The Management of Uncontrolled Pulmonary Tuberculosis Complicated by Endobronchial Tuberculosis*

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Endobronchial tuberculosis as a complication of pulmonary tuberculosis has been recognized by pathologists for many years. However, its clinical importance was not appreciated until the last 10 years, and relatively little attention has been paid to the manner in which this complication affects the treatment of the associated parenchymal disease, particularly with artificial pneumothorax and other collapse measures.

Endobronchial tuberculosis is essentially a problem of mechanical obstruction with interference of the bronchial drainage in a patient with pulmonary tuberculosis, and atelectasis, spread of tuberculous disease, pyogenic infection, bronchiectasis and obstructive emphysema are therefore frequent complications of the disease. This may occur in any of three manners, or in a combination of them: (1) through gross obstruction by granulation tissue in the acute phase or by chronic cicatricial fibrostenosis; (2) through partial obstruction accompanied by stagnation of secretions, owing to loss of ciliary action and bronchial peristalsis, and to increased viscosity of sputum; (3) to exaggerated mobility of the bronchial wall, due to localized cartilaginous weakening with probable destruction and producing a check-valve mechanism with resultant trapping of air. In the production of obstruction with stagnation of secretions the following factors are at work: edema and thickening of the mucosa and other layers of the bronchial wall during the acute phase interfere with ciliary action and peristalsis; extensive involvement of mucous gland may decrease mucous formation, thus resulting in increased viscosity of the sputum, making it difficult to raise. Pathologic secretions from the parenchymal disease thus may stagnate at or beyond the disease area in the bronchus.

Since both acute and chronic endobronchial tuberculosis cause obstruction it is fair to assume that the morbid effects from such obstruction depends on the location and size of the involved

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bronchus, as well as on the degree and duration of the obstruction.

The management of uncontrolled parenchymal disease complicated by endobronchial tuberculosis is the most important therapeutic problem and is essentially this: By what method may the parenchymal disease be controlled with the least danger of producing bronchial obstruction and its sequelae?

Some authors have reported good results with pneumothorax, with symptomatic improvement and sputum conversion, but the ultimate outcome of these cases in regard to re-expansion and the status of the pleural cavity were not mentioned. Our own results were poor; from a total of 206 consecutive cases of endobronchial tuberculosis, in 36 patients pneumothorax was used in an effort to control parenchymal disease. Unfavorable complications occurred with great frequency and atelectasis was noted immediately or shortly after induction of pneumothorax in 20 patients (55 per cent) and in 14 of these cases re-expansion was impossible even when attempted early. Sixteen of the total of 36 cases death was due to progressive tuberculosis, including pleural form, or anaerobic infection of the atelectatic lung or a combination of the two, but in all the cases the beginning of the downhill course could be linked with induction of pneumothorax.

Accordingly with our experience I believe the following general plan of therapy may be suggested. Whenever diagnosis of endobronchial tuberculosis having been made, pneumothorax is contraindicated temporarily, and primary attention is paid to the treatment of bronchial lesion. Bed rest, streptomycin, bronchoscopic treatment of the ulcerations and aspiration of secretions are advisable for two reasons: (1) it may do away with the need for collapse therapy; (2) if, after this treatment the parenchymal disease remains a threat to life and collapse therapy is necessary, the bronchial lesion may be healed or become indolent, and under such circumstances the incidence of complications is greatly reduced.

The choice of procedure depend primarily on the character, extension and localization of the bronchial lesion. If the ulceration is minimal and superficial and is located in the main bronchus or, if moderately extensive, has responded readily to local treatment and there is no narrowing of the bronchial lumen, pneumothorax may be used with safety. If ulceration has healed but has left a residual cicatrical stenosis, narrowing the bronchial lumen by 30 or more per cent, pneumothorax generally should not be the procedure of choice because the stenosis indicates a permanently defective bronchial drainage and immediate complications, including atelectasis, are frequent. In these cases,
Thoracoplasty offers much better hope from a long-range viewpoint. It will control the parenchymal disease in the majority of cases; it reduces the size of pulmonary bed and reduces to a minimum the danger of late suppuration; and eliminates pleural complications.

These contraindications to pneumothorax should be disregarded only in those patients with minimal parenchymal disease so located as not to be amenable to permanent surgical collapse. Pneumothorax should then be induced only after weighing of the dangers of possible complications against those of the inadequately controlled disease.

If pneumothorax has been induced prior to the diagnosis of bronchial disease the management of the case must depend largely on the clinical course of the patient up to that time. In those patients who develop atelectasis and pleural effusion, one must be careful in re-expanding the lung and if a change of therapy is indicated, it is better to do the thoracoplasty over pneumothorax, than open new areas for supuration through re-expansion. In those patients who fortunately have not developed these serious complications, the safest course seems to be maintenance of pneumothorax with the expectation of an unexpandable lung. In cases with pleural adhesions the general principles recommended above should be applied; if the bronchial lesion is minimal or, if moderately extensive appears indolent and there is no narrowing of the bronchial lumen, pneumonolysis may be performed, provided there are no contra-indications. However, if the bronchial disease is extensive with partial stenosis of the bronchial lumen, and particularly, if there is atelectasis or evidence of retained secretions, pneumonolysis may be harmful and followed by complete obstruction of the bronchus and its serious sequelae.

The occurrence of atelectasis with pneumothorax is due to the presence of air about the root of the lung. This results in interference with normal respiratory motions of the major bronchi in addition with moderate decrease in their size and change in their position. On the other hand, the collapse produced by thoracoplasty is not concentric but is most marked away from the hilum and its effect on the position, the physiologic action, and the functional efficiency of the major bronchi appears to be minimal.

A relatively small number of cases, particularly those with severe fibrostenotic obstruction and pulmonary suppuration, cannot be safely or effectively treated by thoracoplasty, or a good thoracoplasty may already have been performed without producing a satisfactory result. For such patients pulmonary resection constitutes the only effective method of treatment.
SUMMARY

The presence of endobronchial tuberculosis confuses the prognosis and greatly complicates the treatment of the parenchymal lesion. Atelectasis, anaerobic infection, progressive tuberculosis, empyema, and unexpandable lung occur with great frequency when pneumothorax is used. Therefore, pneumothorax should be considered as contraindicated in all but the minimal cases of bronchial tuberculosis, or those patients in whom the bronchial lesion has healed without appreciable stenosis. Thoracoplasty should be the procedure of choice in those patients in whom pneumothorax is contraindicated. In other cases pneumonectomy and lobectomy may be the only chance of restoring health.

RESUMEN

La tuberculosis endobronquial es esencialmente un problema de obstrucción mecanica que interferie el drenage normal del arbol bronquial, confundiendo el pronostico y haciendo más complicado el tratamiento de la lesion parenquimatosa.

El neumotorax terapeutico lleva con frecuencia en estos casos a complicaciones tales como: atelectasia, infección anaerobica sobreañadida, agravanación y progresión de la lesion pulmonar, empiema pleural, pulmon inexpansible, etc.

El neumotorax por lo tanto debe considerarse contraindicado excepto en los casos de tuberculosis bronquial minima sin obstrucpción o en aquellos casos en que la lesion bronquial ha curado sin dejar estenosis apreciable.

Parece ser que la toracoplastia es el procedimiento de elección en la mayoría de los casos en que el neumotorax esta contraindicado. En ciertos casos solo la lobectomia o neumonectomia constituyen el único metodo efectivo de tratamiento.