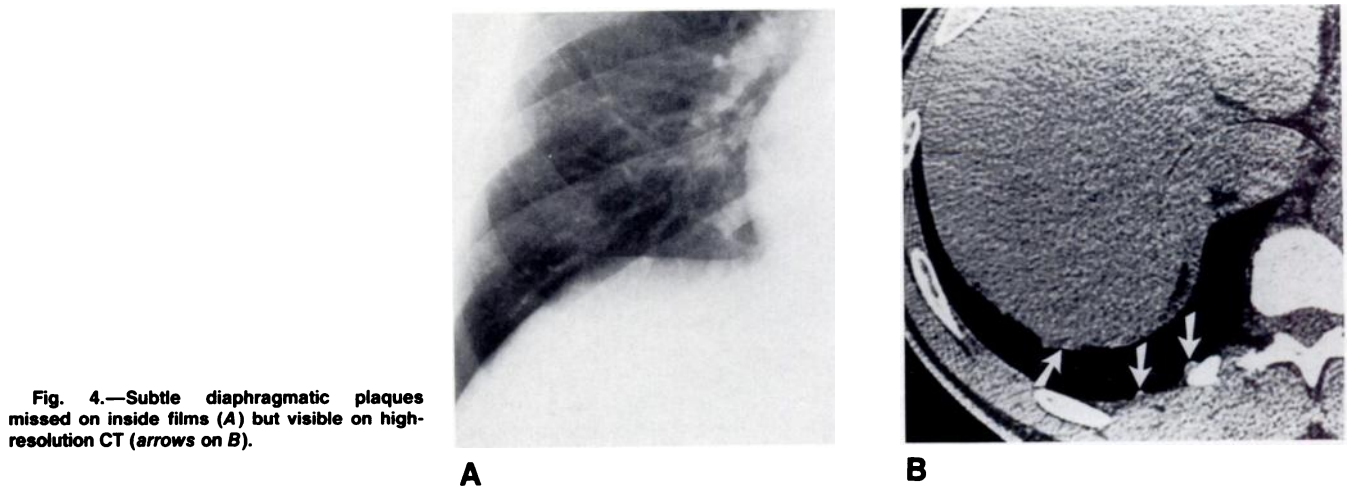


Fig. 4.—Subtle diaphragmatic plaques missed on inside films (*A*) but visible on high-resolution CT (*arrows* on *B*).

TABLE 1: Statistical Evaluation of Outside Films, Inside Films, and HRCT for Diagnosis of Asbestos-Related Disease

Type of Study	TP	FP	TN	FN	% Sens.	% Spec.	% Acc.	% PPV	% NPV
Pleural Disease									
OSF	32	25	3	0	100	11	58	56	100
ISF	30	8	20	2	94	71	83	79	91
HRCT	31	0	28	1	97	100	98	100	97
Parenchymal Disease									
OSF[a]	19	18	20	2	90	53	66	51	91
ISF[a]	19	4	34	2	90	89	90	83	94
HRCT[b]	21	0	38	0	100	100	100	100	100

Note.—TP = true positive; FP = false positive; TN = true negative; FN = false negative; Sens. = sensitivity = 100 × TP/(TP + FN); Spec. = specificity = 100 × TN/(TN + FP); Acc. = accuracy = 100 × (TP + TN)/(TP + TN + FP + FN); PPV = positive predictive value = 100 × TP/(TP + FP); NPV = negative predictive value = 100 × TN/(TN + FN); OSF = outside films; ISF = inside films; HRCT = high-resolution CT. See Gelfand and Ott [17] for statistical methods.

[a] [1 indeterminate (+)].

[b] [1 indeterminate (−)].

In terms of the patient's management, because there is no effective treatment for asbestos-related pleural disease or asbestosis, the ability of HRCT to pick up subtle disease is not important, except perhaps for purposes of compensation for the worker. More widespread use of HRCT for determining compensation should decrease the frequency of unjust compensation due to false-positive interpretations of radiographs.

Since cigarette smoking is prevalent in this population, the ability of HRCT to differentiate between emphysema, bronchiectasis, and interstitial disease, as well as to delineate their relative extent when they coexist, is often useful [8, 13, 18]. HRCT may be even more useful when it is performed in a nonblinded fashion and targeted to radiographically questionable regions.

Finally, HRCT frequently gives the radiologist increased confidence in the final diagnosis, especially in patients with prominent subpleural fat, nonspecific increased parenchymal markings, or extensive plaques obscuring the underlying lungs (Fig. 5).

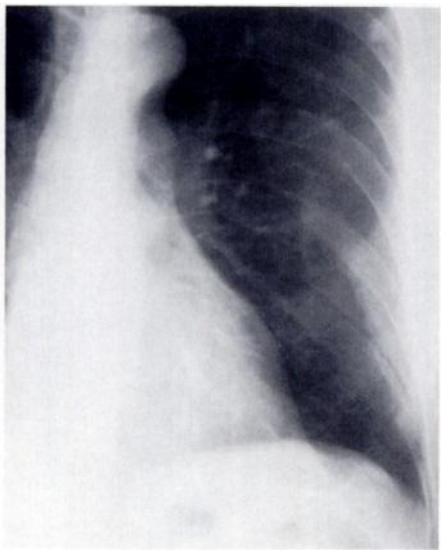
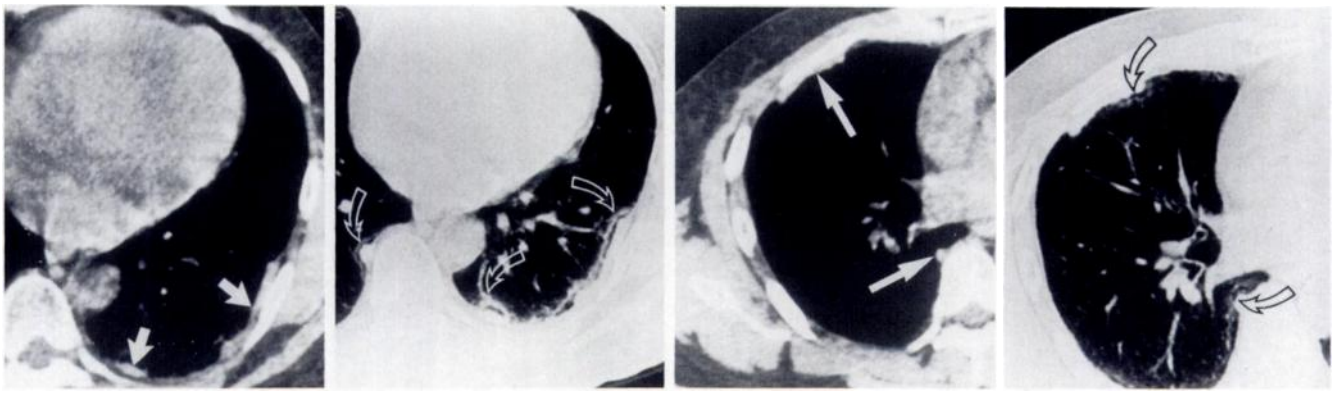


Fig. 5.—Subtle parenchymal asbestosis seen with greater confidence and extent on high-resolution CT than on inside films.

***A*, Obvious plaques on inside films. Left lower lobe interstitial disease was suspected.**

***B* and *C*, Periplaque subpleural curvilinear density (*arrows*) diagnostic of interstitial lung disease. Plaques (*arrows*) are seen better on mediastinal windows (*B*), whereas parenchymal disease is seen better on lung windows (*C*).**

***D* and *E*, Additional (more subtle) interstitial changes adjacent to plaques (*arrows*).**



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