Intraabdominal Complications and Sequellae of Pneumoperitoneum

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In the past few years pneumoperitoneum has been used extensively as a therapeutic measure in patients with pulmonary tuberculosis. In our experience with this procedure at the Municipal Tuberculosis Sanitarium of Chicago, a definite clinical and pathological pattern of local reaction to intraperitoneal air has been recognized, and it is our purpose to describe this phenomenon here.

Several reports in the literature concern animal experiments with pneumoperitoneum and none of these describes any significant local pathologic changes attributed to the presence of air in the peritoneal space.1 Trimble, Eaton and Moore,2 in 1939, reported necropsy findings in 20 patients who had received pneumoperitoneum for varying lengths of time prior to death. Less than half showed any intraperitoneal change, and in those that did, tuberculous enterocolitis or its sequellae were thought responsible. This, of course, was in the pre-streptomycin era. In a later report Trimble et al.3 described the intraabdominal complications encountered in their series of patients. Mitchell, et al.4 report similar low incidence of abdominal complications.

Several authors5-8 have discussed an apparent increase in incidence of appendicitis in patients with pneumoperitoneum, and have remarked on the poor defense reaction exhibited by these patients. Monte and Bradford,9 Rogers and Garrett10 and others have postulated an irritant effect of intraperitoneal air as a factor in the closure of the processus vaginalis in inguinal hernias during pneumoperitoneum therapy. Banyal11 noted no tendency toward formation of intraabdominal adhesion as noted on re-sumption of pneumoperitoneum after temporary discontinuation. Corrigan12 on the other hand reported that two out of five such cases had extensive adhesions preventing adequate re-institution of the pneumoperitoneum.

There are many reports in the literature describing accidents incurred as a direct result of the air instillation. These include air embolism, spontaneous pneumothorax, inflation of hernial sacs, perforation of hollow or

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solid abdominal viscera, etc.\textsuperscript{11-13,16-22} Our experience with such accidents has been similar, and we shall not consider these in this report.

At the Municipal Tuberculosis Sanitarium, pneumoperitoneum has been used extensively for several years. In the years 1948 through 1952 more than 1,500 patients received pneumoperitoneum for varying lengths of time as part of their therapeutic regimen. Most of them were treated with one or more of the common anti-tuberculous drugs, dihydrostreptomycin, PAS and INH prior to or during the period in which the pneumoperitoneum was initiated. Some had temporary phrenic nerve interruption as an adjunct to the pneumoperitoneum. The specific indications and the long range therapeutic results of pneumoperitoneum will not be discussed here, but in general the patients were those with far advanced bilateral pulmonary tuberculosis. Pneumoperitoneum is ordinarily not used in persons who have any significant gastrointestinal complaints even in the absence of any demonstrable pathology. This policy was formulated when our early experience showed us that these complaints were frequently exaggerated after the induction of pneumoperitoneum, resulting in the patient's refusal to continue this treatment. Since the advent of the various antibiotics, the incidence of gastrointestinal tuberculosis has been greatly reduced. At routine necropsy in this institution, evidence of tuberculous enterocolitis has been demonstrated in only 20 per cent of patients in the past two years, as compared to 90 per cent prior to the streptomycin era.

In many of the 1,500 patients mentioned above, the pneumoperitoneum has been discontinued. Most of these were abandoned when some form of surgical collapse or excision could be substituted. Some were given up when it became obvious that the diaphragmatic elevation obtained, if any, was therapeutically ineffective. A majority showed at one time or another evidence of small amounts of intraperitoneal fluid as seen on routine fluoroscopy. Few had fluid which persisted long enough and in sufficient amount to warrant discontinuation of the air refills. Some 15 patients (1 per cent) developed intraabdominal symptoms severe enough to force abandoning pneumoperitoneum. Some of these will be described below. In only two during this four year period was there a fatality which could be directly or indirectly attributed to the use of pneumoperitoneum. These two cases will be described briefly.

