A Case-Study of Perforations of Tuberculous Lymph Nodes into Bronchi and Their Sequelae

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Perforations of caseous tuberculous lymph nodes into bronchi are recognized as a frequent event in primary tuberculosis of childhood,¹ are found increasingly often in the late primary tuberculosis infections of adults,²,³ and have been described as playing a vital part in the pathogenesis of post-primary tuberculous endogenous re-infections.⁴,⁵

This case demonstrates the effects of three such perforative lesions in different bronchi of the same lung.

Case Report—The patient was a 24 year old white man who was first treated in a sanatorium for pulmonary tuberculosis five years prior to admission to Warren State Hospital in July 1954.

The tuberculous lesion at the time of initial treatment was confined to the right lung and consisted of spontaneous collapse of the upper lobe with diffuse infiltrations. His sputum contained acid-fast bacilli. Right phrenic crush was performed and pneumoperitoneum was induced.

Eighteen months later a chest radiograph revealed in the right lung "a fairly heavy amount of infiltration in the upper two-thirds, with an area of cavitation extending from the clavicle down to the upper border of the second rib anteriorly—just outside the hilum." His sputum continued to be positive, and he was placed on streptomycin, para-aminosalicylic acid and isoniazid. Pneumoperitoneum was discontinued because an inguinal hernia developed.

Radiographs taken four years after first admission to the sanatorium were reported to show that the cavernous lesion at the level of the first right interspace had become greatly enlarged and its cubic capacity was estimated to have become five or six times its former figure. The lesions were noted to have spread slightly below the cavernous area, but the basal segments of the lung were found to have undergone little, if any change. No lesion was observed in the left lung. The root area, however, appeared more congested and the pulmonary conus enlarged. As his heart was also beginning to deteriorate, it was thought that "he may be developing a cor pulmonale, and if so, would undoubtedly present clinical symptoms of that condition in the not too distant future."

Six months later, on admission to Warren State Hospital for a mental disorder, the pertinent physical findings included cyanosis, marked clubbing of the fingers, and a greatly increased second heart sound in the pulmonary area. There was no evidence of a mediastinal shift, and the chest was symmetrical and appeared to expand equally on both sides. Medium inspiratory rales were heard throughout the entire right lung field, especially in the mid-axillary region, and breath sounds were diminished anteriorly. His sputum was positive and the chest radiographs essentially unchanged.

Ten months after admission he developed gross pitting edema of both legs and bilateral basal rales. In spite of continued intensive treatment his condition steadily deteriorated and he died three months later of cor pulmonale.

Post-mortem examination showed the following relevant findings in the right lung (Figure 1). The upper lobe was found to form no more than an approximately egg-sized, contracted, fibrotic and airless lump. It was separated from the main bronchus of the upper lobe which was mutilated by scar tissue and completely closed at the point of origin of its main subdivisions.

Adhering to the upper lobe remnants were the middle lobe and the apex of the lower lobe. Both the lobes were markedly displaced upwards, and the middle lobe was enlarged to about twice its normal size.

The apex of the lower lobe contained a walnut-sized tuberculous cavity surrounded by fibrotic pulmonary tissue within which few lentil-sized, hard, whitish foci of caseation were clearly visible. The apical bronchus leading directly to the cavity was markedly

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stentotic. In its anterior wall were two narrow fistulae, but the mutilation was most pronounced at the opening of the bronchus into the cavity, where all that remained of an emptied parabronchial lymph node was a small anthracotic mass.

Another typical perforative scar, almost 2 cm. long, was present in the main lower lobe bronchus, only a short distance below the orifice of the apical bronchus. The bronchial wall was somewhat deformed but no parenchymal lesion was detected in the basal segments.

The left lung, also, showed no significant pathological findings.

**Comment**

Early in the disease, a large complex of tuberculous lymph nodes located in the superior hilum angle perforated into the right upper lobe bronchus, causing destruction of the bronchial wall and distally an aspiration-infiltration of the lung. The resulting collapse of the right upper lobe was observed in the early radiographs although the later progressive contrac-

FIGURE 1: Bronchi of the right lung. Note perforative bronchial scars (B) at the site of the amputation of the upper lobe bronchus (U.L.) by tuberculous lymph nodes; perforative tuberculous lymph node fistulae (C) in the apical bronchus of the lower lobe (A.L.L.) and tuberculous cavity (CAV) surrounded by caseous tuberculous foci (A); perforative bronchial scar (D) in the main bronchus of the lower lobe (L.L.).
tion and fibrosis of the infiltrated pulmonary tissue was established neither clinically nor radiologically.

