Computed Tomography in Pulmonary Tuberculosis

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= Abstract = **CT scans in patients with primary tuberculosis commonly show findings of lympho-hematogeneous spread of the disease, while those of reactivation tuberculosis commonly show findings of bronchogenic spread. Typical CT findings of primary tuberculosis are airspace consolidation of the middle and lower lobes with mediastinal and hilar lymphadenopathy showing central lower attenuation and peripheral rim enhancement. Typical CT findings of reactivation tuberculosis are nodular and linear pulmonary lesions at the apex without lymphadenopathy. High-resolution CT is extremely helpful in understanding the patho-morphologic changes, mode of spread of the disease, sequential morphologic change after antituberculous chemotherapy, and possibly in diagnosing activity of the disease. Centrilobular small nodule or branching linear lesions are the most common findings of fresh active pulmonary tuberculosis, which represent intra- and peri-bronchiolar caseation necrosis. CT is also useful in the evaluation of longstanding destructive pulmonary lesions and tracheobronchial tuberculosis. The importance of the role of CT scan in patients with pulmonary tuberculosis is increasing.**

Key Words: *Tuberculosis, Lung, Computed Tomography*

INTRODUCTION

Tuberculosis has a long historical background as evidence of the disease has been found in ancient Egyptian mummies. Though tuberculosis was once the leading cause of death, the prevalence of the disease has steadily decreased with the advent of effective antituberculous chemotherapy in the 1950s (Boyars 1990). In the United States, tuberculosis was regarded as a disappearing disease until 1985, when a 2. 6% increase in its incidence was noticed in 1986 (Centers for Disease Control 1988), which continued to rise thereafter. The major cause of the increasing incidence of tuberculosis was attributed to the increasing incidence of acquired immunodeficiency syndrome (AIDS) (Goodman 1990). Likelihood of tuberculous infection in patients with AIDS is 500 times greater than in the immunocompetent population (Barnes *et al.* 1991). Though the prevalence of AIDS in oriental countries is still low, it is anticipated that the increase of the AIDS population will inevitably result in an increase in the incidence of tuberculosis.

Chest radiography is the mainstay in the detection and follow up examination of patients with tuberculosis. Computed tomography is not routinely used in the evaluation of patients with pulmonary tuberculosis. However, CT scan is required in many circumstances, such as in patients with extensive hemithorax opacification due to destroyed lung or pleural disease, or in
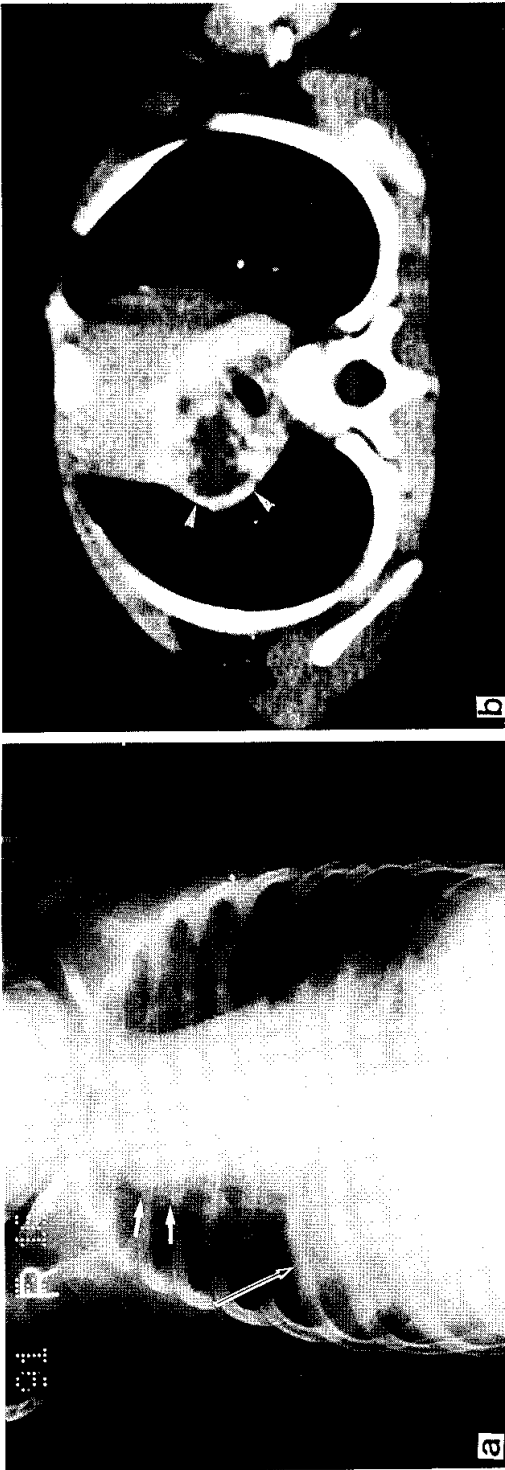
Fig. 1. Chest radiograph (A) and CT scans (B,C) obtained after intravenous injection of contrast medium in a 6-year-old boy with primary tuberculosis.
A. Chest radiograph shows bulging of the upper mediastinum (short arrows) and consolidation of the right lower lung.
B. CT scans obtained at the level of the aortic arch show right paratracheal lymphadenopathy containing central necrotic low attenuation and peripheral rim enhancement (arrowheads). Central low attenuation represents caseation or liquefaction necrosis and peripheral enhancing rim represents granulomatous tissue with inflammatory hypervascularity.
C. CT scan obtained at the level of the lower lung shows dense air-space consolidation of the right lower lobe containing spots of calcification.

patients with complicated airway or mediastinal disease. CT scan is also useful in demonstration of the cavities and in the evaluation of the route of spread(Kulman et al. 1990). High-resolution CT is extremely useful in understanding the pathologic process of the disease, route of spread, and possibly in the evaluation of the disease activity (Im et al. 1993).

Primary Versus Post-Primary Tuberculosis

Primary tuberculosis is the disease in a host unsensitized to tuberculous bacteria who is unable to kill the phagocytized tuberculous bacilli, thus the host is unable to localize the disease, giving rise to local and lympho-hematogenous spread (Powell et al. 1984; Amodio et al. 1986; Stansberry 1990). In populations with a high prevalence of tuberculosis, primary tuberculosis invariably occurs in childhood.

