Pulmonary Tuberculosis: CT Findings—Early Active Disease and Sequential Change with Antituberculotic Therapy

To evaluate findings of active pulmonary tuberculosis on computed tomographic (CT) scans and their sequential changes before and after antituberculous chemotherapy, 29 patients with newly diagnosed pulmonary tuberculosis and 12 patients with recent reactivation were studied prospectively. The diagnosis of active pulmonary tuberculosis was based on positive acid-fast bacilli in sputum \((n = 29)\) and changes on serial radiographs obtained during treatment \((n = 12)\). Twenty-six patients were followed up with CT during treatment for 1-20 months. Lungs from the cadavers of nine other patients, who died of pulmonary tuberculosis, were studied to provide a pathologic basis for diagnosis. At examination with CT, centrilobular lesions (nodules or branching linear structures 2-4 mm in diameter) were most commonly seen \((n = 39 (95\%))\); in the 26 patients with follow-up, most of these lesions disappeared within 5 months after the start of treatment. In 11 of 12 patients with recent reactivation, CT clearly differentiated old fibrotic lesions from new active lesions. Lesions in and around the small airways appear to be the most characteristic CT feature of early active tuberculosis and may be a reliable criterion for disease activity.

**Materials and Methods**

Forty-one patients with active pulmonary tuberculosis were prospectively studied with high-resolution CT at our institutions from June 1989 to January 1992. These patients were divided into two groups. The first group consisted of 29 patients who had no history of tuberculosis but in whom radiographic and laboratory evidence of active pulmonary tuberculosis had been recently found (6 days to 3 weeks before this study). The second group consisted of 12 patients who had received antituberculous chemotherapy 9 months to 26 years (mean, 6 years) before this study. The duration of treatment in these patients had ranged from 6 to 24 months (mean duration, 12 months). All 12 patients had findings consistent with pulmonary tuberculosis on chest radiographs; recent follow-up radiographs showed subtle \((n = 8)\) or definite \((n = 4)\) new lesions. CT was performed in these patients for evaluation of possible reactivation.

Twenty-four patients were men and 17 were women. Their ages ranged from 18 to 75 years (mean age, 36 years). The diagnosis of active pulmonary tuberculosis was based on \((a)\) detection of acid-fast bacilli in sputum \((n = 29)\) that were identified by means of smear \((n = 27)\) or culture \((n = 2)\) in 22 of 29 patients with newly diagnosed pulmonary tuberculosis and seven of 12 patients who received treatment, \((b)\) findings on chest radiographs (including serial changes during antituberculotic chemotherapy) \((n = 12)\), along with positive results of the purified protein derivative test \((n = 7)\).

All 41 patients underwent examination with CT within 3 weeks of diagnosis \((n = 29)\) and within 2 weeks of suspected reactivation \((n = 12)\). Twenty-six patients were followed up with CT \((17)\) patients with newly diagnosed tuberculosis and nine patients with suspected reactivation) 1 month \((n = 8)\), 3 months \((n = 9)\), 5 months \((n = 10)\), 9 months \((n = 11)\), 12 months \((n = 6)\), 15 months \((n = 3)\), and 20 months \((n = 3)\) after the start of antituberculotic chemotherapy. Such therapy included administration of various combinations of isoniazid, rifampin, ethambutol, hydrochloride, streptomycin, or paraaminosalicylic acid.

CT scans were obtained with a commercially available scanner (model 9800; GE Medical Systems, Milwaukee, Wis) (1.5-mm collimation, 140 kVp, 170 mA, 2-second scan time, 512 x 512 matrix, and a bone algorithm) without injection of contrast media. They were obtained with a 35-cm field of view and were retrospectively targeted with an 18-21-cm field of view, depending on the size of the thorax. For the initial CT scan, five to 10 levels were selected with the chest radiograph; for follow-up examinations, levels were matched to those on the initial CT scans. Descriptive terms used to interpret CT scans were:

- **Lung:** Hypoattenuated areas, usually seen in interlobular septa or along bronchovascular bundles.
- **Nodule:** A small, round, well-defined lesion with uniform attenuation.
- **Lesion:** A larger, irregularly shaped area with variable attenuation.
- **Calcification:** A dense, round, well-defined area with uniform attenuation.
- **Bronchial wall thickening:** An increased thickness of the bronchial wall, usually seen along the bronchovascular bundle.
- **Necrotic lesion:** An area with low attenuation, often seen in patients with a history of tuberculosis.

