<table>
<thead>
<tr>
<th>Title</th>
<th>A Case of Spontaneous Nonsurgical Pneumoperitoneum Associated with Adenocarcinoma in the Esophagogastirc Junction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Author(s)</td>
<td>KASAHARA, YOH; TANAKA, SHIGERU; YAMADA, YUKIKAZU; SONOBE, NARUMI; MATSUMOTO, HIROKI; SUDO, TAKAAKI; UMEMURA, HIROYA; SHIRAHA, SEI; KUYAMA, TAKESHI; KAWAI, SHUJI</td>
</tr>
<tr>
<td>Citation</td>
<td>日本外科宝函 (1982), 51(5): 805-813</td>
</tr>
<tr>
<td>Issue Date</td>
<td>1982-09-01</td>
</tr>
<tr>
<td>URL</td>
<td><a href="http://hdl.handle.net/2433/208966">http://hdl.handle.net/2433/208966</a></td>
</tr>
<tr>
<td>Type</td>
<td>Departmental Bulletin Paper</td>
</tr>
<tr>
<td>Textversion</td>
<td>publisher</td>
</tr>
</tbody>
</table>
A Case of Spontaneous Nonsurgical Pneumoperitoneum Associated with Adenocarcinoma in the Esophagogastric Junction

YoH KASAHARA, SHIGERU TANAKA, YUKIKAZU YAMADA, NARUMI SONOBE, HIROKI MATSUMOTO, Takaaki Sudo, HIROYA UMEMURA, SEI SHIRAHA and TAKESHI KUYAMA

The Second Department of Surgery, Kinki University School of Medicine
(Director: Prof. Dr. TAKESHI KUYAMA)

SHUJI KAWAI
Surgical Service, Wakakusa Daiichi Hospital
(President: Dr. KOHKI KAWAI)

Received for Publication, July 9, 1982.

The presence of pneumoperitoneum on plain films of the chest or abdomen usually indicate prompt laparotomy because pneumoperitoneum except for postoperative condition of the abdominal surgery almost always means perforation of the hollow viscus or intra-abdominal proliferation of gas-producing microbes. Whereas several cases called “nonsurgical” or “medical” pneumoperitoneum have been reported in the literature. Urgent laparotomy is unnecessary for these patients.

Report of A Case

A 47-year-old housewife complaining of loss of appetite and difficulty in swallowing was admitted on March 25, 1982. She had lost 7 kg of body weight for these two months. The patient had resection of the uterus at her age of 27. Personal and family histories were unremarkable. Several studies including barium swallow and esophagogastric endoscopy with biopsy revealed adenocarcinoma in the esophagogastric junction (Fig. 1). On admission, she complained of the sense of abdominal distention and progressive difficulty of postprandial eructation. Physical and roentgenologic examinations of the chest and abdomen revealed no particular change. Laboratory studies showed no serious abnormality.

On April 1, 1982, she underwent preoperative management of intravenous hyperalimentation...
through the left subclavian vein. The plain chest film confirming the location of tip of the catheter incidentally showed massive pneumoperitoneum (Fig. 2). No pneumothorax was found. Although pneumoperitoneum was present, she neither complained of abdominal pain nor had peritonitis sign. Computed tomography of the abdomen also revealed pneumoperitoneum without ascites (Fig. 3). Following the repeated x-ray and physical examinations of the abdomen and laboratory studies, the diagnosis of nonsurgical pneumoperitoneum was established. The patient had been in relatively well condition until the surgery.

On April 7, 1982, the patient was explored through left thoraco-abdominal approach to resect the carcinoma. On entering the pleural space and the mediastinum, there were no particular changes in both regions. Only a moderate amount of gas escaped from the peritoneal cavity when the peritoneum was opened. There was no possible sign of peritonitis or perforation of the viscus in the peritoneal cavity. Investigations of the intestines demonstrated no abnormality. Resection of the lower esophagus, the stomach, the tail of pancreas and the spleen with reconstruction of esophagojejunostomy was performed successfully. Following uneventful postoperative course, she was discharged on May 19, 1982. Postoperative repeated x-ray
Fig. 2. Plain chest film showing massive pneumoperitoneum.

examinations of the chest and abdomen had never demonstrated recurrence of pneumoperitoneum except only the postoperative usual phenomenon of short duration.