Typical CT findings are dense large air-space consolidation in the middle and lower lobes associated with hilar and mediastinal lymphadenitis (Fig. 1). Association of miliary disease and pleural effusion are also common (Lament et al. 1986; Starke 1988; Morrison 1973). Cavitation of the pulmonary lesion is known to occur less frequently in primary tuberculosis than in post-primary tuberculosis (Choyke et al. 1983; Weizman et al. 1980). In a study of 103 adult patients with primary tuberculosis by Choyke et al (1983), cavitation was observed in 8% of the patients on chest radiographs, while it was seen in 22% of the patients with adult post-primary tuberculosis on chest radiographs and in 58% on high-resolution CT (Im et al. 1993). Mediastinal lymphadenitis appears typically as central low attenuation, which represents caseation necrosis, and peripheral rim enhancement, which represents inflammatory hypervascularity in granulomatous tissue, on scans after injection of intravenous contrast dye, with a predilection of right paratracheal and subcarinal area (Im et al. 1987). Usually, the larger and the more conspicuous the low density areas are, the more

Fig. 2. High-resolution CT targeted to the right upper lung in a 35-year-old woman with reactivation tuberculosis.
Note thickening of the posterior segment of the right upper lobar bronchial wall (arrowheads) and multiple centrilobular nodular and branching linear structures at the bronchial territory (arrows), suggesting bronchogenic spread.

severe the constitutional clinical symptoms are.

Post-primary tuberculosis is the disease in previously sensitized hosts who are competent to kill the phagocytized tuberculous bacilli, thus the hosts are able to localize the disease. Bronchogenic spread is the usual route of spread (Fig. 2). Lympho-hematogeneous spread is unusual in an immunocompetent host (Buckner and Walker 1990). As the disease occurs by reactivation of latent foci, predilection sites are apical portions of the lung, where
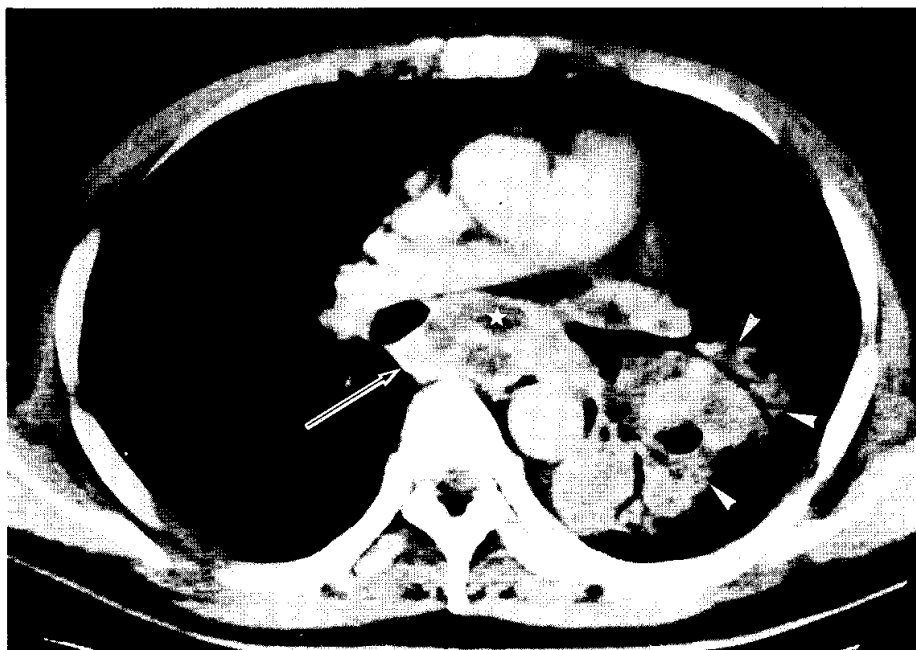
Fig. 3. CT scans in a 35-year-old man with acquired immunodeficiency syndrome (AIDS) and pulmonary tuberculosis.
Note dense air-space consolidation of the left lower lobe (arrowheads) and subcarinal lymphadenopathy (arrow), containing areas of central caseation necrosis (asterisk). The number of helper T-lymphocyte (CD 4+ cell) in this patient was markedly decreased (below 40). Radiological manifestations are similar to that of primary tuberculosis.

oxygen tension is relatively high and lymphatic clearance is relatively ineffective (Goodwin and DesPerez 1983).

Adult-onset pulmonary tuberculosis (primary tuberculosis in the adult) has commonly been described as an unusual radiological presentation because findings were different from those of post-primary tuberculosis seen in most adult patients (Miller and MacGregor 1978; Palmer 1979; Hadlock et al. 1980; Lee et al. 1993; Berger and Granada 1974). However, as the disease represents primary tuberculosis in adults, it is not surprising that radiological findings are different from usual post-primary tuberculosis in adults. Adult-onset pulmonary tuberculosis is especially problematic in populations with lower prevalence of positive purified protein derivative(PPD) test, because the possibility of tuberculosis is usually not considered.

Radiological manifestations of pulmonary tuberculosis in patients with AIDS depend on the degree of cellular immune impairment. In patients early in the course of infection with HIV (human immunodeficiency virus), radiological findings are generally compatible with reactivation tuberculosis, whereas late in the course of infection, with falling helper T-lymphocyte (T-4 lymphocytes or CD4+ cells) counts, the appearance is more consistent with primary infection (Figs. 3) (McLoud and Naidich 1992).

High-Resolution CT in the Evaluation of Pulmonary Tuberculosis

High-resolution CT is extremely helpful in the understanding of morphologic changes of tuberculous lesion, and in the understanding of

Fig. 4. High-resolution CT targeted to the 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 (white arrow). Small bronchiolar branching linear lesions show £tree-in-bud£ appearance (black arrow).

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the spread and healing of the disease (Im et al. 1993).

1) Bronchogenic spread tuberculosis

(1) Fresh untreated lesions

The most frequent route of spread in patients with pulmonary tuberculosis is through the bronchial lumen. The source of bronchogenic spread is usually from the adjacent tuberculous cavity which contains soft or liquefied necrotic materials that are rich in tuberculous bacilli (Fig. 4) (McLoud and Naidich 1992). After lodging of the bacilli, compact caseation necrotic material is formed within and around the small airway (Fig. 5).

According to our experience (Im et al. 1993), the most common high-resolution CT findings of fresh bronchogenic spread tuberculosis is 2-4 mm centrilobular nodules and branching linear structure which represent caseation necrosis within and around the bronchioles (Figs. 5, and 6). Those centrilobular lesions, though small, show clear margin and considerably high attenuation which is unusual for other causes of bronchopneumonia. Multiple branching lesions show a tree-in-bud appearance (Fig. 6). Other findings are 5-8 mm fuzzy nodules, commonly at the centrilobular area, lobular consolidation, and thickening of the interlobular septa, in order of frequency. Thickening of the bronchial wall supplying the lesion site suggests bronchogenic route of spread (Figs. 2, and 4). In patients with newly spread lesions bronchovascular arrangement and lobular architecture are not distorted.

