Primary Pulmonary Tuberculosis as a Cause of Bronchiectasis in Children*

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A very large percentage of children with the primary complex of tuberculosis are symptomless, and usually these patients show small parenchymal shadows which lead into regional lymph nodes and slowly calcify. However, symptoms may be present, fever being the most common. This is especially true in the cases showing more massive shadows on the roentgen films, the type referred to as epituberculosis. Biggs¹ has recently called attention again to this type of lesion, although in his series only three of 142 patients with primary infection showed massive shadows. In a ward for tuberculous children at the San Francisco Hospital in 1928, about 10 per cent had these shadows, while in 1950 and 1951 the percentage is about 20. There have been on this ward at all times a large number of children of Oriental, Mexican, and Negro parentage, and these groups have been increasing in numbers through the past two decades. They are known to be more highly allergic, as manifested by the size of tuberculin reactions, than children of the white race,² which may in part explain the common incidence of massive shadows.

Jones and Cournand³ in 1933, mentioned enlargement of lymph nodes in primary tuberculosis as being a possible etiological factor in bronchiectasis. Wallgren⁴ called attention in 1935 to the frequency of bronchiectasis following primary tuberculosis of the perifocal or epituberculous type, and reported seven such cases within a single year. In 1937 there was reported from our clinic⁵ two cases in older children of left lower lobe bronchiectasis with positive sputum following primary tuberculous infection. Brock, Cann, and Dickinson⁶ in 1937, suggested that epituberculosis might cause bronchiectasis, and presented a case history. Kent⁷ in 1942, demonstrated asymptomatic bronchiectasis in seven out of 10 children who previously had roentgenographic evidence of atelectasis during their primary tuberculosis. About the same time Jones, Rafferty, and Willis,⁸ demonstrated a relationship of tuberculosis of the bronchi to atelectasis, and in a follow-up study Jones, Peck,

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and Willis\textsuperscript{8} in 1946, showed unequivocal signs of bronchiectasis in 24 such children. This amounted to 70 per cent of 34 who had proved obstructive lesions associated with primary infection. They reported another follow-up of this series in 1950.\textsuperscript{10} These authors concluded that pulmonary tuberculosis in children was a common cause of bronchiectasis. Graham and Hutchison\textsuperscript{11} in 1947, reported 45 cases of pulmonary collapse during the primary infection in childhood, and in four of these bronchiectasis developed while they were under observation. These authors, as well as Kent,\textsuperscript{7} however, believed that collapse does not play an appreciable role in the etiology of infected bronchiectasis. In 1949 Head and Moen\textsuperscript{12} discussed complications of calcified hilus lymph nodes, and stated that basically all of the secondary changes are caused by bronchial ulceration, narrowing and occlusion, and that every type of acute and chronic bronchial and pulmonary disease may be simulated by these complications. Recently Rubin and Rubin\textsuperscript{13} reported 16 cases of shrunken right middle lobe in adults, presenting strong evidence that in each instance there was a pre-existing tuberculous focus, probably in the regional lymph nodes. In seven of these patients bronchiectasis was demonstrated, and in several other it was suspected.

The following cases are presented as illustrative of the various sites at which atelectasis can contribute to the production of bronchiectasis in children during or after primary infection.

\textit{Case 1:} R. M., girl, age 3, Mexican. The patient was symptomless. A friend of the family who lived with them when the child was six months old, had later developed open tuberculosis. On survey of the family the child was found to have a positive tuberculin test, and was brought to our clinic. Physical examination was negative except for ringworm of the scalp and a few wheezing sounds over the apices of the lungs. Coccidioidin and histoplasmin skin tests were negative. The sedimentation rate was 21 mm. in one hour (Wintrobe). Roentgenogram showed enlargement of soft tissue density at the right hilus and infiltration into the right apex and first interspace (Figure 1). Laminography showed compression of the right main bronchus from extrinsic pressure of lymph node situated between the trachea, which was pulled over to the right, and the upper margin of the right main bronchus (Figure 2). Gastric lavage was negative on two occasions for tubercle bacilli by smear, culture and guinea-pig inoculation. Nothing was advised except continued observation.

\textit{Case 2:} W. M., boy, age 6, Negro. At the age of four he entered the hospital for acute illness with fever and cough. Physical examination showed only congenital nystagmus. Roentgenogram showed a massive shadow in the right lobe (Figure 3). Sedimentation rate was 30 mm. (Wintrobe). Tuberculin test was positive. Stomach washings were positive for tubercle bacilli on culture and guinea-pig, and remained positive at intervals of three months for one year. He became rapidly symptomless and did well. When the collapsed lobe partially cleared, three small
cavities were seen (Figure 4). Sedimentation rate was now normal and stomach washings negative, and have remained so to date. Laminogram showed bronchiectasis of the pectoral segment of the upper lobe and a large hilar node pressing on the bronchus of this segment (Figure 5). The patient two years after the initial infection is symptomless except for a marked predisposition to intermittent acute respiratory infections, when rales are usually present throughout the chest.

