Report of a Case of Right Pneumothorax Following Induction of Pneumoperitoneum*

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With the increasing indications of therapeutic pneumoperitoneum and with the wider application of this method for the treatment of pulmonary tuberculosis in recent years, reports of various complications of this procedure are accumulating in the medical literature.

One of these complications is the appearance of right pneumothorax during pneumoperitoneum treatment. This complication has been reflected in medical literature during the last decade and the number of reported cases exceeds 20. In this paper we would like to report one more case of a right pneumothorax which occurred immediately after the induction of pneumoperitoneum and which differs in many respects from the cases already published.

Case Report: M.M., No. 4532. History: 34 year old white female admitted to the sanatorium on September 30, 1951 with the history of known pulmonary disease since June 1951, when she, following measles pneumonia, had a miniature x-ray film taken by a mobile unit which was reported to show evidence of pulmonary disease. This proved to be active pulmonary tuberculosis and she was admitted for treatment.

Family History: Parents, two sisters and six brothers living and well. No tuberculosis in family. Personal History: Mumps, chicken pox and whooping cough in childhood; measles in April 1951, complicated by pneumonia in April 1951. She had four pregnancies—three normal deliveries and one miscarriage. Three children are living and well. From January to June of 1951 she was a contact to two men found suffering from active tuberculosis, who were living in the same house and were using the same stairway.

On admission, examination revealed a rather emaciated white female, 163 cm. (5 feet 4 inches) tall and weighing 42.13 kilograms (93 pounds) who offered no complaint except a slight productive cough and occasional chest pain. On physical examination the abnormal findings were: pallor of skin and mucous membranes, a slightly decreased resonance over the left apex on percussion, and few fine posttussive rales over the spine of the scapula and supraclavicular fossa at the end of expiration on auscultation. All other systems were essentially normal.

Chest x-ray film on admission revealed density over the left apex, extending to the lower border of the anterior projection of the first rib, with several small highlights, suggestive of cavitation. The remainder of the lung fields were normal. The left costophrenic sinus was slightly blunted. The right diaphragm was tented in the mid portion. The heart was within normal limits (Figure 1).

Sputum was positive on direct smear. Hemoglobin, 72 per cent; red blood cells, 4,430,000; white blood cells, 4,800. Differential: neutrophiles, 70; lymphocytes, 27; monocytes, 3; sedimentation rate, 18 mm. Urine: no sugar, no albumen; sediments within normal limit. Blood: negative for syphilis.

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Course in the Sanatorium: Her temperature ranged between 98 and 99 degrees F.; pulse between 70 and 96; respiration between 18 and 24 per minute. Amount of sputum was one-fourth cup daily. Her appetite was fair. Bowel movements were normal.

On October 11, 1951 induction of pneumoperitoneum by one of us (C.V.) was done under local novacain anesthesia with an 18 gauge needle which was inserted in the left upper quadrant. Five hundred cc. of air were given with a slight (plus 2) positive reading at the end of insufflation. Immediately after introduction of air, she complained of slight pressure in the right chest and pain in the right shoulder, which was considered as a normal pneumoperitoneum reaction. The day of pneumoperitoneum induction she was not fluoroscoped. In the afternoon she complained of slight dyspnea and tightness in her right chest. There was an absence of substernal pain, pressure and other signs of mediastinal emphysema.

The following day, fluoroscopy (H.H.B.) revealed the complete absence of air in the peritoneal cavity and was refilled with 700 cc. (H.H.B.). Initial reading was not obtained. She was still complaining of shortness of breath, slight pain and tightness in the right chest. Fluoroscopy a day after, on October 13, 1951, by both of us, again revealed an absence of pneumoperitoneum but the presence of a right pneumothorax with 40 per cent collapse. As she was complaining of only slight dyspnea, the air was not removed and after a reasonable period of time left pneumothorax was started and has been maintained to date. The right lung has completely re-expanded (Figure 2).

Comment: This case resembles those previously published—pneumothorax occurred during pneumoperitoneum treatment and developed on the right side. However, it differs in many respects from the previous cases of this kind. Many authors have pointed out the importance of the length of time between institution of pneumoperitoneum and development of complications as a condition which predisposes this complication. Due to prolonged pneumoperitoneum treatment, often associated with phrenic crushes, the stretching of the diaphragm, atrophic and degenerative processes of the diaphragmatic muscle and formation of blebs on the diaphragmatic surface may occur. All of these processes and especially the formation of blebs facilitate the formation of hernias and shunts between peritoneal and pleural cavities with the escape of air.1-3 For instance this

FIGURE 1

FIGURE 2

Figures 1 and 2: There is a right-sided pneumothorax which appears to be seen on the left side because x-rays at this institution are taken in a different manner.
complication occurred after one year of pneumoperitoneum treatments in the case of Banyai and Jurgens⁴; in the case of Yannitelli and associates¹ it occurred 10 months after induction of pneumoperitoneum and 20 days after the last refill; in the case of Ross and Farber² between the institution of pneumoperitoneum and occurrence of right pneumothorax more than one year elapsed, and in one of these cases phrenic crush was performed. In the case of Repa and Jacobson,³ pneumothorax occurred after 14½ months after pneumoperitoneum was started, a year after a phrenic crush and 13 days after the last refill. In our case the time interval was absent.