So called "acino-nodose" lesion (Aschoff 1924), which was used to describe consolidation of a single acinus by tuberculous inflammatory exudate or granulation tissue, and which was used to describe classic radiographic findings of bronchogenic spread tuberculosis thereafter (Christensen et al. 1981), appeared invariably as a 5-8 mm fuzzy nodule mostly at the center of the secondary pulmonary lobule on the high-resolution CT scans of our study (Im et al. 1993). Many recent researchers believe that most of the "acino-nodose" lesion do not represent true acinar
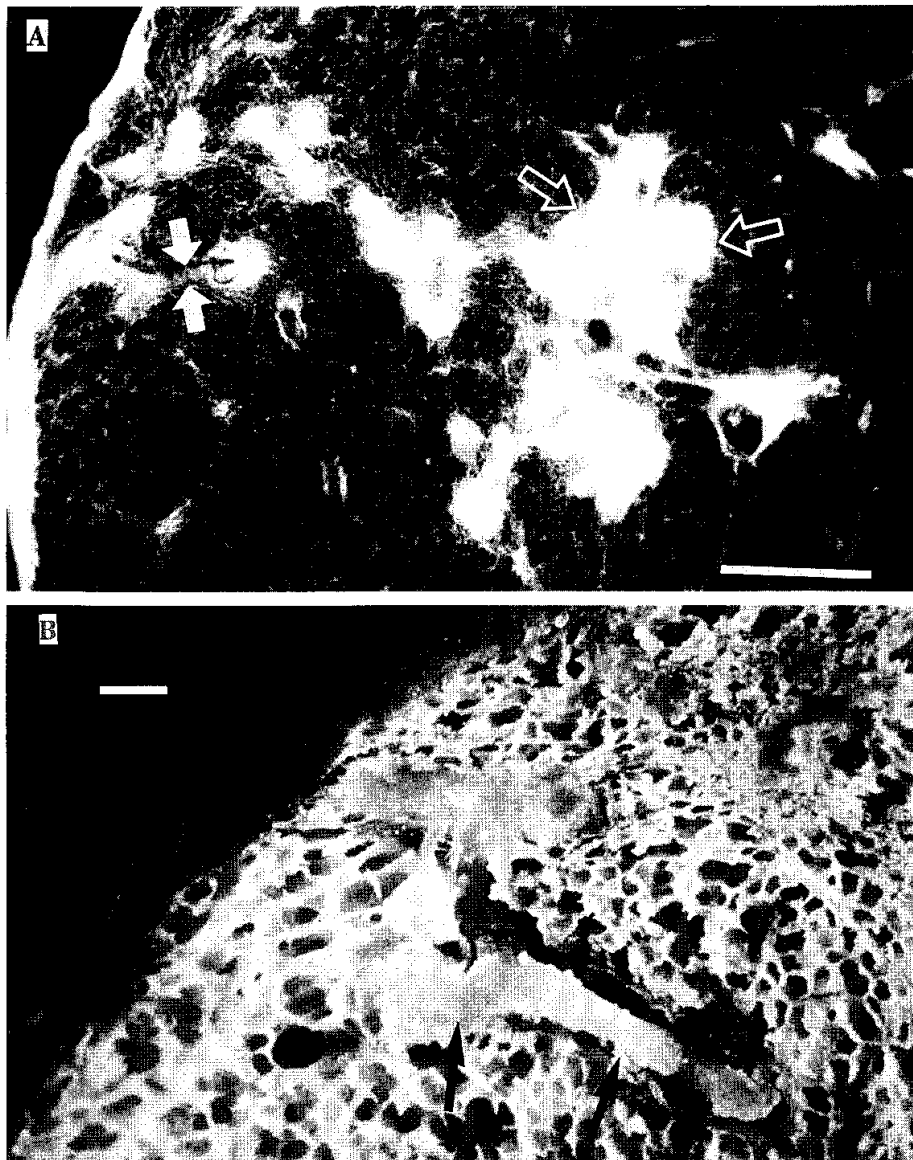
Fig. 5. Images of isolated lung from the cadaver of a woman aged 50 years with bronchogenic spread of tuberculosis. A. Radiograph of a lung section shows centrilobular branching linear lesions (solid arrows) that fill the bronchiolar lumen, with terminal clubbing due to peribronchiolar extension. Note also contiguous branching linear lesions with a tree-in-bud appearance produced by caseous material within the respiratory bronchioles and alveolar ducts (open arrows). The bar at top left indicates 5 mm. B. Close-up photograph of the centrilobular lesions, marked with solid arrows in (A), shows yellowish caseous necrotic material that fills bronchioles 1mm in diameter and peripheral alveolar ducts (arrows). The bar in the left upper corner indicates 1 mm. (Im, J-G, Itoh H, Shim Y-S, et al. Pulmonary tuberculosis: CT findings -- early active disease and sequential change with antituberculous therapy. Radiology 1993;186:653-660. Reprinted with permission)
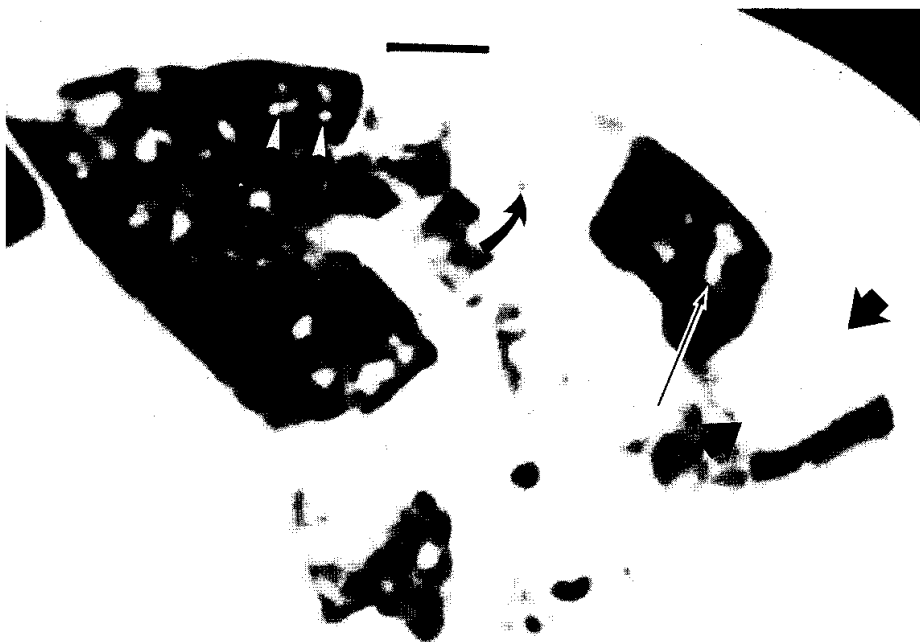
Fig. 6. High-resolution CT scan of the left upper lung in a 34-year-old man with high fever and chills due to bronchogenic spread of tuberculosis. Note the approximately 2-mm-thick centrilobular branching linear structure (long arrow), which corresponds to caseation necrosis within the bronchiole shown in Figure 5. Note also 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 bronchiole. The bar at top indicates 10 mm.
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lesions but grown-up or coalescent small centrilobular or bronchiolar nodules (Itoh *et al.* 1978; Recavarren *et al.* 1967). On follow up high-resolution CT with antituberculous chemotherapy, fuzzy marginated 5-8 mm nodule became multiple 2-4 mm centrilobular nodules, suggesting initially the lesions were at the center of the lobule, not an acinus (Fig. 7). Radiology-pathology correlation demonstrated that the peripheral ill-defined area which disappeared with antituberculous chemotherapy consisted of non-specific exudative inflammation, while centrilobular 2-4 mm nodules which remained on follow up examination, consisted of dense caseation necrosis (Fig. 8)

(Im *et al.* 1993).