Case I: L.S., a 27 year old Japanese woman was admitted to the Sanitarium on February 11, 1949 with a diagnosis of pulmonary tuberculosis, far advanced with extensive bilateral involvement and positive sputum. Pneumoperitoneum was initiated on March 11, 1949 and maintained until June 10, 1949. The last few refills had been followed by right upper quadrant pain, nausea, vomiting and moderate febrile reaction so that finally the pneumoperitoneum was discontinued. These symptoms, though intermittent and mild in nature, persisted despite a 60-day course of daily dihydrostreptomycin. In January 1950, there was an acute exacerbation of the abdominal symptoms with high fever and loss of weight. A right subphrenic abscess was diagnosed and surgically drained on January 23, 1950. The pus contained acid fast bacilli and streptococci and other coccal organisms. Her condition improved for a few weeks, but then she became progressively worse and died on April 6, 1950. Necropsy showed that the liver was adherent to the
diaphragm by fibrinous to fibrous adhesions. The capsule of the spleen was covered by a fibrinoid coat. There was a large caseous nodule in one adrenal and the pelvic organs were bound together by dense fibrous adhesions.

Upon opening the uterus and the fallopian tubes it was found that the right tube was the seat of caseation necrosis. The gastrointestinal tract showed no ulceration but several nodular areas in the mucosa were noted.

Microscopic studies of the tissues disclosed that the fibrinous exudate between the liver and the diaphragm contained many typical epithelioid tubercles with caseation necrosis. The caseous nodule in the adrenal likewise was tuberculous in nature as was the right fallopian tube. Apparently this diffuse process throughout the peritoneal cavity had its origin from the caseous tuberculous salpingitis.

**Case 2:** L.F., a 45 year old white man, was admitted to the Municipal Tuberculosis Sanitarium on January 3, 1950 with a diagnosis of pulmonary tuberculosis, far advanced with positive sputum. Pneumoperitoneum was initiated on April 26, 1950 after two months of streptomycin and PAS. He was discharged, culture negative on July 7, 1951 with the classification of far advanced apparently arrested. Pneumoperitoneum was continued at one of our out-patient clinics. On December 19, 1951, one day after a pneumoperitoneum refill, he complained of abdominal pain and malaise. His condition did not seem alarming and he was observed at home until January 5, 1952 when he was readmitted to the Sanitarium as an emergency. His abdomen was markedly distended, and large amounts of air were aspirated on January 5, 1952 and January 7, 1952. A small amount of clear amber fluid was also aspirated from the abdomen at this time. He had a good appetite, and his bowels moved normally until January 20, 1952 when his condition deteriorated rapidly. The abdomen again became distended and the temperature climbed rapidly. On January 21, 1952 some 2,000 cc. of foul “pea soup” pus was aspirated from the abdomen. This fluid showed no organisms on smear, and subsequent cultures demonstrated no organisms whatsoever. The patient failed to improve despite intensive antibiotic therapy and died on January 22, 1952.

At necropsy the abdomen was markedly distended and when it was opened approximately 6,000 cc. of foul smelling purulent material was found in the peritoneal cavity. The loops of intestine were matted together, and the liver and spleen were adherent to the surrounding structures. The liver was deformed with blunting of the free margin and there was considerable thickening of the capsule over the dome which was rather typical of that change noted in other cases of pneumoperitoneum; in addition, there was purulent exudate superimposed on this fibrinoid thickening. The gastrointestinal tract was examined meticulously and no ulcerations in the mucosa could be noted at any point. The appendix was opened from the cecal ostia and the distal end of the appendix ended in and was continuous with a retro-peritoneal abscess cavity which had ruptured into the peritoneal cavity and produced this generalized peritonitis. Cultures of the abdominal fluid and sections of the various tissues failed to reveal the presence of acid-fast bacilli or tuberculous lesions.

In retrospect one can speculate that the original perforation of the appendix took place on the 19th of December and the infection remained walled-off until about one month later when it was discovered that he had generalized peritonitis. The presence of pneumoperitoneum obviously had masked the original findings and had served to prevent the normal defensive mechanisms within the abdomen from localizing the infection.