Case 3: J. H., boy, age 7, white. Since the age of three he had intermittent acute respiratory infections, with temperature of 103 to 104 degrees F. Pneumonia was diagnosed on one occasion, and at the age of four he was under observation at a County Hospital for tuberculosis. About six months ago he began to have three to four day febrile episodes every two or three weeks. The family history revealed that a 12 year old

![Figure 1](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21216/)

**FIGURE 1**

*Figure 1: Enlargement of soft tissue density at right hilus and infiltration into right apex.*

![Figure 3](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21216/)

**FIGURE 3**

*Figure 3: Massive shadow in upper right lobe.*

![Figure 2a](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21216/)

**FIGURE 2a**

*Figure 2: Laminagram showing large lymph nodes at upper right hilus causing collapse and bronchiectasis of upper right lobe (a); with partially calcified node (b).*

![Figure 2b](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21216/)

**FIGURE 2b**
brother had been diagnosed by roentgenography as having pulmonary tuberculosis. Physical examination revealed some enlarged cervical nodes, and coarse and fine rales over the left base, and left axilla. The spleen was easily felt and reached 8 cm. below the left costal margin. There was slight clubbing of the fingers. The tuberculin test was strongly positive, coccidioidin and histoplasmin tests were negative. The sedimentation rate was 40 mm. (Wintrobe). A liver biopsy showed chronic inflammation. Roentgenographic examination of chest showed heavy shadows in left lung with retraction into the left chest. Bronchograms showed cylindrical bronchiectasis of both branches of the lingula and apical, anterior middle and posterior basal branches of the left lower lobe (Figure 6). Tubercle bacilli were isolated on culture from gastric washings.

Figure 4: Two years, 8 months after Figure 3, showing two calcified primary foci, with three small cavities medial to upper focus, and massive hilar nodes. Figure 5: Laminagram showing bronchiectasis of pectoral segment, with large, partly calcified hilar node pressing on bronchus.

Figure 6: (a) Cylindrical bronchiectasis of lingula and left lower lobe.—(b) Spot film of lingula and anterior basal segment of left lower lobe.
Cases 4 and 5: A.F. and E.A. These two girls were first reported in 1937 as cases of primary tuberculosis occurring at ages 10 and nine years, followed by bronchiectasis of the left lower lobe in both, and demonstrated by bronchograms at ages of 14 and 13. Both had clinical bronchiectasis with large amounts of sputum and long histories of severe polypoid sinusitis, neither sinusitis responding to surgical treatment. In the sputum of each tubercle bacilli were found constantly until lobectomy was performed, for the first girl in 1938, for the second in 1939. Both had very soon complete and lasting cessation of the symptoms of both bronchiectasis and sinusitis, and tubercle bacilli were never found in sputum or stomach washings following the surgical removal of the affected lobes. The first girl married and had two children, both patients were followed for over five years after lobectomy and were entirely well at the end of this period.

Discussion

In acquired bronchiectasis, the bronchial wall is injured with destruction of the epithelium, loss of cilia and mucous glands, and weakening of both elastic and muscular elements of the bronchus. The contraction of the surrounding connective tissue may contribute to this condition, and dilatation of the bronchi may be caused by peribronchial disease. The resulting stiff tubes are incapable of their normal action.

In bronchiectasis following primary infection, the mucous membrane is conceivably damaged by the infiltration of the elements associated with tuberculin hypersensitivity, which not only swell the interstices of the lung, but also surround the bronchi. The roentgen shadows caused by this process are hardly distinguishable from the true atelectasis caused by extrinsic pressure on a bronchus by peribronchial lymphangitis and lymphadenitis. The latter processes may be expansile or contractile, and may cause ulceration and penetration through the bronchial wall to produce internal occlusion of a bronchus, with subsequent collapse of that portion of a lung. The ultimate result of these different mechanisms, if not corrected, may all be bronchiectasis.

In treatment of this type of bronchiectasis the site of the disease in the lung is of some importance. In general writers on the subject seem to consider the disease in the upper lobes as usually not infectious. Many patients are without symptoms after the tuberculosis has become quiescent, but this is not always true (Case 2). When lesions are in the middle lobe symptoms may be present in childhood, or they may be postponed until adult life, with the possibility that symptoms of bronchiectasis may begin at any time during the life of the individual. In Field’s cases of non-tuberculous bronchiectasis in children the lingula was infected in 65.5 per cent, but in not a single case alone. When in the lower lobes from any cause bronchiectasis is seldom silent, and should
be considered the most dangerous to be left untreated. Sinusitis is often present with lower lobe bronchiectasis, and in the tuberculous cases there is a suggestion in some that it may be secondary to the bronchial disease (Cases 4 and 5).