Cavitation occurs most commonly at the center of the lobule around the bronchioles, suggesting that the oldest lesions undergo liquefaction necrosis earlier (Figs. 8,9). Contact radiographs of lung specimens of patients who died of tuberculosis demonstrated that cavitation begins as 3-4 mm centrilobular cavities (Fig. 8a), which may undergo fusion forming a larger cavity (Fig. 8b) (Kuhlman 1990).

Sometimes, when caseation materials fill the bronchial lumen, it appears as a larger branching tubular structure commonly showing bulbous ends of the peripheral smaller branches (Fig. 10).
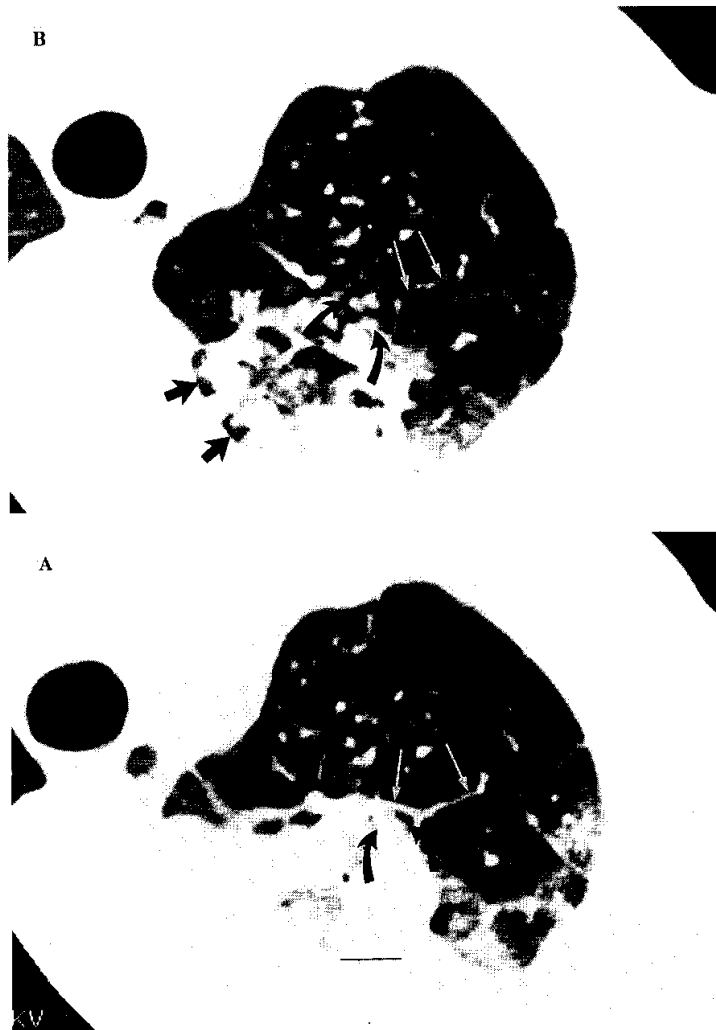
Fig. 7. High-resolution CT scan from a man aged 34 years with exudative tuberculosis and sputum positive for acid-fast bacilli. (A). CT scan obtained in the left upper lung at the time of diagnosis shows air-space consolidation posteriorly. Note poorly defined nodule approximately 7mm in diameter in the central portion of a secondary pulmonary lobule (curved arrows). Note also markedly thickened interlobular septa (straight arrows). 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 (white 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 (black straight arrows). (Im, J-G, Itoh H, Shim Y-S, et al. Pulmonary tuberculosis: CT findings -- early active disease and sequential change with antituberculous therapy. Radiology 1993;186:653-660. Reprinted with permission)
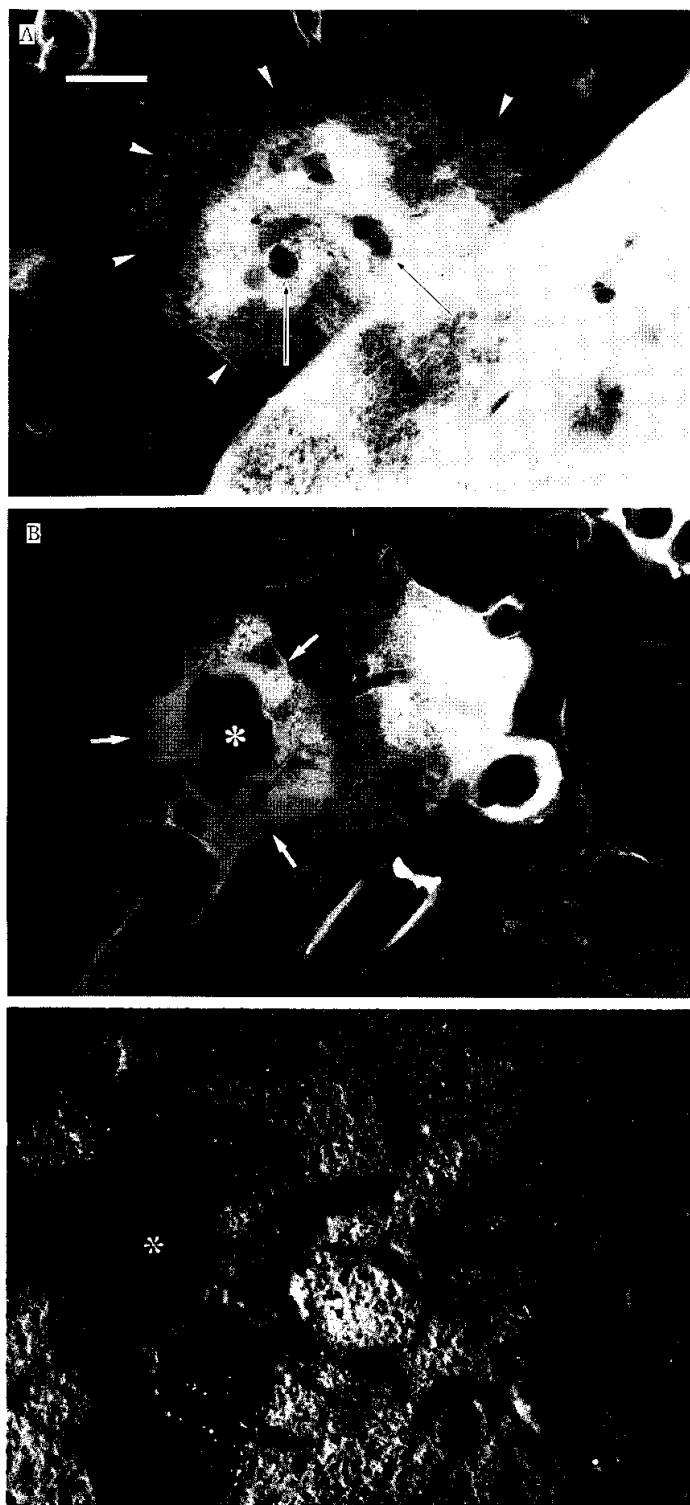
Fig. 8. Contact radiographs (A, B) and surface photograph (C) of lung section from the cadaver of a woman aged 67 years with bronchogenic spread of tuberculosis A. Contact radiograph shows lobular consolidation and centrilobular cavities. Lobular consolidation (arrowheads) consists of loose periphery and compact center. Microscopic examination (not shown here) revealed caseation necrosis at the dense centrilobular region and non-specific inflammation at the loose periphery. Note also centrilobular small cavities 2-3 mm in diameter (arrows). The bar indicates 5 mm. B. Contact radiograph of the lung specimen adjacent to (A) shows consolidation of a secondary lobule (arrows) which contains larger centrilobular cavity (asterisk). C. Photograph of the lung surface identical to (B) shows yellowish consolidation of the lobule containing caseation necrosis (arrows). Note also centrilobular cavity (asterisk). (Im, J-G, Itoh H, Shim Y-S, et al. Pulmonary tuberculosis: CT findings-early active disease and sequential change with antituberculous therapy. Radiology 1993;186:653-660. Reprinted with permission)