Of the 15 patients in whom severe abdominal complaints necessitated abandoning pneumoperitoneum six developed a well-defined clinical syndrome of small bowel obstruction. In each the picture was that of low-grade peritonitis with paralytic ileus rather than mechanical obstruction.
FIGURE 1

*Figure 1:* Photomicrograph of parietal peritoneum from a patient who had received pneumoperitoneum for 15 months. Note the piling-up of mesothelium and the vascular stroma. x50.—*Figure 2:* A thin projection of serosa with an intact mesothelial layer. x50.
All had at least a small amount of intra-peritoneal fluid, but only one had paracentesis, and the fluid obtained in this instance was sterile. All responded to conservative therapy with continuous gastric suction, antibiotics and parenteral feeding. The pneumoperitoneum in each was, of course, discontinued. Following is a representative case:

Case 3: C.A., a 40 year old white male was admitted to the Municipal Tuberculosis Sanitarium on July 1, 1949 with a diagnosis of pulmonary tuberculosis, moderately advanced with positive sputum. Pneumoperitoneum was started on July 26, 1949 because of bilateral pulmonary involvement. On December 1, 1949, three days after a refill, he began to notice slight abdominal distress. This progressed slowly until on December 8, 1949 he was markedly distended and had persistent nausea and vomiting. The temperature ran from 100 to 102 degrees F. The abdomen was tympanitic, and no bowel sounds could be heard. X-ray film showed the typical picture of a small bowel obstruction.

A Miller-Abbot tube was introduced and was seen fluoroscopically to enter the duodenum. Dihydrostreptomycin and penicillin were started and parenteral alimentation. Bowel sounds soon returned and the distension was relieved, but interruption of the suction resulted in prompt relapse on three occasions. It was not until January 6, 1950 that he was finally able to tolerate oral feedings—a total of 29 days from the initiation of decompression. He remained symptom-free thereafter and was discharged from the Sanitarium on November 21, 1950 as apparently arrested. (Old National Tuberculosis Association classification.)

Pathology:

Since early 1949 some 50 patients who received pneumoperitoneum have been studied at necropsy. In 45 (90 per cent), inflammatory changes were noted in the peritoneal surfaces—especially those covering the liver and spleen. The extent of this alteration was directly proportional to the length of time the patients received pneumoperitoneum, and, as near as we have been able to determine, no regression of this pathologic change was apparent even though pneumoperitoneum may have been abandoned some time previously.

The picture seen following a period of therapy resembles closely the condition referred to in the spleen as "sugar-coating" or "zuckerguss" which connotes a translucent pearly-gray or pale blue sheet covering the serous surface of the organ. Many times, however, the process progressed at such a rapid rate that there was a piling-up of the fibrinous and fibrous material and almost complete adhesion to surrounding structures. Oddly enough the coating in some instances seemed to be rather loosely attached to the organ and could be quite easily separated. This is comparable to the layer one finds overlying the visceral pleura following long standing pneumothorax and which, because of its loose attachment, permits successful decortication in some cases. A fibrous perihepatitis of long standing may be of such a degree as to actually deform and shift the liver from its normal shape and position. In more than one instance complete atrophy of the left lobe of the liver was noted and the free margin of the organ was displaced upward (cephalad). Just what effect this immobilization and displacement of abnormal viscera may have on function is difficult to say.
Figure 4: Photomicrograph of liver showing a marked fibrous periphery with extension down into the liver parenchyma. x50. Figure 5: Photomicrograph of spleen showing a thickened tortuous capsule with a fibrous membrane superimposed. Note the collections of lymphocytes in the inflammatory tissue. x50.
The peritoneum is a serous membrane which consists of two layers: a connective tissue stroma and a mesothelium. The stroma is composed of loosely arranged connective tissue bundles which interlace in a plane parallel to the surface. There are numerous elastic fibers, especially in the deeper layers, where one also finds a rich capillary network and lymphatics. The mesothelium consists of a layer of flat polygonal cells with bulging nuclei. This delicate structure quickly reacts to any irritant or stimulus and one needs but to read Hertzler's classic accounts of his studies of the peritoneum to appreciate this fact. While the peritoneum has been subjected to many chemical and physical irritants, little is known about its reaction to the introduction of air. And perhaps even more important is the presence of this air under pressure. It is a well known fact that increased pressure within the peritoneal cavity reduces the rate of absorption (because of the interference with the circulation of blood), and indirectly this may have a bearing on the picture that is seen following prolonged use of pneumoperitoneum. Elevation of the diaphragm by prolonged increased intra-abdominal pressure certainly causes displacement of viscera lying close to it, especially the liver with its ligamentous attachments. In addition there may be factors related to the posture of man that have a bearing on the picture seen following pneumoperitoneum. In our laboratory air was introduced into the peritoneal cavity of guinea pigs at five to seven day intervals for a long period of time without any noticeable effect on the serous surfaces.