The bronchiectatic lesion itself may be quite removed from the site of the active tuberculous process if the lymph-node causing the bronchial compression is one secondarily infected along the course of the lymphatic drainage, and the collapsed lobe may even be on the opposite side of the chest from the primary focus of infection, and not involve the tuberculous lobe at all.

An attempt should probably be made to expand all collapsed lobes following the initial tuberculous infection, although Field\textsuperscript{15} in non-tuberculous cases in children noted no significant difference in the incidence of expansion of the lungs between those treated with bronchoscopy and those treated without it. During the time when the tuberculous process is considered acute, whether bronchiectasis is present or not, the use of streptomycin seems logical, as advocated by Jones and Howard.\textsuperscript{16} If bronchiectasis is believed to have already occurred, postural drainage may be of value as supplement to antibiotics in various forms, depending upon the location of the lobe involved.

Surgery is certainly the treatment of choice in any case of chronic symptomatic bronchiectasis in children. The surgeon may be influenced in choosing his time by the acuteness of the tuberculous process, if it be of this type, and by the presence of tubercle bacilli in stomach washings. He need not be influenced greatly by the age of the patient if the latter has passed infancy. The affected lobe is probably a non-functioning one, and as Field states:\textsuperscript{17} "The fact that after lobectomy the remaining lung increases its functional capacity in growing children makes it desirable to operate during childhood."

Several authors quoted have noted the common incidence of bronchiectasis following the primary infection of tuberculosis, and Caffey\textsuperscript{18} in his textbook emphasizes this fact. Wallgren\textsuperscript{4} states that bronchiectases are "commoner than true tertiary pulmonary tuberculosis following primary tuberculosis. The more one is on the lookout for these postepituberculous bronchiectases the more often they will be found." I believe this fact should be re-emphasized often, and that every patient with primary tuberculosis with massive shadows, either of the parenchyma or hilar nodes, should be closely watched throughout his childhood for subsequent bronchiectasis. The use of the term epituberculosis, however, is not adequate in describing the massive shadows which are caused by several different types of lesions not too closely related pathologically.
Since the often benign primary infection may in many children be followed closely by more serious disease, we must not neglect prophylactic measures against tuberculosis in patients, including the separation of open cases of the disease from other children, nor let down in any way in our use of all our safe and proved weapons in combatting this disease.

SUMMARY

1) Bronchiectasis following the primary infection with tuberculosis is a common complication, and should be looked for in the presence of any massive roentgen shadow.

2) Bronchiectasis in the upper lobes is less apt to be symptomatic, but in other parts of the lung demands treatment.

3) Treatment should first be directed toward re-expansion of atelectasis, which predisposes to bronchiectasis.

4) Lobectomy is the treatment of choice when symptoms are present, and should be resorted to in children.

5) The term epituberculous should be dropped as being inadequate to describe shadows that have varied pathological and mechanical implications.

6) The serious character of bronchiectasis as a complication of the primary infection gives added weight to the importance of prophylaxis against tuberculosis, especially in children.

RESUMEN

1) La bronquiectasia después de la primo-infección tuberculosa es una complicación común y debe investigarse en presencia de cualquiera masa gruesa a los rayos X.

2) La bronquiectasia de los lóbulos superiores es menos frecuentemente sintomática pero en otras partes del pulmón requiere tratamiento.

3) El tratamiento debe dirigirse primero hacia la reexpansión de la atelectasia que predispone a la bronquiectasia.

4) La lobectomía es el tratamiento de elección cuando hay síntomas y debe realizarse en los niños.

5) El término epituberculous debe ser descartado como inadequado para describir sombras que tienen varias significaciones patológicas y mecánicas.

6) El carácter serio de la bronquiectasia como complicación de la infección primaria tuberculosa da un peso mayor a la importancia de la profilaxis contra la tuberculosis especialmente entre los niños.

RESUME

1) La primo-infection tuberculeuse se complique communément de dilatation bronchique. Il faut la rechercher en présence de toute ombre radiologique massive.
2) La dilatation des bronches du lobe supérieur s'accompagne moins de manifestations cliniques, mais dans les autres parties du poumon, il y a lieu d'envisager un traitement.

3) Ce traitement doit d'abord être dirigé contre l'atélectasie qui préddispose à la dilatation des bronches.

4) La lobectomie est le traitement de choix quand la dilatation des bronches s'accompagne de manifestations cliniques et devrait être appliquée aux enfants.

5) Le terme d'épitéuberculose devrait être supprimé, car il ne rend pas compte des causes anatomiques et mécaniques variées qui sont à l'origine des ombres radiologiques.

6) La gravité des bronchiectasies qui compliquent la primo-infection, donne un argument de plus à l'importance de la prophylaxie contre la tuberculose, plus particulièrement chez les enfants.

REFERENCES