Index terms: Computed tomography (CT), high-resolution • Lung, CT, 60.211 • Lung, diseases, 60.2311, 60.2313, 60.232 • Lung, nodules, 60.2311, 60.2313, 60.232 • Tuberculosis, pulmonary, 60.2311, 60.2313, 60.232

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findings were defined as follows: (a) centrilobular nodule or linear structure: well-defined lesions 2-4 mm thick, separated by more than 2 mm from the pleural surface or interlobular septa (Fig 1); (b) “tree in bud”: a branching linear structure with more than one contiguous branching site (Fig 2); and (c) poorly defined nodule: a poorly defined nodule 5-8 mm in diameter (Fig 3).

Chest radiographs obtained at the time of initial examination with CT were reviewed with a focus on the presence of cavities and findings of bronchogenic spread of tuberculosis.

To provide a pathologic basis for the CT findings, we studied the lungs from nine cadavers of patients who died of pulmonary tuberculosis, six with bronchogenic spread and three with miliary tuberculosis. Three of the nine patients had undergone antituberculous chemotherapy until just before death. The lungs obtained at autopsy were inflated with formalin (three cases) or specific fixative (six cases) in the manner described by Heitzman (8). Radiologic and pathologic findings were correlated with findings on radiographs of the specimen by use of the technique described by Itoh et al (9).

RESULTS

Radiologic and Pathologic Correlation in Nine Isolated Lungs Obtained at Autopsy

We found regions of consolidation that varied in size. Smaller areas of consolidation consisted mainly of caseation necrosis, with either a centrilobular or subacinar distribution (Fig 4). Centrilobular lesions simultaneously involved the bronchioles and marginal alveoli. Caseous materials that filled both the bronchiolar lumen and alveolar ducts caused a small, branching subacinar consolidation, which appeared finer than the centrilobular type of consolidation. Larger areas of consolidation had a lobular or lobar distribution. Typically, lobular consolidation consisted of centrally located granulomas that contained caseation necrosis and marginal nonspecific inflammation; the area of inflammation appeared relatively loose compared with the central granulomas with caseation necrosis (Figs 5, 6).

Cavity formation usually began centrally within the secondary lobule (Fig 5a). Several small cavities may fuse within the lobule, producing a larger cavity within a wall separated from the interlobular septa (Fig 5b). Another mechanism for cavity formation appeared to involve bronchial wall necrosis. In this instance, macroscopic cavities resembled ectatic bronchi. However, histologic examination proved that the bronchial walls had been destroyed by tuberculosis.

Bronchial walls were thickened, and sometimes the bronchi were filled with compact, hard caseous material. Calcified scar was always associated with local emphysema. Bronchi adjacent to areas of scar formation were ectatic and distorted.

Findings of active pulmonary tuberculosis were present in all nine patients. In six patients, findings of old healed tuberculosis, such as calcification, fibrosis, or bronchiectasis, were also present.

Miliary tuberculosis consisted of small nodules in the alveolar region scattered randomly throughout the lungs, without any relationship to the bronchioles.

Figure 1. CT scan of left upper lung in a 34-year-old man with high fever and chills due to bronchogenic spread of tuberculosis. A sputum smear was positive for acid-fast bacilli. Note the approximately 2-mm-thick centrilobular branching linear structure (long straight arrow), which corresponds to caseous necrosis within the bronchiole shown in Figure 4. Multiple discrete centrilobular nodules 2-3 mm in diameter (arrowheads) are seen. Some secondary lobules are entirely consolidated, with (curved arrow) or without (short straight arrow) a patent bronchiule. The bar at top indicates 10 mm.

