Spontaneous Pneumothorax Due to Diaphragmatic Defect Complicating Pneumoperitoneum Therapy

Report of a Case

ISAAC EPSTEIN, M.D., F.C.C.P.*
Temple, Texas

Complications which occasionally occur in pneumoperitoneum have been reported by different authors.1-22 Mellies14 in 1939 described a case of right sided pneumothorax in a 16 year old female as a result of a perforation of a tuberculous lesion of the diaphragm. Smith21 in 1943 reported a death from bilateral pneumothorax during a diagnostic pneumoperitoneum. Necropsy of his case revealed small defects connecting the peritoneal with both pleural cavities. Air entering the pleural cavity from the peritoneum by migrating along the mediastinal structures have been described by Simmonds,20 Moyer,13 Banyal1 and others. Lumsden12 recently described a case of pneumothorax complicating artificial pneumoperitoneum in a 38 year old woman. He believed that air entered the pleural cavity from the peritoneum through a pleuro-peritoneal canal.

Case History

L.J.B., No. 7726: Negro male, 21 years of age, was admitted to the Veterans Administration Center, Temple, Texas, on May 31, 1948, for treatment of pulmonary tuberculosis with symptoms of two months duration. A marginal pneumothorax on the left side was induced in April 1948 (Figure 1).

Physical examination revealed a well-developed and well-nourished Negro male who did not appear acutely ill and the positive findings in this case were limited entirely to the chest. The serology, urinalysis and complete blood count on admission were essentially negative; sputum examinations were continuously positive for acid-fast bacilli; initial roentgenogram of the chest revealed about 30 per cent collapse of the left lung with a patent cavity in the dorsal division of the lower lobe. The right lung was clear and well-aerated.

The pneumothorax on the left side was discontinued as ineffective on July 16, 1948, and pneumoperitoneum, supplemented with left phrenic crush, was instituted. Bronchoscopy showed evidence of tuberculous bronchitis and a 42 day course of streptomycin therapy of 0.5 grams daily, was begun July 29, 1948. Because of relapse of the endobronchial

*Chief of Thoracic Surgery, Veterans Administration Center, Temple, Texas. Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the author are the result of his own study and do not necessarily reflect the opinion or policy of the Veterans Administration.
Figure 1: Roentgenogram, taken after admission, showing marginal pneumothorax, left, with an open cavity in the lower lobe.

Figure 2: Roentgenogram taken April 8, 1949, showing extensive pneumoperitoneum. Pulmonary cavity still open.

Figure 3: Postoperative film after resection of the left lower lobe and partial thoracoplasty.
disease, he was given a second course of streptomycin with the dosage increased to 1 gram daily for an additional 42 days. The cavity in the dorsal division of the lower lobe, however, remained in tension and did not respond to the induced collapse, as well as to the antibiotic therapy (Figure 2).

A resection of the left lower lobe with partial thoracoplasty was performed on April 13, 1949. The postoperative course was uneventful except for transient atelectasis of the right upper lobe. Pneumoperitoneum was continued, with weekly refills of approximately 1,200 cc. of air (Figure 3).

On August 25, 1949, the patient received a refill of 1,050 cc. of air in the peritoneal cavity with a final reading of plus 18. In the morning of August 29, 1949, he experienced a sudden pain in the right side of the chest with dyspnea. In a matter of a few minutes he became orthopneic, pulseless and almost moribund. Examination of the chest revealed marked hyperresonance to percussion on the right side with displacement of the heart to the left. Immediately 2,700 cc. of air were removed and supportive treatment was given. His pulse promptly improved, consciousness was restored and recovery ensued. Fluoroscopy revealed 30 per cent collapse of the right lung with markedly diminished pneumoperitoneum in comparison with the previous examinations. A tube, connected with a water trap, was inserted into the right pleural cavity and after 72 hours the right lung re-expanded and the tube was removed (Figure 4). Following this complication, pneumoperitoneum was continued cautiously with small refills at frequent intervals.

On September 13, 1949, a few hours following a refill, he again experienced the same signs and symptoms as above and the same result followed deflation. Fluoroscopy as well as x-ray inspection (Figure 5) again showed a decrease in the amount of pneumoperitoneum and marginal pneumothorax on the right side. It was decided, therefore, to discontinue pneumoperitoneum and an attempt was made to continue with right pneumothorax. Thoracoscopy was done on October 12, 1949, and the adhesions were found to be unseverable. An attempt was made to inspect the possible defect in the right side of the diaphragm, but the patient became progressively dyspneic, necessitating discontinuance of thoracoscopy and immediate withdrawal of air from the right pleural space.

In view of the above complications, it was decided to discontinue for the time being every type of collapse therapy and the patient was placed on strict bed rest regime alone. He continued to improve, sputum became negative for acid-fast bacilli in smears, cultures and guinea-pig inoculation, and he was discharged August 25, 1950, as an arrested case of pulmonary tuberculosis (Figure 6).

Discussion

There is no question that our patient had a leakage of air from the peritoneal cavity into the right pleural space. A thorough check on any possible breach in technique did not reveal any accountable errors. An attempt, however, to visualize the right diaphragmatic defect through the thoracoscope was unfortunately unsuccessful because of the increased dyspnea.

There was no reverse flow of air, probably because of lack of negative pressure in the peritoneal cavity, lack of positive pressure
Figure 4: Note catheter in the right pleural cavity and loss of pneumothorax on the right side.

Figure 5: Roentgenogram of the chest prior to discharge.

Figure 6: Note pneumothorax and adhesions.
in the pleural cavity, pressure of the liver on the diaphragm, and a possible check-valve-like defect in the diaphragm.

SUMMARY

1) A case of spontaneous pneumothorax occurring during pneumoperitoneum therapy is described.

2) Congenital diaphragmatic defect was apparently the cause of leakage of air from the peritoneal cavity into the right pleural space.

RESUMEN

1) Se describe un caso de neumotórax espontáneo que ocurrió durante el tratamiento con neumoperitoneo.

2) Aparentemente la causa del paso del aire de la cavidad peritoneal a la pleural, fue un defecto congénito del diafragma.

RESUME

1) L'auteur rapporte un cas de pneumothorax spontané survenant au cours d'un traitement par pneumopéritoine.

2) Une brèche congénitale du diaphragme était apparemment l'origine du passage de l'air de la cavité péritonéale dans la plèvre droite.

REFERENCES


