Short reports

Late complication of plombage thoracoplasty

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Before specific chemotherapy the most successful type of surgery for pulmonary tuberculosis evolved from the idea of putting the affected part to rest by allowing the lung to collapse.

Artificial pneumothorax received widespread popularity after the report of successful series by Forlanini in 1948' and Murphy in 1941.² Phrenic nerve paralysis and pneumoperitoneum were suggested by Stuertz and Vajda respectively.³⁴ The standard thoracoplasty was used when other procedures failed to collapse the tuberculous pulmonary cavity.⁵

Plombage was introduced as an alternative to standard thoracoplasty. A subcostal pocket was developed by stripping the periosteum off the ribs overlying the diseased area and by filling this pocket with a plomb, such as a polythene bag, paraffin, or lucite balls.⁶ This operation had the advantage of permitting selective collapse of the diseased lung with less derangement of pulmonary function than standard thoracoplasty; the procedure could be carried out in one stage and there was less deformity.⁷

The purpose of this paper is to describe the natural history of a plomb inserted in 1951 and to report a serious late complication that has not be described before.

Case report

The patient, now 58 years old, was diagnosed as having cavitating pulmonary tuberculosis of the left upper lobe in 1949. She was admitted to a sanatorium in North Wales and remained there from March 1950 to July 1951. During this period a left artificial pneumothorax was performed, but this failed because of extensive apical pleural adhesions. Subsequently left phrenic nerve crush and pneumoperitoneum were performed. These two procedures also failed to achieve an acceptable degree of collapse of the left upper lobe cavity. Plombage was then performed and the tuberculous cavity collapsed completely. She improved and was discharged with outpatient follow up.

In 1975 she was symptom free. Radiographs showed the plomb to be solid and intact but the ribs overlying it appeared to be atrophic, osteoporotic, and smaller than the other ribs (fig 1).

In July 1981 she felt unwell and complained of a productive cough with green sputum, persistent fever, and chest pain in the left axillary region. Sputum examination

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showed no tubercle bacilli but grew *Haemophilus influenzae*. Radiographs showed the plomb to be bigger in size and to contain multiple small air filled spaces, and the ribs in contact with the plomb to be atrophic and osteoporotic (fig 2). A course of antituberculous treatment was started but the pyrexia and malaise persisted. The antituberculous treatment was stopped and the patient given doxycycline. She improved and the pyrexia settled.

In October 1981 the patient complained of persistent left axillary pain and a lump appeared in the left axilla. She refused to have a biopsy of the mass but bronchoscopy showed no endobroncial lesion; bronchial washings were negative for malignant cells and did not grow tubercle bacilli on culture.

In July 1982 the axillary mass became inflamed and frank pus was aspirated. *H influenzae* was cultured and no acid fast bacilli were isolated. She was put on a course of doxycycline. Although the inflammation settled a chronic discharging axillary sinus persisted.

In early July 1984 she was admitted to hospital with bleeding from the axillary sinus and increasing left sided axillary pain. A sinogram showed that the sinus was communicating with the plomb cavity. At this stage the patient rejected advice to have an exploratory thoracotomy and discharged herself.

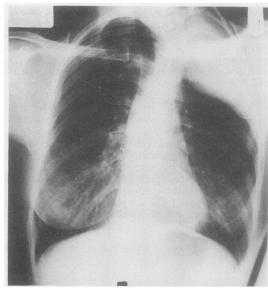


Fig 1 Chest radiograph in 1975 showing the intact plomb in the left upper zone. The overlying ribs are atrophic.

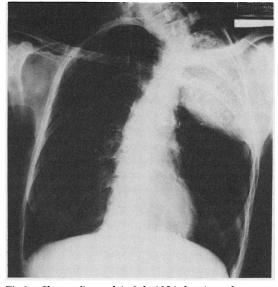


Fig 2 Chest radiograph in July 1981 showing enlargement of the plomb, which contains multiple small air filled spaces.

In mid July 1984 she was admitted to another hospital with increasing shortness of breath and haemoptysis. Ventilation and perfusion scans showed multiple mismatched defects in both lung fields and she was given anticoagulation treatment with heparin and warfarin.

In early August she was admitted with massive haemoptysis and profuse bleeding from the axillary sinus. The haemoglobin concentration had dropped from 13 g/dl to 9 g/dl and the British corrected ratio (BCR) was 1.6. As she was extremely dyspnoeic and shocked, she was ventilated and given a transfusion; the warfarin was stopped and vitamin K given. The haemoptysis gradually subsided and her general condition improved.

Left thoracotomy was then performed. The plombs consisted of two polythene bags. Both were tense and full of blood, air and plastic sheets and were surrounded by blood clot. The bags were found to have multiple holes in them. They were eroding the lung and the chest wall and communicating with the axillary sinus. The plombs were removed with relative ease but after removal of the surrounding clot massive bleeding occurred; it was coming from an erosion in the aortic arch about 0.5 cm in diameter. The erosion in the aorta was closed and a four rib thoracoplasty with removal of the first rib was performed. The plombs, with their blood contents, were sent for bacteriological examination. No acid fast bacilli were cultured or seen on direct examination but H influenzae and diphtheroids were grown. After operation the patient made an excellent recovery and the left lung maintained full expansion. She was seen in the outpatient clinic two months later and her general condition was found to be satisfactory. The left lung was clear and fully expanded.

Discussion

The plombs in this patient appeared to have eroded through the periosteum, intercostal muscle, endothoracic

fascia, and parietal pleura and settled on the left upper lobe. By July 1981 there was radiological evidence of deterioration in the plombs in that multiple air spaces appeared in them, presumably owing to a combination of holes in the plombs (as a result of the wear and tear process) and erosion of the lung.

The atrophy and osteoporosis of the ribs overlying the plomb is most probably due to a combination of the pressure of the plomb and the fact that these ribs were denuded of the periosteum when the plomb was inserted. The periosteum does not regenerate appreciable amounts of bone when it is in contact with plomb material.⁷ It is surprising that these ribs did not undergo necrosis as they had been denuded of the periosteum and compressed by the plomb.

Entry of air into the plombs may have brought about increased distension within them, causing compression of the chest wall and leading to the persistent axillary pain associated with the erosion of the plomb through the chest wall and formation of the axillary mass and later the axillary sinus. Air entry to the plomb would also have led to contamination with pathogenic organisms from the respiratory tract, infection, and sinus formation. The combination of compression and infection must have also led to the erosion of the aortic arch, producing massive haemoptysis and profuse bleeding from the axillary sinus. Anticoagulation would have played a part in the continuation of the bleeding but obviously was not the prime cause of it.

Many other complications of plombage have been reported—for example, bronchopleural fistulas, space infections, and wax migration into soft tissues outside the ribs and formation of paraffinomas.⁸ There have, however, been no reports of erosion by a plomb into the aorta. Haemoptysis associated with plombage should therefore be regarded seriously and the plomb removed as a matter of urgency.

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