Fig. 9. High-resolution CT targeted to the right upper lung in a man aged 41 years with pulmonary tuberculosis shows small centrilobular cavities (black arrows) within the consolidated secondary pulmonary lobules. Note also larger cavities occupying almost entire secondary pulmonary lobules (white arrows). These findings suggest cavitation begins at the center of the lobule and becomes a coalescent larger cavity.

(2) High-resolution CT findings after antituberculous chemotherapy

When effective antituberculous chemotherapy is given, most of the lesions resolve with time, leaving some residual fibrotic or emphysematous change. The degree of residual change depends on the amount of tissue necrosis. Non-specific exudative inflammation resolves completely (Fig. 7). However, areas of caseation necrosis invariably result in some degree of residual fibrosis and emphysema (Fig. 11).

Lobular consolidation and 5-8 mm fuzzy nodule begin to resolve from the peripheral part within a month after initiation of chemotherapy and commonly show small centrilobular nodules in their course of resolution. Thickening of the interlobular septa disappears completely without leaving any residual changes (Fig. 7).

Centrilobular 2-4 mm nodules become smaller with treatment and finally disappear leaving minimal distortion of bronchovascular arrangement and emphysematous change. Cavitary lesions result in more residual cicatricial change.

Development of emphysema is invariably seen in patients with bronchogenic spread tuberculosis after treatment (Fig. 11). The most

Fig. 10. High-resolution CT in a patient with endobronchial tuberculosis involving the right bronchus· intermedius shows narrowing of the proximal segmental bronchi of the right lower and middle lobe. Note large branching lesions in the right lower lobe (arrows) which have clubbed terminal tufts, suggesting impaction of caseous material within the bronchi. Note also hyperaeration around the bronchial impaction.

common mechanism of the development of emphysema is believed to be traction by adjacent scar, so called, paracicatricial emphysema (Liebow 1959). However, bronchiolar or bronchial stricture also results in emphysema as was seen both on high-resolution CT scans of patients (Fig. 12) and on contact radiographs of the isolated lung (Fig. 13).

Mosaic hypoperfusion implies that localized lower attenuation area demarcated typically by interlobular septa on high-resolution CT (Martin et al. 1986; Eber et al. 1993). Possible mechanisms for the development of mosaic hypoperfusion are obstruction of small arteries at the level of the terminal bronchiole (Martin et al. 1986), vascular constriction due to primary vascular disease or secondary to hypoxia, and localized air trapping secondary to airway disease (Eber et al. 1993). We believe most of the mosaic hypoperfusion in patients with history of tuberculosis is caused by air trapping with or without combined hypoxic vasoconstriction, due to bronchiolar stenosis (Fig. 13).

Focal bronchiectasis develops invariably after treatment (Im et al. 1993). Thickening of the bronchial wall and the extent of bronchiectasis are usually minimal as compared with other causes of bronchiectasis, such as post-bacterial or post-viral pneumonia.

(3) High-resolution CT in the determination of disease activity

Diagnosis of disease activity in patients with pulmonary tuberculosis is commonly difficult because acid-fast bacilli are found in sputum in only 20-55% of the patients with active pulmonary disease(Im et al. 1993) and findings on chest radiographs are commonly classified as indeterminate. Out study showed that branching centrilobular lesions without evidence of fibrotic bronchovascular distortion were typical for fresh active disease, while treated or healed lesions showed a mixture of fibrosis, calcification, and bronchiectasis (Fig. 14), suggesting that high-resolution CT is useful in determination of disease activity in selected cases.

2) Miliary tuberculosis

Miliary nodules are usually well-defined and bear no relationship to the airway (Fig. 15) (Im et al. 1993; McGuinness et al. 1992). In the acute stage of miliary dissemination, fine reticulation and ground-glass opacification can be seen (McGuinness et al. 1992). Ground-glass opacification in patients with miliary tuberculosis may suggest transient exudative change or sometimes heralds adult respiratory distress syndrome (Dee et al. 1980). In some patients, findings of both miliary and bronchogenic spread are seen.

Fig. 11. High-resolution CT scan in a patient with extensive bronchogenic spread of tuberculosis obtained at the time of diagnosis (A), and 9 months after (A) with antituberculous chemotherapy (B).

(A). There are multiple compact small nodular and branching linear lesions in the left lower lobe with localized sparing of the normal lung (short black arrows). Note also larger fuzzy nodules (long arrow).

(B). Follow-up CT scan shows irregular emphysema with distortion of bronchovascular arrangement at the previous lesion sites leaving localized sparing of the normal lung density and architecture (arrowheads), which corresponds with normal lung area marked by short black arrow in (A).

Long-standing Destructive Pulmonary Tuberculosis

Typical presentations of long-standing destructive pulmonary tuberculosis on CT scans are atelectatic and fibrotic upper lungs with emphysematous bullae and bronchiectasis commonly containing calcified nodules. The trachea and proximal airway commonly show dilatation probably due secondary to pulmonary fibrosis and distal obstructive airway disease.

