Infection of the residual pleural space fifty-nine years after artificial pneumothorax for pulmonary tuberculosis.

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ABSTRACT: A 79-year old man with serious comorbidities developed empyema of the residual pleural space that was the sequela of collapse therapy with artificial pneumothorax performed 59 years ago for pulmonary tuberculosis. Drainage of the infected pleural space through open-window thoracostomy resulted in reversal of systemic toxicity and full recovery.

Key Words: Tuberculosis, Collapse therapy, Artificial pneumothorax, Late complications of collapse therapy, Pleural infection.

INTRODUCTION

“Collapse therapy” was extensively used during the first half of the previous century for the treatment of pulmonary tuberculosis. Artificial pneumothorax, thoracoplasty, one of the various available plombage techniques and phrenic nerve crash were the available techniques to collapse the diseased part of the lung. All these techniques were abandoned since the development of effective antituberculous chemotherapy in the mid-fifties. Artificial pneumothorax was the commonest way to perform “collapse therapy” because of its simplicity, minimal invasiveness and better preservation of overall pulmonary function.

The late complications of collapse therapy for pulmonary tuberculosis are well known from the past. Currently, these complications are rarely seen because these techniques were abandoned more than fifty years ago. A recent case of pleural empyema with Escherichia Coli which has developed 59 years after the creation of artificial pneumothorax for the treatment of pulmonary tuberculosis is presented. The possible mechanisms of residual pleural space infection and the therapeutic options of this rare clinical entity are discussed.

CASE PRESENTATION

A 79-year old man was urgently admitted in the Department of Internal Medicine with clinical signs of systemic inflammatory response. He was recently treated with oral administration of ciprofloxacin for 14 days for urinary infection with Escherichia Coli. Urinary infection was detected one month before, when the patient was examined in the outpatient department for complaints of malaise and weight loss. The patient had history of prostate hypertrophy, arterial hypertension, coronary artery disease and acute myocardial infarction that occurred before 14 years. He underwent surgery for myocardial revascularization seven years ago.

At admission the patient had low \( \text{paO}_2 \) (57 mmHg), low oxygen saturation (87%) on breathing room air and hypercarbia (\( \text{paCO}_2 = 55 \text{ mmHg} \)), while arterial blood \( \text{ph} \) was normal (\( \text{ph} = 7.39 \)). Hypoxemia was partially reversed with the administration of oxygen. The patient had also anaemia (Ht: 27%, Hb: 8.8g/dl, MCV: 65.5, MCH: 21.1), low serum ferrum levels (Fe = 15 mg/dl) and normal serum ferritin levels.

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White blood counts and serum levels of C-reactive protein and erythrocyte sedimentation rate were considerably elevated. Urine test and culture were negative. Mantoux test was negative and his electrocardiogram showed the presence of Q wave in II, III and AVF leads. Doppler echocardiography revealed right ventricle dilatation, pulmonary hypertension (right ventricular systolic pressure: 60 mmHg) and preservation of left ventricular function (EF = 50%). Spirometry results showed a severe restrictive pattern (FVC: 0.96L, FEV₁: 0.75L, FEV₁%: 77%).

Plain chest radiography at admission demonstrated extensive opacity of the left hemithorax with deviation of the trachea to the left side and complete collapse of the left lower lobe. (Figure 1a) Chest computed tomography showed a fluid-filled residual pleural space enclosed within a calcified pleural envelop. (Figure 1b) A small air-fluid level was observed within the loculated pleural effusion. (Figure 1b).

Thoaracentesis of the pleural effusion revealed purulent fluid. The Gram stain of pleural fluid has detected the presence of a Gram negative microorganism which at culture was found to be Escherichia Coli. The patient received Timentin 5.2 g every 8 hours and chemoprophylaxis against tuberculosis with the twice-daily administration of tablets combining rifampicin (300 mg) and isoniazid (150 mg). In addition, the patient underwent chest tube drainage of the loculated pleural collection. Chest tube drainage of the space was considered to be inadequate and the patient was finally submitted to open-window thoracostomy which was performed in the lowest portion of the pocket with partial resection of two ribs under local anaesthesia. The late manoeuvre resulted in complete drainage of the pleural space, complete subsidence of the infectious process and re-expansion of lung parenchyma to a large degree. Cultures and polymerase chain reaction of the pleural fluid failed to detect the presence of Myobacterium and consequently antituberculous chemotherapy was discontinued after three weeks.