Figure 2. CT scans from a woman aged 20 years with extensive bronchogenic spread of tuberculosis. (a) CT scan obtained at the level of the right hilum shows extensive bronchiolar and, possibly, alveolar ductal lesions with a typical tree-in-bud appearance (arrow). The bar indicates 10 mm. (b) Follow-up CT scan obtained after 9 months of antituberculous chemotherapy shows that the tree-in-bud lesions have disappeared, leaving tiny discrete dots and lines within emphysematous lung. The previously uninvolved portion of the lung (arrowheads) maintains normal architecture and attenuation characteristics. A large branching lesion (arrow) suggests endobronchial caseous material or mucus plug with proximal bronchial stenosis.
CT Findings in Patients with Tuberculosis

CT findings at initial presentation in 41 patients are summarized in Table 1. Of the 29 patients with newly diagnosed active pulmonary tuberculosis, 28 had findings of bronchogenic spread of the disease (Figs 1, 7) and one had miliary tuberculosis (Fig 8). Findings consistent with bronchogenic spread were a centrilobular nodule or branching linear structure (n = 28 [97%]) (Fig 1), bronchial wall thickening (n = 23 [79%]) (Fig 7) with bronchiectasis (n = 14 [48%]) or without bronchiectasis (n = 9 [31%]), a tree-in-bud appearance (Fig 2) (n = 21 [72%]), or poorly defined nodules 5–8 mm in diameter (n = 20 [69%]). Other findings included cavitary nodules or consolidation (n = 19 [66%]), lobular

Figure 3. CT scans from a man aged 34 years with exudative tuberculosis and sputum positive for acid-fast bacilli. (a) CT scan obtained in the left upper lung at the time of diagnosis shows air-space consolidation posteriorly. Note the poorly defined nodule approximately 7 mm in diameter in the central portion of a secondary pulmonary lobe (curved arrows). Note also the markedly thickened interlobular septa (straight arrows) and multiple centrilobular nodules and branching linear lesions. The bar indicates 10 mm. (b) CT scan obtained at the same level as a after 2 weeks of antituberculous chemotherapy. The poorly defined nodule in a has become three discrete centrilobular (bronchiolar) nodules approximately 3 mm in diameter (curved arrows). This change suggests that the poorly defined nodule in a initially began as a bronchiolar nodule that grew to acinar size. Note remarkable thinning of the interlobular septa (straight solid arrows) in comparison with a; this thinning suggests an interval decrease in the amount of fluid transported through septal lymphatic channels. Disappearance of lobular consolidation tends to begin at the periphery (open arrows).

Figure 4. Images of isolated lung from the cadaver of a woman aged 50 years with bronchogenic spread of tuberculosis. (a) Radiograph of a lung section shows centrilobular branching linear lesions (arrowheads) that fill the bronchiolar lumen, with terminal clubbing due to perbronchiolar extension. Note also multiple contiguous branching linear lesions with a tree-in-bud appearance produced by caseous material within the respiratory bronchioles and alveolar ducts (arrows). The bar at top left indicates 5 mm. (b) Close-up photograph of the centrilobular lesions, marked with arrowheads in a, shows yellowish caseous necrotic material that fills bronchioles 1 mm in diameter and peripheral alveolar ducts (arrows). The bar in the left upper corner indicates 1 mm.
consolidation (n = 15 [52%]) (Fig 1), thickening of interlobular septa (n = 10 [34%]) (Fig 3), emphysema (n = 7 [24%]), bronchovascular distortion (n = 5 [17%]), bronchial impaction (n = 5 [17%]), and fibrotic bands (n = 5 [17%]) (Table 1). Whenever a cavity developed within a consolidated lobule, it occurred centrally within the lobule.

Of the 12 patients with a history of treated tuberculosis who underwent evaluation with CT for possible reactivation, 11 had findings of bronchogenic spread such as centrilobular nodules or branching linear lesions (n = 11 [92%]), a tree-in-bud appearance (n = 8 [67%]), or bronchial wall thickening (n = 7 [58%]). The presence of these findings was distinct from that of concomitant fibrocalcified lesions at other sites, shown in eight patients (67%), and provided evidence of reactivation (Fig 9). Compared with CT findings in the group of patients with newly diagnosed pulmonary tuberculosis, CT findings of bronchovascular distortion (n = 7 [58%]), bronchiectasis (n = 7 [58%]), emphysema (n = 6 [50%]), and fibrotic bands (n = 6 [50%]) were more commonly seen (Table 1). The difference in prevalence between the group with newly diagnosed disease and the group with reactivated disease was significant in lobular consolidation (P = .0095), bronchovascular distortion (P = .043), and fibrotic bands (P = .052).