When fungus ball (aspergilloma) is complicated, it appears as an area of low attenuation associated with internal sponge-like or marginal curvilinear air shadow (Fig. 16). Fungus ball develops invariably within a cystic bronciectatic space rather than within a tuberculous cavity. Peripheral fungus ball results commonly in thickening of the adjacent pleura.
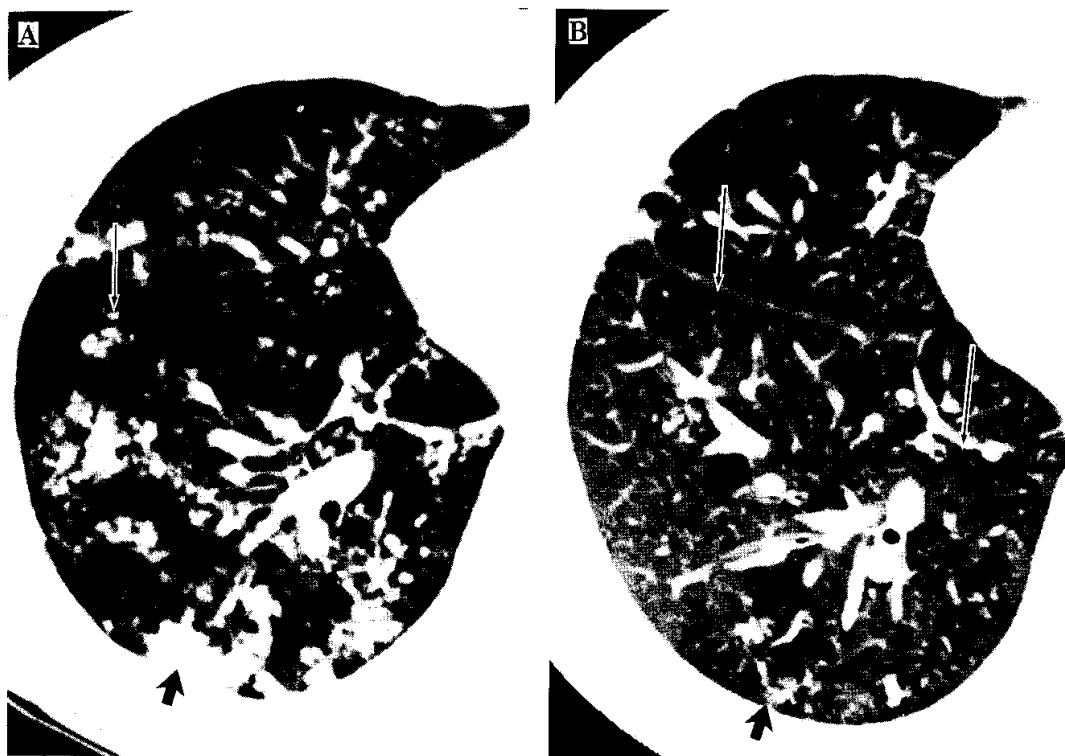
Fig. 12. High-resolution CT from a woman aged 20 years with extensive bronchogenic spread tuberculosis obtained at the time of diagnosis (A), and after 9 months' of antituberculous chemotherapy (B), at the same level.

(A). There are diffuse small nodular and branching linear bronchiolar lesions (long arrow) and fuzzy conglomerate nodules(short arrow) as well.

(B). Note multiple focal areas of emphysema 7-10 mm in diameter with tiny central dots or lines (white arrows with black core) 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 marginated emphysema with distortion of bronchovascular structures (black arrows).

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Apical homogeneous subpleural opacity of thicker than 1-cm, usually seen on chest radiographs of patients with chronic destructive tuberculosis, which may be confused with thickened pleura or loculated fluid collection, has been documented by using high-resolution CT as combined opacity consisting of extrapleural fat and thickened pleura externally, and atelectatic lung centrally (Fig. 16) (Im et al. 1991). Long-standing inward traction of the adherent pleura by cicatricial lungs is a reasonable explanation for the excessive accumulation of the extra pleural fat.
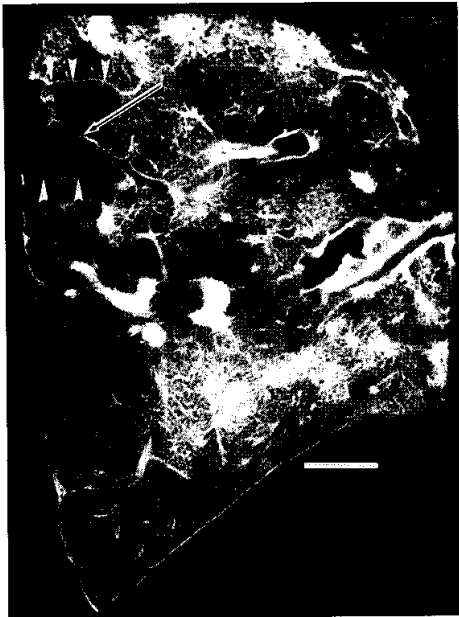
Fig. 13. Contact radiograph of a lung slice isolated from a 85-year-old woman with bronchogenic spread of tuberculosis who had a history of antituberculous chemotherapy showing lobular emphysema due to bronchiolar stenosis. Bar indicates 10 mm.
Note fibrous stenosis of the bronchiole (arrow) resulting in lobular emphysema (arrowheads).

Tracheobronchial Tuberculosis

Tracheobronchial tuberculous involvement has been reported in 10-20% of all patients with pulmonary tuberculosis (Lukomsky et al. 1979). Tuberculous involvement of the peripheral airway results in focal bronchiectasis combined with stenosis. However, stenotic lesions of the peripheral airways are seldom demonstrated on CT scans. In contrast to the peripheral airway, tuberculous involvement of the trachea and proximal bronchi results in stenosis without ectatic change (Fig. 17) (Do et al. 1990; Choe et al. 1990). Dilatation of the trachea and main bronchi, which are seen in some patients with longstanding extensive de-

structive tuberculous pulmonary lesion, is due secondary to fibrotic lung lesion, rather than tracheobronchial lesion itself.

Tracheobronchial tuberculosis is caused by repeated implantation of the organism from cavitary pulmonary lesion which contains abundant M. tuberculosis. The other mechanism is local extension from adjacent mediastinal tuberculous lymphadenitis (Fig. 18) (Ip et al. 1986; Smith et al. 1987). Higher prevalence of both tracheobronchial tuberculosis (Choe et al. 1990; Lee et al. 1991) and mediastinal tuberculous lymphadenitis (Im et al, 1987) in young adult females supports their causal relationship. Of the 23 patients with mediastinal tuberculous lymphadenitis, 13 patients (56 %) were women between 20-39 years (Im et al. 1987), and of the 15 patients with tracheobronchial tuberculosis , 8 patients (53 %) were women between 20-39 years (Do et al. 1990). Diffuse stenosis of the trachea and proximal bronchi may lead to respiratory failure which is a clinically malignant situation (Fig. 17).