Although right sided pneumothorax was discovered only 48 hours after pneumoperitoneum induction, pain in the right chest and dyspnea almost immediately after the induction of pneumoperitoneum, and the absence of air in the peritoneal cavity 24 hours after the induction, make very probable the possibility of pneumothorax development immediately following the beginning of treatment. The factors mentioned by some of the authors¹-³ as—a prolonged pneumoperitoneum treatment, phrenic crush, continuous stretching of the diaphragm, degeneration, atrophy, and inflammation of the diaphragm and pleb formation—do not apply to our case. Anderson’s explanation, accepted by some authors¹-³ as one of the possible mechanisms of pneumothorax formation after pneumoperitoneum, does not apply to our case. Anderson⁵ explains the tendency for penetration of air from the peritoneum into the pleural cavity by the differences in pressures in both cavities. According to Anderson’s opinion the intrapleural pressure increases immediately after pneumoperitoneum refills and gradually drops during the following days. The longer the time interval after pneumoperitoneum refills, the greater the difference of pressures in both abdominal and thoracic cavities and the greater is the possibility of pleuropneumothoracic shunt formation. Also the absence of symptoms and signs of mediastinal emphysema and the presence of right pneumothorax (spontaneous pneumothorax, accompanying mediastinal emphysema, is almost always located on the left side according to Dickle, cited by²) makes improbable the possibility of the mechanism given by Banyai⁴ for the explanation of the complication under discussion. (Banyai thinks the air enters the mediastinum along the large mediastinal structures, causing mediastinal emphysema, and then pneumothorax, by escaping from the mediastinum into the pleural cavity.)

There are no indications or leads in the history indicative of some previous inflammatory process (e.g. subdiaphragmatic abscess), which might have explained the weakness of the diaphragm. Also completely excluded is the possibility of entering the pleural space by the 18 gauge needle⁴—(Pneumoperitoneum was induced on the left side)—or any other kind of injury or trauma.

Many authors consider increased physical activity or physical strain as a predisposing factor for the development of this complication. Physical
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strain and activity were made partially responsible for this complication\(^1\)\(^-\)\(^3\) and also taken into consideration in almost all previously reported cases. There was no activity, however, in our case. Since admission the patient has been on absolute bed rest.

Our case resembles that published by Wu and Neptune\(^6\) and like them we are inclined to explain it by the presence of a congenital diaphragmatic defect. The displacement of the liver due to the pneumoperitoneum procedure and stretching of ligaments might have led to widening of a pre-existing diaphragmatic defect and permit the escape of air directly from the peritoneal to the pleural cavity. The fact of liver displacement and stretching of ligaments might explain the occurrence of pneumothorax on the right side.

According to Meyer,\(^7\) the weak points in the diaphragm which are the most common sites of diaphragmatic hernias, and thus might be a source of direct communication between the peritoneal and pleural cavities, are:

1) Foramen of Bochdalek, which is a pleuro-peritoneal hiatus, situated dorso-laterally. The apex is curved toward the central tendon, upward and forward, and the base is turned downward and backward and partially rests on the 12th rib.

2) Outer crus. There may be a triangular space which separates the muscle fibers of the outer crura of the lumbar portion and the last muscular portion of the costal portion of the diaphragm. This space is filled by connective tissue and covered above by the pleura and below by the peritoneum. The liver plays a protective role on the right, and hernias through this opening occur mostly on the left. We think that the displacement of the liver in pneumoperitoneum may remove this protection and facilitate the formation of right hernia and shunt.

3) Foramina of Morgagni or Larrey's spaces, which are the spaces on both sides of the xiphoid process due to the failure of fusion of the sternal portion with the costal portion of the diaphragm. They are filled with the areolar tissue and covered above with pleura and below with peritoneum. All of these spaces are possible sources of diaphragmatic hernias and pleuro-peritoneal shunts. According to Meyer the foramina for the aorta, vena cava, esophagus, splanchnic nerves and azygos veins are not sites for herniae.

We believe that one of the first three congenital defects and displacement of the liver to be responsible for the occurrence of the right pneumothorax in our case.

**SUMMARY**

A case of right pneumothorax which occurred immediately after the induction of pneumoperitoneum is explained by a congenital diaphragmatic defect.

**RESUMEN**

Se explica la aparición de un neumotorax intrapleural que ocurrió inmediatamente después de la inducción de neumoperitoneo, por la existencia de un defecto diafragmático congénito.
RESUME

Un cas de pneumothorax droit qui survint immédiatement après la création d'un pneumopéritoine semble pouvoir être expliqué par une malformation congénitale du diaphragme.

REFERENCES