Microscopic studies of parietal peritoneum removed during the repair of inguinal hernias in pneumoperitoneum patients revealed, for the most part, a piling-up of the mesothelium and some increase in the vascularity of the subserous tissues. At necropsy, changes in the parietal (Figure 1)

FIGURE 5: Photomicrograph of spleen showing a thickened capsule, portions of mesothelium remaining and dense hyalinized fibrous tissue covering. x50.
were seen much less frequently and to a lesser degree than those in the visceral peritoneum. One is led to believe that the parietal peritoneum with its richer subserous layer can cope with this prolonged subjection to air pressure much better than the extremely thin serous layer covering the viscera, especially the liver and the spleen. The earliest lesion we noted was that depicted in Figure 2 where there was simply a thin outward projection of the peritoneum without any significant alteration of the mesothelium but an increase in the thickness of the subepithelial layer. One cannot say with certainty, however, that this was not an abnormal variation of a normal serous membrane as pointed out by Hertzler.

In more advanced cases the membrane seen on the peritoneal surface of the organ appeared to be dense fibrous tissue which in some instances was hyalinized. There were many large vascular spaces seen in the region of the organ capsule and the fibrous process extended down into the parenchyma of the organ (Figure 3). There is no doubt, that in some cases the capsule of the organ thickens considerably (Figure 4) before there is actually any deposition of fibrinous or fibrous material upon it. Figure 5 shows portions of the mesothelial layer still intact in spite of the layer of fibrous tissue over it.

The inflammatory process may become more marked until all semblance of normal peritoneum is eradicated and the fibrous tissue extends in a tentacular fashion over the organ (Figure 6); eventually the entire surface was involved and became adherent to the surrounding structures.

SUMMARY

Pathologic changes occurred in some 90 per cent of our cases. Speculation is made as to the pathogenesis of this process and the possible disturbance
of abdominal visceral function. Several patients developed low grade peritonitis with a dynamic small bowel obstruction. All responded to nonsurgical management. Pneumoperitoneum has been definitely incriminated as contributing to death in two patients. The overall incidence of significant complication has been about 1 per cent.

RESUMEN

Ocurrieron cambios patológicos en aproximadamente 90 por ciento de nuestros casos. Se especula acerca de la patogenia de este proceso y el trastorno posible de la función de las vísceras abdominales. Varios enfermos presentaron peritonitis poco grave con obstrucción dinámica del intestino delgado. Todos respondieron al tratamiento no quirúrgico. El neumoperitoneo ha sido incriminado definitivamente como factor contribuyente a la muerte de dos enfermos. La incidencia total de complicaciones importantes es aproximadamente de uno por ciento.

RESUME

Les auteurs ont constaté des altérations anatomiques dans environ 90% de leurs cas. Ils envisagent la pathogénie de ce processus et la possibilité d'un dérèglement de la fonction des viscères abdominaux. Plusieurs malades furent atteints d'un lent degré de péritonite avec occlusion paralytique de l'intestin grêle. On obtint pour tous de bons résultats sans faire appel au traitement chirurgical. Chez deux malades, on a pu admettre avec certitude que le pneumopéritone avait contribué à la mort. La moyenne des complications dans l'ensemble fut de 1%.

REFERENCES

23 Hertzler, A. E.: "Surgical Pathology of the Peritoneum," 1935, Chapters I, II and IV.