CT findings of tracheobronchial tuberculosis depend on the stage of the disease. In patients with active caseation process, irregular thickening of the tracheobronchial wall is seen with some degree of enhancement on post-contrast scan (Fig. 17) (Choe et al. 1990). Enlarged adjacent mediastinal lymph node is commonly seen. However, in patients with healed fibrotic lesion, smooth diffuse narrowing of the tracheobronchial lumen without enhancement or thickening of the wall is typically seen. Differentiation from bronchogenic carcinoma can usually be made by their smooth diffuse narrowing of the lumen without intraluminal mass and by the patients' younger age.

Spiral Volumetric 3-dimensional CT

Spiral volumetric data acquisition with 3-dimensional display of the tracheobronchial tree gives more comprehensive information in the evaluation of major airway stenosis than conventional section by section CT, especially when surgical resection, balloon bronchoplasty,
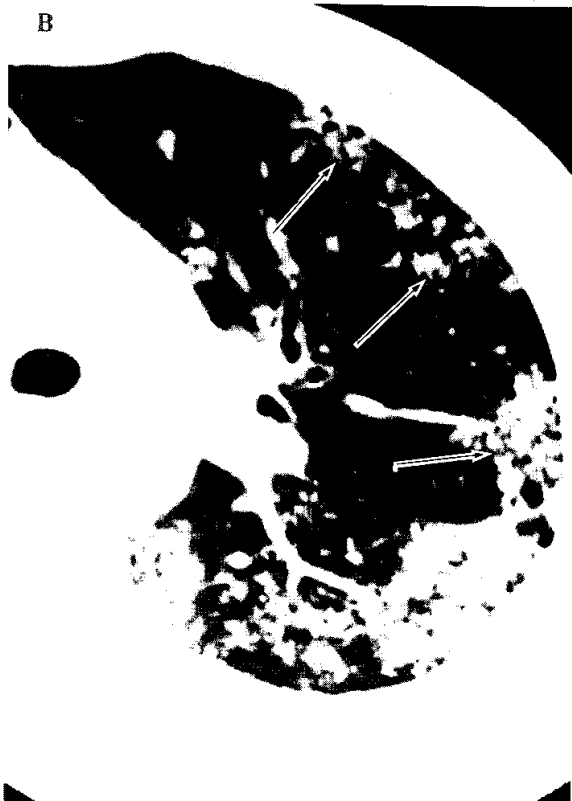
Fig. 14. High-resolution CT scan from a man aged 51 years who had received antituberculous chemotherapy 20 years previously and in whom reactivation of disease was suspected on the basis of recent chest radiographs. (A). CT scan obtained at the level of the lung apex shows conglomerate nodules with calcification (short arrows) and aggregated ectatic bronchi (long arrows); both findings indicate treated lesions. (B). CT scan obtained on the same day as (A) shows multiple small nodules and branching linear structures (arrows). Bronchovascular structures are not distorted, a finding that indicates that these lesions represent new active disease. (Im, J-G, Itoh H, Shim Y-S, et al. Pulmonary tuberculosis: CT findings -- early active disease and sequential change with antituberculous therapy. Radiology 1993;186:653-660. Reprinted with permission)
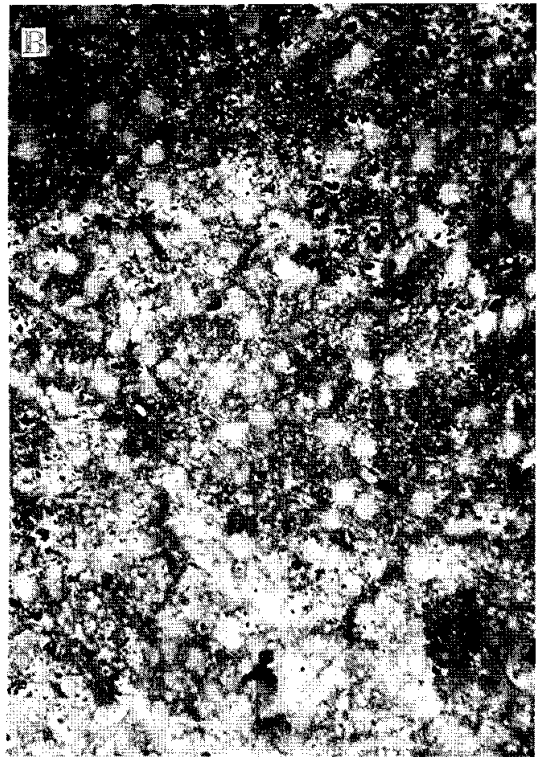
Fig. 15. High-resolution CT (A) and photograph of the sliced lung surface (B) showing miliary tuberculosis.

(A). High-resolution CT in a woman aged 41 years with miliary pulmonary tuberculosis shows multiple discrete miliary nodules distributed throughout the lung. These nodules are uniform in size, and their distribution bears no relation to the airways.

(B). Photograph of the lung shows evenly distributed yellowish-white 2-3 mm nodules.

or insertion of stents are under consideration. Shaded surface display of the tracheobronchial tree is usually most informative (Fig. 19). Coronal or sagittal 2-dimensional reformatted image is also useful in the evaluation of severity and extent of stenosis.