Follow-up CT scans, obtained at various times during treatment, showed gradual disappearance of lobular consolidation, septal thickening, poorly defined nodules, the tree-in-bud appearance, and centrilobular nodules or branching linear lesions, in that order (Figs 3, 9) (Table 2). Resolution of lobular consolidation began usually at the periphery, with eventual transformation into a poorly defined nodule, followed by appearance of a centrilobular nodule or branching linear lesions (Fig 3). In all 17 patients who had newly diagnosed tuberculosis and underwent follow-up CT, centrilobular nodules or branching linear structures were seen on initial CT scans; on follow-up CT scans, centrilobular lesions gradually decreased in prevalence and were no longer seen after 12 months (Table 2). Compared with centrilobular lesions, cavities disappeared with more residual fibrotic change (Fig 10).

Distortion of bronchovascular structures, emphysema, fibrotic bands, and bronchiectasis tended to increase in prevalence on follow-up CT scans. Prevalences on initial CT scans and follow-up CT scans obtained at 20 months were 24% and 100%, 35% and 100%, and 18% and 100% for bronchovascular distortion, emphysema, and fibrotic bands, respectively. Distortion of bronchovascular structures was commonly associated with emphysematous change and fibrotic bands. Emphysema developed only at previous lesion sites and had a lobular or subsegmental distribution, a finding that suggests that bronchiolar or bronchial stenosis may be the underlying mechanism for emphysema (Fig 11). Whenever bronchovascular distortion was severe, the lobular or subsegmental distribution could not be identified and had an appearance similar to that of paracartilaginous emphysema.

Mediastinal lymph node enlargement (defined as a lymph node greater than 10 mm in diameter) was seen in nine of 29 patients with newly diagnosed disease and in two of 12 patients with reactivation.

Nine patients who were studied for possible reactivation and underwent follow-up CT showed no substantial interval change of fibrocalcific treated lesions, while presumed newly spread or reactivated lesions showed chronological change similar to that in patients with newly diagnosed disease on follow-up CT scans.

**Chest Radiographic Findings in Patients with Tuberculosis**

Chest radiographs at initial presentation showed findings of bronchogenic spread tuberculosis, defined as the presence of multiple, poorly defined nodules approximately 5 mm in diameter, in 10 patients (24%). Cavities were seen in nine patients (22%), eight of whom had newly diagnosed disease and one of whom had undergone treatment.

**DISCUSSION**

Tuberculosis is a chronic granulomatous infection characterized by caseation necrosis and great propensity for fibrosis and calcification (10). Tu-
genic dissemination in our study was that of a centrilobular nodule or branching linear structure 2–4 mm in diameter. In spite of their small size, such lesions had sharp margins and relatively high attenuation. These centrilobular lesions consist of solid caseation material within or around the terminal or respiratory bronchioles, as was demonstrated in pathologic specimens and on radiographs of the specimens (Fig 4).

Multiple branching linear structures of similar caliber that originated from a single stalk (the tree-in-bud appearance) were commonly seen in patients with extensive bronchogenic spread (Figs 2, 4). Terminal tufts of the “tree-in-bud” structure may represent lesions within the bronchioles and alveolar ducts, while the stalk may represent a lesion that affects the last-order bronchus within the secondary lobule (Fig 4).

Hematogenous dissemination of tuberculosis, shown on both CT scans (Fig 8) and radiographs of the specimen, differs from bronchogenic spread in that it is evenly distributed throughout the lung, with nodules of uniform size and without associated bronchial wall thickening.