Because of the poor performance status of the patient, a permanent open-window thoracostomy was considered to be the appropriate solution for the infected residual pleural space. The patient recovered well and he still remains in relatively good clinical condition three years later, while he recently underwent operative repair of a large inguinal hernia under local anaesthesia.
DISCUSSION

Artificial pneumothorax for the treatment of tuberculosis was introduced in Italy during 1882 by Carlo Forlai, while it became widely adopted in Europe and in the United States of America quite later, after 1912. However, the first report belongs to James Carson from Liverpool who first had the idea to create an artificial pneumothorax for the treatment of pulmonary tuberculosis in 1822. The “philosophy” of collapse therapy was to produce compressive atelectasis of the diseased portion of the lung and to “encapsulate” the disease within the atelectatic lung. By putting the diseased part of the lung at rest with “collapse therapy”, the healing process was promoted and the spread of the tuberculous material to the non-diseased parts of both lungs was avoided. Multiple adhesions between pleura and lung that could prevent the lung to effectively collapse were considered to be a contraindication for “collapse therapy” until Jacobaeus has introduced thoracoscopy in 1910 for breaking-down these adhesions (pneumolysis), allowing that way the lung to effectively collapse. Multi-drug resistant tuberculosis, intolerance of antituberculous drug treatment because of toxicity and the high incidence of tuberculosis observed in the HIV patients could involve the renaissance of artificial pneumothorax as part of the modern treatment of tuberculosis in the above mentioned groups of patients.

After the application of artificial pneumothorax for tuberculosis, the lung sometimes did not completely expand because of extensive fibrosis of lung parenchyma and/or excessive pleural thickening and calcification, as in the presented case. A residual pleural space can be left behind because of partial re-expansion of the lung that is normally filled with fluid which by the progress of time is transformed to a fibrin gel. The residual space usually remains stable in size and sterile for life-long. Occasionally, an increase in the size of the residual pleural space that appears radiographically as an extensive opacification of the hemithorax can be observed, even several decades later. The phenomenon of progressive swelling of the residual pocket is called the “exudative process”. Late tuberculous empyema is rarely the cause of “exudative process”. Most common causes for the development of this process are bronchial and esophageal fistulas which lead to infection of the space or the development of pleural lymphoma.

A point that needs special attention in the presented case is that infection of the residual pleural space with Escherichia Coli was associated with a recent urinary infection with the same micro-organism. The empyema could be attributed to a small bronchopleural fistula as suggested by the small air-fluid level seen within the pocket on CT scan done at admission. Hematogenic spread of the urinary infection within the residual pleural cavity could be another, unusual cause of empyema formation. Whatever the mechanism of residual pleural space infection, it represents a serious and life-threatening complication for the old, debilitated and suffering from serious comorbidities patient who underwent artificial pneumothorax for the treatment of his pulmonary tuberculosis several decades ago.

The treatment options of late infection of the residual pleural space after artificial pneumothorax are thoracotomy and decortication of the lung (with or without the addition of thoracoplasty that mainly depends on the post-decortication expansion of the lung parenchyma), extrapleural pneumonectomy in case of totally destroyed lung and open window thoracostomy. The later way is the preferable option to drain the heavily calcified, infected pleural space in the high-risk for major intervention, old patient with serious comorbidities; it offers eradication of the infection without operative risk, while by the progress of time, the daily care of permanent thoracostomy window becomes very easy and the patient is able for self-care.

In conclusion, late infection of the residual pleural space should be kept in mind when patients who underwent artificial pneumothorax for the treatment of pulmonary tuberculosis many years before deteriorate and their plain chest radiography shows considerable changes, with the form of extensive opacification of the hemithorax or swelling of a stable for many years pleural pocket. Infection of the residual pleural space can be successfully treated by the creation of a permanent open-window thoracostomy that can be easily performed under local anaesthesia and without significant operative risk.
Φλεγμονή της υπολειμματικής υπεζωκοτικής κοιλότητας 59 χρόνια μετά τη δημιουργία “τεχνητού” πνευμοθώρακα για πνευμονική φυματίωση.

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ΠΕΡΙΛΗΨΗ: Ένας άνδρας 79 ετών με επιβαρυμένο ιατρικό ιστορικό, εμφάνισε εμπύημα σε υπολειμματική υπεζωκοτική κοιλότητα, η οποία αποτελούσε κατάλοιπο «τεχνητού» πνευμοθώρακα που είχε γίνει πριν από 59 χρόνια για τη θεραπεία πνευμονικής φυματίωσης. Η παροχέτευση της κοιλότητας με ανοιχτή θωρακοστομία είχε σαν αποτέλεσμα την αναστροφή της τοξικής κατάστασης και γρήγορη ανάρρωση.

Λέξεις Κλειδία: Πνευμονική φυματίωση, Θεραπεία σύμπτωσης του πνεύμονα, Τεχνητός πνευμοθώρακας, Απώτερες επιπλοκές της θεραπείας σύμπτωσης του πνεύμονα, Εμπύημα θώρακα.

REFERENCES