More than a year later, a relapse of the chronic tuberculous process coincided with further perforations of tuberculous lymph nodes, this time into the apical bronchus of the lower lobe. The aspiration-infiltration of the discharged caseous matter into the apical lung segment led to the development of a cavity. The compensatory enlargement of the lower lobe that accompanied the shrinkage of the upper lobe contributed to the increasing distention of the cavity in the second half of the illness.

The age of the scar in the main bronchus of the lower lobe is, in view of the absence of clinical changes at the base, impossible to assess.

Discussion

Tuberculous hilar lymphadenopathies most frequently cause compression of the bronchus of the right upper lobe. Apart from their great density, the lymph nodes of that region often receive lymph flow directly not only from the superior, middle and inferior parts of the right lung, but also from the middle and inferior regions of the left lung.

If compression of the bronchus is severe enough, the upper lobe will collapse. It is then especially vulnerable, since mechanical factors do not act in favor of rapid re-expansion, and the delay may further progressive fibrosis, collapse induration and irreversible shrinkage—changes which probably occur more often than they are diagnosed.

Schwartz described such lesions in a six year old boy who died of tuberculous meningitis. The bean-sized remains of the right upper lobe were wedged in the angle between the trachea and the grossly enlarged middle lobe. This defect was not detected on the routine radiographs.

The radiological appearance of the initial phase of this condition is probably indistinguishable from the so-called “epituberculous infiltration.” This is particularly well illustrated by a case reported by Terplan where the chest radiographs of a two year old boy showed “an area of consolidation in the right upper lung and dense infiltration,” which, “looked like epituberculous pneumonia.” During the following three years his condition steadily improved, and ultimately the radiological shadow of the right upper lung field almost completely disappeared. He died unexpectedly at the age of five from tuberculous meningitis after a long period of excellent health.

The post-mortem examination demonstrated complete occlusion of the right upper lobe bronchus, the lobe itself comprising no more than a fibrotic, cherry-sized lump. The upper tracheo-bronchial lymph nodes were calcified and fused with the wall of the bronchial stump. The middle and lower lobes occupied the entire right pleural cavity. There was no mediastinal shift to the collapsed side, which, Terplan states, can only be expected if the major bronchus leading to the entire lung is occluded.

The introduction of modern chemotherapeutic agents in the treatment of tuberculosis has emphasized the growing importance of tuberculous
lymph nodes. Unlike many tuberculous parenchymatous lesions in the lungs, tuberculous lymph nodes are hardly accessible to the effects of present-day antituberculous drugs, and remain, often for years, a potential threat to the patient. Intercurrent infections, endocrine changes, such as those of puberty, or a lowering of the general resistance of the patient for any reason, might precipitate a reactivation of the disease through the reservoir of tubercle bacilli in the hilar lymph nodes. They then affect the major bronchi through diseased lymphatics and the extracartilagenous mucous glands as described by Reichle and Frost, through a process of penetration-infiltration, and directly through perforation into the lumen.

The recent trend towards this type of dissemination of the disease is seen in the decreasing incidence in the frequency of the classical caseous, ulcerative type of bronchial tuberculosis and the increase in the bronchial wall of more localized processes suggestive of perforative lesions. Of note, in this connection, is a report of occurrences of tuberculous bronchial fistulae during prolonged and intensive treatment with streptomycin and para-amino-salicylic acid.

The perforative lesions often heal without ill effects and the scars that remain blend themselves with the passage of time to the bronchial wall so completely that they can be detected years later only with difficulty. Less frequently they induce a stenosing bronchitis, with disturbances in the ventilation and drainage of the related pulmonary segments, causing atelectasis, bronchiectasis, and the development of fixed stenoses and bronchial strictures of varying degrees of severity. These bronchial stenoses differ from the more diffuse processes that occur in bronchi draining tuberculous cavities or secretory lesions with retention of pus; in the latter, tuberculous lymph nodes may or may not be found in proximity to the affected bronchus and perforative lesions will not be present.

It is, unfortunately, not always possible to determine the pathogenesis of the bronchial lesion in every case, although valuable aids continue to be furnished through advances in the field of radiological and bronchoscopical techniques.

The importance of detecting fresh bronchial perforative lesions as a cause of a recent parenchymatous flare-up lies in the indication for conservative therapy. Treatment aimed at inducing collapse of the affected lobe in these cases risks initiating or furthering the irreversible pathological changes that have been described.

REFERENCES