Aschoff (13) described the consolidation of a single acinus by tuberculous inflammatory exudate or granulation tissue as an “acinonodose” lesion. The presence of multiple fluffy nodules approximately 5 mm in diameter, described as acinar nodules, is the classical radiographic pattern for bronchogenic dissemination of tuberculosis (14). However, some researchers have proved that many of these nodules are actually peribronchial and bear no direct anatomic relationship to the acinus (9,15). Itoh et al (9) observed that in most cases of acino-nodose tuberculosis, small peribronchial nodules grow and coalesce into a larger nodule, with a diameter almost equal to that of the pulmonary acinus, by direct extension of inflammation through the pores of Kohn.

Our study supports the observations of Itoh et al (9) in three ways: (a) Poorly marginated nodules that were the same size as an acinus on the initial CT scan became smaller, well-marginated, centrilobular nodules after antituberculous treatment (Fig 3); (b) poorly marginated nodules were usually located in the central portion of the secondary pulmonary lobule (Fig 3); and (c) cavitation within areas of lobular consolidation occurred centrally (Fig 5), a finding that suggests that the initial lesion was in the peribronchial region.

Table 1

<table>
<thead>
<tr>
<th>CT Finding</th>
<th>No. of Patients without Previous Treatment (n = 29)</th>
<th>No. of Patients with Previous Treatment (n = 12)</th>
<th>Total No. of Patients (n = 41)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Centrilobular nodule or branching linear lesion</td>
<td>28 (97)</td>
<td>11 (92)</td>
<td>39 (95)</td>
</tr>
<tr>
<td>Bronchial wall thickening</td>
<td>23 (79)</td>
<td>7 (58)</td>
<td>30 (73)</td>
</tr>
<tr>
<td>Tree-in-bud appearance</td>
<td>21 (72)</td>
<td>8 (67)</td>
<td>29 (71)</td>
</tr>
<tr>
<td>Poorly defined (&quot;fuzzy&quot;) nodule</td>
<td>20 (69)</td>
<td>5 (42)</td>
<td>25 (61)</td>
</tr>
<tr>
<td>Cavity</td>
<td>19 (66)</td>
<td>5 (42)</td>
<td>24 (58)</td>
</tr>
<tr>
<td>Bronchectasis</td>
<td>14 (48)</td>
<td>7 (58)</td>
<td>21 (51)</td>
</tr>
<tr>
<td>Lobular consolidation</td>
<td>15 (52)*</td>
<td>2 (17)</td>
<td>17 (41)</td>
</tr>
<tr>
<td>Emphysema</td>
<td>7 (24)</td>
<td>6 (50)</td>
<td>13 (32)</td>
</tr>
<tr>
<td>Septal line thickening</td>
<td>10 (34)</td>
<td>2 (17)</td>
<td>12 (29)</td>
</tr>
<tr>
<td>Bronchovascular distortion</td>
<td>5 (17)*</td>
<td>7 (58)*</td>
<td>12 (29)</td>
</tr>
<tr>
<td>Fibrotic bands</td>
<td>5 (17)*</td>
<td>6 (50)*</td>
<td>11 (27)</td>
</tr>
<tr>
<td>Bronchial impaction</td>
<td>5 (17)</td>
<td>0 (0)</td>
<td>5 (12)</td>
</tr>
<tr>
<td>Milary nodules</td>
<td>1 (3)</td>
<td>0 (0)</td>
<td>1 (2)</td>
</tr>
</tbody>
</table>

Note.—Numbers in parentheses are percentages.

* The difference was significant at analysis with the χ2 distribution (P < .05).

Figures 7, 8. (7) CT scan targeted to right upper lung in a woman aged 23 years with a sputum culture positive for acid-fast bacilli. Note thickening of the bronchial wall (arrowheads) and the distal poorly defined nodule (short arrow). Multiple smaller bronchiolar nodules with branching linear lesions of various lengths (long arrow) are seen. (8) CT scan from a woman aged 28 years with miliary tuberculosis. Multiple discrete miliary nodules are seen in an even distribution throughout the lung. These nodules are uniform in size, and their distribution bears no relation to the airways.