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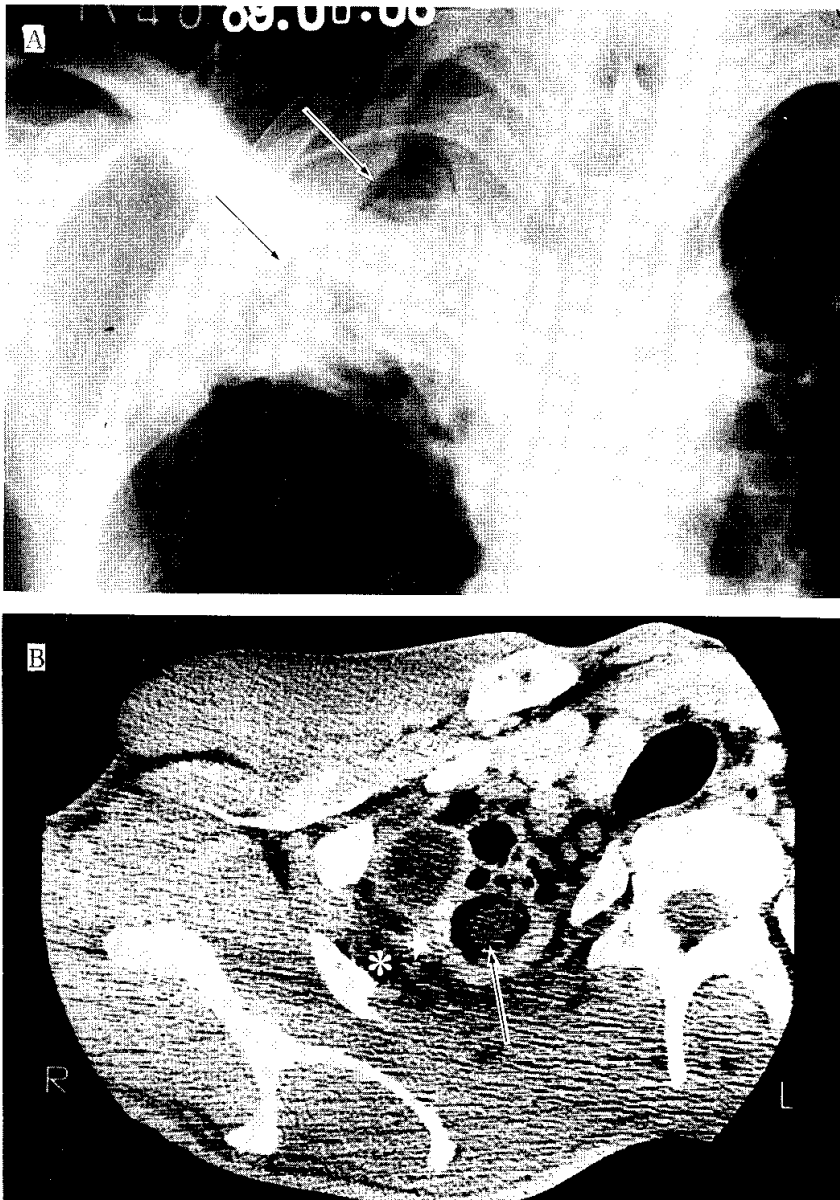
Fig. 16. Images of a 53-year-old woman with a thick apical opacity (apical cap) with an intracavitary aspergilloma. A. Chest radiograph shows a homogeneous 2-cm thick apical opacity that is demarcated by an air meniscus (arrows) marginating the upper aspect of the intracavitary mycetoma. B. High-resolution CT at the level of the black line in (A). The homogeneous opacity seen laterally on the chest radiograph at the black line consists of extrapleural fat laterally (asterisk) and thickened pleura and atelectatic lung medially (star). Note the air-containing intracavitary fungus ball (arrow) and ectatic bronchi medially. (Im J-G, Webb WR, Han MC, Park JH. Apical opacity associated with pulmonary tuberculosis: high-resolution CT findings. Radiology 1991;178:727-731. Reprinted with permission.)
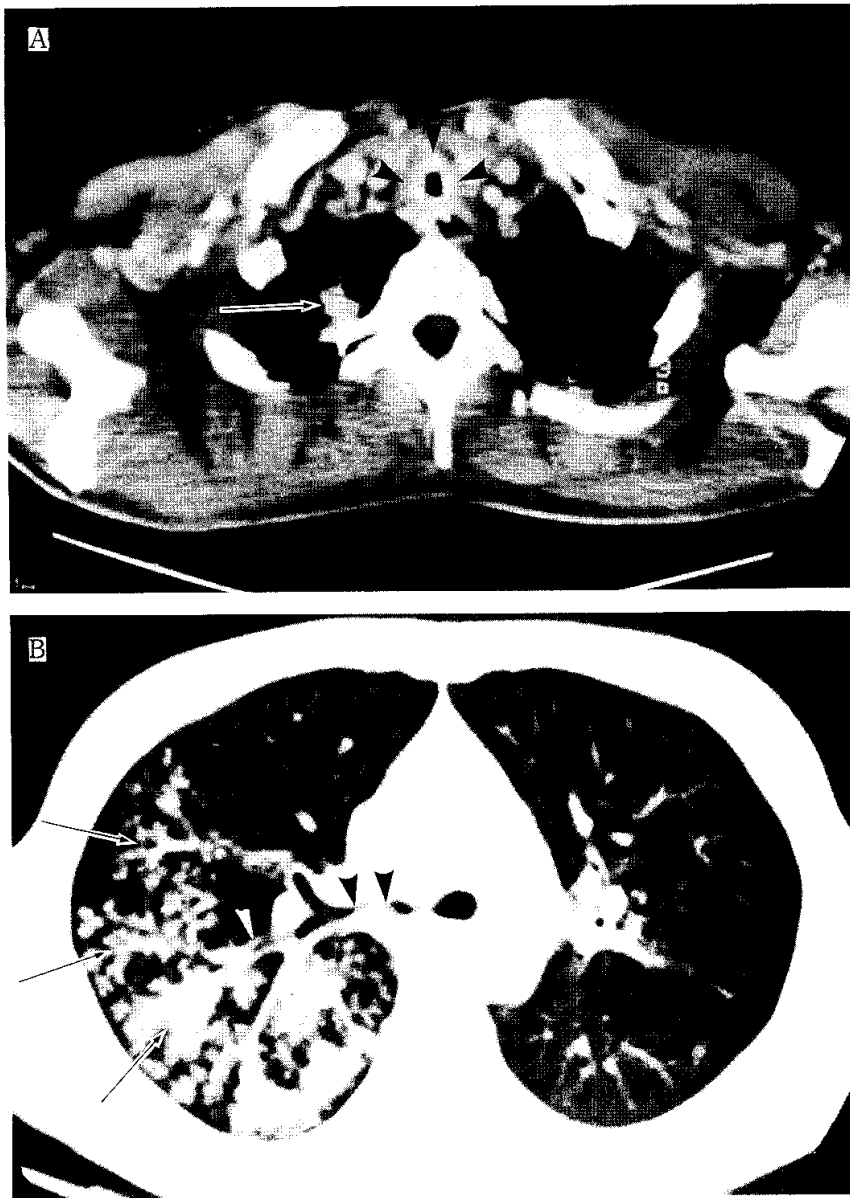
Fig. 17. CT scans obtained after injection of intravenous contrast medium in a 35-year-old man with diffuse tracheobronchial tuberculosis. A. Note marked narrowing of the tracheal lumen and enhancing thickened tracheal wall (arrowheads). On bronchoscopy, there were active caseating granulomas on the surface of the trachea and bronchi. Note also nodular pulmonary tuberculous lesion in the right upper lung (arrow). B. Note diffuse narrowing of the right main and upper lobar bronchi with nodular inner surface (black arrowheads). There is thickening of the wall of the posterior segment of the right upper lobe (white arrowhead) and multiple branching linear infiltrations (arrows) distal to the thickened bronchial wall indicating bronchogenic spread.

Fig. 18. CT scan after intravenous injection of contrast medium in a patient with mediastinal tuberculous lymphadenitis infiltrating to the lumen of the trachea. Note enlarged right paratracheal lymph node (arrowheads) compressing and invading to the tracheal lumen resulting in nodular intraluminal protrusion (arrow). On bronchoscopy, the nodular protrusion consisted of active caseating granuloma.

Fig. 19. Shaded surface display of a 3-dimensional CT image reconstructed from spiral CT data (table feed 3mm, thickness 3 mm, reconstruction 1mm) in a 31-year-old woman with diffuse tracheobronchial tuberculosis. Note diffuse irregular narrowing of the entire trachea (arrowheads) and left main bronchus (arrow). The surface of the lung is also seen with depressions by the ribs.

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