A Case of Bight-sided Spontaneous Pneumothorax as a Complication of pneumoperitoneum

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The occurrence of pneumothorax as a complication during pneumoperitoneum treatment is uncommon. In this context it is worthwhile to mention that Trimble et al (1948) have enumerated the various complications that took place when pneumoperitoneum treatment was introduced, Gerald, et al in 407 consecutive cases of P.P. treatment, of which 382 patterns received on an average, 800—1,000 cc, of air at weekly intervals for four to five years, no case of spontaneous pneumothorax was recorded. The condition may occur with or without loss of air from the peritoneum.

The case reported here that was observed by the author during his stay in U.K. in 1955 is one that occurred on the right side with great diminution of air in the peritoneal cavity.

A fourteen year old girl, Miss B. C., was admitted to High Wood Hospital, Brentwood, England, on 14th April, 1954, pulmonary tuberculosis having been diagnosed at a Mass Radiography Unit. On admission her x-ray (Fig. 1) showed extensive infiltration in both upper and mid zones, with numerous large cavities on the left side.

![Fig. 1. Culture of laryngeal swabs on 20-6-1954 showed tuberculosis. She received chemotherapy as follows:]

Streptomycin 1 G. daily (22-4-1954 to 5-1-1955).
INAH 100 mgm.b.d. (22-4-1954------still being continued).
PAS 10 G. (28-2-1955------still being continued).

She made a remarkable improvement. Her x-ray on 1-3-9-1954 showed considerable clearing of the mottling in both lung fields. Tomography on 25-10-1954 showed no evidence of cavitation. On 8-11-1954 artificial pneumothorax was tried on the left side, but failed. To stabilise the improved condition, pneumoperitoneum was induced on 29-11-1954. The site chosen was 2" lateral to the umbilicus on the left side. Refills were given at the same site.

She received weekly refills of 1,000 cc of air quite comfortably and final pressures always remained round about +10+12 cm. of water. X-rays showed good rise of both domes of the diaphragm (Fig. 2).

On 22-5-1955, the night before a refill, the patient complained of backache, pain in the chest and slight discomfort on deep inspiration. She had no dyspnoea or temperature.

On 23-5-1955 (Fig. 3), routine screening examination before the refill revealed a pneumothorax on the right side with associated loss of air in the peritoneal cavity.

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The patient had not had any undue strain or exertion. On 23-5-1955, the initial pressure was -6 +4, and 800 cc. of air was removed from the right pleural cavity with final pressure recorded at -10 -12.

The pneumoperitoneum was discontinued and air in the pleural cavity got completely absorbed (Fig. 5).

DISCUSSION

Two interesting questions arise:

1. What was the mechanism of the spontaneous pneumothorax?
2. How to treat the case?

Since there had been a diminution of the volume of air in the peritoneum following the pneumothorax, the logical conclusion is that air had escaped from the peritoneal cavity into the right pleural cavity.

It had passed either through the normal apertures of the diaphragm to the mediastinum and produced a pneumothorax or through a congenital defect in the diaphragm. Vajda (1933) referred to the possibility of air escaping from the pleural cavity into the abdomen through a congenital defect and producing a pneumoperitoneum. Banjai (1933) also gave instances of two cases in which artificial pneumothorax produced an indirect pneumoperitoneum, which he said was due to air passing along mediastinal structures. Banjai and Jurgens and later Simmonds assert that if the former be the possibility i.e. through normal apertures, the A.P., occurs always on the left side. But one may argue in this case that, since the left pleural cavity was not free, as shown by the failure to induce an A.P., the air might have tracked through to the pleura on the right side. Lack of evidence of any mediastinal emphysema does not support this theory of passing through openings of aorta, vena cava of oesophagus.
The other possible channel for escape of air from P. P. is a defect in the diaphragm. This may be (1) congenital, (2) inflammatory, (3) traumatic, or (4) degenerative in origin. Inflammatory perforation of diaphragm can be ruled out as it is general secondary to liver abscess or subdiaphragmatic abscess on the right side. Traumatic can also be eliminated as there is no history of trauma. As for the degenerative defect, it is common only when there is an atrophy of a portion of the diaphragm, subsequent to phrenic nerve paralysis. But in this particular case there was no phrenic crush done. So we are left with only possible cause, namely, the congenital defect.

There have often been seen at thoracoscopy, in cases where a pneumothorax and pneumoperitoneum were present, (Laird, 1945), small subpleural air blebs or pneumatoceles over the right dome of the diaphragm due to herniation of the peritoneum through small defects caused by positive abdominal pressure. These defects in the diaphragm are freely exposed to abdominal pressure as the support of the liver is lost when it is pushed down by a P.P. In this connection it is very interesting to record the case described by Sita Lumsden (1949). He argues that the possibility of escape of air through normal apertures is impossible without the presence of a defect in the parietal peritoneum; such a peritoneal defect would have no embryological basis. In order to reach the mediastinum, air would have to be injected extra-peritoneally. In the same case of his, A.P. was induced on the left side for study purposes and Vernon Thompson examined the pleura on both sides. The finding was that two subpleural air bubbles were seen over the right dome of the diaphragm (the side of spontaneous pneumothorax) and no abnormality of the left dome of the diaphragm (the side A.P. was induced).

Meyer (1950), points out three weak points in the diaphragm which are the commonest sites of diaphragmatic hernias. These are:

1. Foramen of Bochdalek—which is a pleuropertoneal hiatus situated dorso-laterally.

2. Outer Crus—at the costal areas.

3. Foramen of Morgagni—on both the sides of xiphoid process due to developmental failures of fusion.

Meyer does not believe in escape of air through normal foramina of aorta, vena cava and oesophagus.

Concerning the treatment in this case, 800 cc. of air was removed from the right pleural cavity on 23-5-1955 and 1,000 cc. of air on 30-5-1955. The pneumoperitoneum was discontinued as there was considerable resolution of the lesion with no cavitation in the right lung. One would be justified to maintain the pneumothorax on the right side if there was significant disease in the right lung. Some have re-instituted pneumoperitoneum and have had successful maintenance of the same. But recurrences are more common and an unnecessary risk, as is well shown by the results of the questionnaire and case studies at California (Reference Table 1 P. 73, Am. Rev. Tub. 63). Out of 15 cases followed up, there were recurrences twice in two cases, once in two other cases, pneumothorax maintained in five cases, pneumoperitoneum abandoned in 10 cases.
Summary

A case of spontaneous pneumothorax occurring as a complication of pneumoperitoneum is described. The possible mechanism is discussed with reference to similar cases described in the literature. It is concluded that the most likely mechanism is presence of a congenital pleuro-peritoneal defect.

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