Tuberculous lesions may develop in the lung in a variety of ways: local progression, lymphogenous dissemination, bronchogenic dissemination, or hematogenous dissemination. Bronchogenic dissemination is the most common means of spread in the postprimary or reinfection type of tuberculosis. Prerequisites for bronchogenic spread are necrosis of a bronchial wall and softening or liquefaction necrosis of the caseous material, which in most lesions is otherwise so viscous that flow into the bronchial lumen would seem unlikely (10). Endobronchial spread of tuberculosis has been described as occurring in 20% of patients with postprimary tuberculosis seen on chest radiographs (11,12). However, in this study, all patients but one (n = 40 [98%]), who had miliary tuberculosis, had findings of endobronchial spread on CT scans, in contrast to 10 patients (24%) with such findings on chest radiographs.

The earliest CT finding of bronchi-
Cavities are formed when the caseous necrotic material liquefies and is extruded through the connecting airway. The presence of a cavity is an important sign that indicates active disease. High-resolution CT demonstrates small cavities within areas of consolidation that cannot be seen on chest radiographs. In our study, the prevalence of cavities demonstrated on initial CT scans was 58% (24 of 41 patients), whereas the prevalence of cavities on radiographs was only 22% (nine of 41 patients). Follow-up CT scans obtained after antituberculous treatment showed that healing of cavitory lesions caused more cicatrical change than in other areas without cavitation (Fig 10). Eventual thinning and ballooning of the cavity wall sometimes made it difficult to differentiate a cavity from an emphysematous bulla during treatment.

Complete disappearance of a tuberculous lesion is rare but can occur if a lesion heals before necrosis has developed (10). In our study, CT showed that lesions that tended to disappear completely after treatment were mostly along the periphery of the consolidated lobules (Fig 3) or had a ground-glass appearance adjacent to an area of consolidation. On the other hand, centrilobular lesions invariably caused various degrees of fibrosis in association with bronchovascular distortion or emphysema (Fig 11).

The development of emphysema during healing in patients with bronchogenic dissemination was clearly shown on CT scans in our study (Figs 9, 11). Areas with compact bronchiolar or, presumably, alveolar ductal lesions on CT scans obtained before treatment showed clear evidence of emphysema after treatment, demarcated by adjacent normal lung (Fig 2). Some

![CT scans showing cavities and emphysema](image_url)

**Figure 9.** CT scans from a man aged 51 years who had received antituberculous chemotherapy 20 years previously and in whom reactivation of disease was suspected on the basis of recent chest radiographs. (a) CT scan obtained at the level of the lung apex shows conglomerate nodules with calcification and aggregated ectatic bronchi (arrows); both findings indicate treated lesions. (b) CT scan obtained on the same day as (a) and 6 cm below (a) shows multiple peripheral small nodules and branching linear structures (arrows). Bronchovascular structures are not distorted, a finding that indicates that these lesions most likely represent new active disease.

**Table 2**

<table>
<thead>
<tr>
<th>CT Finding</th>
<th>Prevalence at Initial Manifestation</th>
<th>Prevalence of Each Finding at Follow-up CT</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Initial (n = 17)</td>
<td>1 month (n = 3) 3 months (n = 5) 5 months (n = 8) 9 months (n = 6) 12 months (n = 4) 15 months (n = 1) 20 months (n = 2)</td>
</tr>
<tr>
<td>Centrilobular nodule/branching</td>
<td></td>
<td></td>
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<tr>
<td>Linear structure</td>
<td>100</td>
<td>67</td>
</tr>
<tr>
<td>Tree-in-bud appearance</td>
<td>71</td>
<td>33</td>
</tr>
<tr>
<td>Poorly defined (fuzzy) nodule</td>
<td>71</td>
<td>33</td>
</tr>
<tr>
<td>Lobular consolidation</td>
<td>47</td>
<td>20</td>
</tr>
<tr>
<td>Septal line thickening</td>
<td>35</td>
<td>0</td>
</tr>
<tr>
<td>Cavity</td>
<td>76</td>
<td>67</td>
</tr>
<tr>
<td>Bronchial wall thickening</td>
<td>76</td>
<td>100</td>
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<tr>
<td>Bronchiectasis</td>
<td>12</td>
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</tr>
<tr>
<td>Emphysema</td>
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<td>Fibrotic bands</td>
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<td>33</td>
</tr>
<tr>
<td>Bronchial impaction</td>
<td>12</td>
<td>33</td>
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</table>

Note.—All numbers are percentages.
of the bronchiolar nodules caused tiny central dots with surrounding round to oval areas of emphysema 7–10 mm in diameter after treatment (Fig 11). These findings suggest that fibrotic stenosis of the bronchioles or last-order intralobular bronchi, which appears as a central dot, occurs after treatment and causes a distal postobstructive emphysema. This obstructive mechanism in the development of emphysema was seen only in patients with minimal bronchovascular distortion. In patients with severe bronchovascular distortion, CT findings were similar to those of paracatrical emphysema.

Pathogenetic factors in emphysema include traction, obstruction of air passages, and loss of elasticity of the alveolar wall. Traction and obstruction of air passages seem to constitute the dominant factor. These two abnormalities are interrelated, and one may contribute to the development of the other (16). Paracatrical emphysema is a well-known type of com-

Figure 10. CT scans from a woman aged 23 years with hemoptysis and sputum positive for acid-fast bacilli. (a) Initial CT scan obtained at the time of diagnosis shows thick-walled cavity (arrow) and multiple surrounding nodular and linear branching lesions in right upper lobe. (b) Follow-up CT scan obtained 12 months after a was obtained shows extensive fibrotic change at the previous cavity site (black arrow). Small nodular and linear lesions have almost completely disappeared. Note the presence of bronchiectasis (white arrow).

Figure 11. CT scans from a woman aged 20 years with extensive bronchogenic spread of tuberculosis. (a) Initial CT scan shows diffuse small nodular and branching linear bronchiolar lesions as well as fuzzy conglomerated nodules. (b) Follow-up CT scan obtained 9 months after a was obtained, and after antituberculous chemotherapy, shows multiple focal areas of emphysema 7–10 mm in diameter with tiny central dots or lines (white arrows) at previous bronchiolar lesions in a; this finding suggests that bronchiolar stenosis due to fibrosis causes lobular or acinar emphysema. A previously visualized large conglomerate nodular area shows irregular margined emphysema with distortion of bronchovascular structures (black arrows).
pansatory emphysema caused by traction of distal air spaces adjacent to a fibrotic lesion, as was seen in most of the patients in our study who received treatment (Fig 9). However, even in those patients, traction might occur after initial obstruction.

Diagnosis of disease activity in patients with pulmonary tuberculosis depends on the detection of acid-fast bacilli in sputum and is supported by other factors such as changes on serial chest radiographs or a history of antituberculous chemotherapy. However, because acid-fast bacilli are found in sputum in only 20% (17) to 55% (18) of patients with active pulmonary disease and findings on chest radiographs are commonly classified as indeterminate, the diagnosis of disease activity is frequently difficult. Our study shows that centrilobular branching linear structures, some of which have a tree-in-bud appearance, were seen in all 28 patients with newly diagnosed pulmonary tuberculosis, except in the patient with miliary tuberculosis. These lesions disappeared after 5–9 months of treatment in all 28 patients. Centrilobular lesions without evidence of surrounding bronchovascular distortion or fibrosis were the most common and reliable CT finding in cases of active tuberculosis. Because we found during our prospective study that tree-in-bud lesions were characteristic of newly disseminated disease, we also studied 12 patients in whom reactivation of disease was suspected on the basis of CT findings. Tree-in-bud lesions, which are distinct from old fibrocalcified lesions, were characteristically seen in patients with recent reactivation of disease (Fig 9).

Transient thickening of the interlobular septa on CT scans was seen mainly in patients with exudative lesions in a pattern of lobular consolidation (Fig 3). These findings suggest that there may be local increase in lymphatic flow from the exudative lesions that causes thickening of the interlobular septa. Since this finding disappeared completely without any residual changes after antituberculous chemotherapy, it is unlikely that septal thickening indicates caseous material within the septa. Impaired lymph drainage due to associated central lymphadenopathy could also be responsible for thickening of the interlobular septa (19).

In conclusion, branching centrilobular lesions were the most common and characteristic finding on CT scans obtained in patients with newly disseminated pulmonary tuberculosis. Because these lesions are not seen on chest radiographs, CT may provide valuable information about disease activity, especially in patients in whom recent reactivation of disease is suspected.

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References