Discussion

Generally pneumoperitoneum signals perforation of the gastrointestinal tract and requires prompt surgical treatment. However, pneumoperitoneum on rare occasion may occur without any leak of the gastrointestinal tract. Although some of them resulted from proliferation of the gas-producing microbes and urged to laparotomy\(^8,17,30,31,36\), several cases called as "nonsurgical" or "medical" pneumoperitoneum have been reported in the literature. Miller and others\(^27\) collected briefly and clearly the causes of nonsurgical pneumoperitoneum as listed in Table 1.

The causes of free air in the peritoneal cavity were classified into; 1) intrathoracic, 2) abdominal, 3) gynecologic and 4) iatrogenic, by Gantt and others\(^13\). Intrathoracic causes of pneumoperitoneum are miscellaneous. If the diaphragm or the hiatus in the perivascular spaces had direct communication between the pleural space and the peritoneal cavity, the occurrence of
pneumoperitoneum at the presence of pneumothorax is easily understood. GLAUSER\textsuperscript{14} reported a diverse case that air sufflated into the peritoneal cavity escaped into the pleural space through a defect of the diaphragm resulting pneumothorax. A fistula between the bronchus and the peritoneal cavity was also described\textsuperscript{24}. When such an apparent continuity is absent, pneumomediastinum may indicate the cause of pneumoperitoneum that results from air-escape via transdiaphragmatic routes\textsuperscript{23,33}.

DONAHOE and others\textsuperscript{9} showed that air from ruptured pulmonary alveoli in rats dissected along vessel sheaths, and may rupture into the pleural space, the retroperitoneum, the peritoneum, and the subcutaneous tissues. This occurs when air under pressure is forced into the retroperitoneum along the esophagus and great vessels. MACKLIN and MACKLIN\textsuperscript{23} have also demonstrated clinically and experimentally that air could escape from a ruptured alveolus into the interstitial tissue of the lung and enter the mediastinum via the sheaths of the pulmonary blood vessels. Others postulate that interstitial air from alveolar ruptures entering the pulmonary lymphatics, being forced retrograde by intermittent positive pressure created by the ventilator and eventually escaping into the peritoneal cavity through the peritoneal lymphatics\textsuperscript{4,20}. Experimental studies have demonstrated that sudden blasts of air introduced into the trachea under increased pressure

**Table. 1.** Etiologies of “nonsurgical pneumoperitoneum”

| 1. | Postoperative, diagnostic, and experimental |
| 2. | Introduction of air through fallopian tubes |
| 3. | Increased intra-alveolar-extra-alveolar pressure difference |
| 4. | Pneumothorax-associated |
| 5. | Miscellaneous |
in dogs produced a pneumothorax, followed by pneumoperitoneum^{36}.

Miller and others^{37} described that increased intra-alveolar-extra-alveolar pressure difference produced the rupture of pulmonary alveoli. A case reported by Rose and Jaczk^{22} in scuba diving may be due to rapid decline in extra-thoracic pressure (e.g. rapid ascent to surface in that diving), and other cases^{26,36} may be due to rapid increase in intra-alveolar pressure. In the latter cases, straining against a closed glottis is also included.

Gynecologic causes of pneumoperitoneum reported in the literature were due to cunnilingus^{11}, due to knee-chest exercises in the postpartum period^{21}, due to orogenital insuffiation^{11}, due to vaginal douching^{26}, due to Rubin test for patency of Fallopian tubes^{26}, and due to pelvic examination^{8}. The air leakage into the peritoneal cavity through the Fallopian tube may be the decisive cause of pneumoperitoneum in these cases.

Abdominal causes of nonsurgical pneumoperitoneum reported in the literature were due to pneumatosis cystoides intestinalis^{7,38}, due to chronic jejunal diverticulosis^{30,12,15,34,39}, and other factors. According to Wolloch and others^{38}, a mechanical factor alone does not seem sufficient to produce intestinal cysts and an additional factor, a mucosal permeability change, is apparently needed to cause appearance of the disease. The cysts found in infants and children are generally submucosal, whereas those in older children and in adults are subserosal. Cysts of the intestinal wall are commonly believed to be dilated lymph vessels^{39}.

Several theories have been proposed: infectious^{18}, biochemical-nutritional^{39}, and mechanical. Among these theories, mechanical theory seems to be most appropriate. It is postulated that a break in the continuity of the mucosa permits air to pass from the intestinal lumen to the lymph vessels. Intestinal obstruction increases the pressure of the gas and large amounts may penetrate the wall causing cyst formation. Enteritis, ulcerations, tumors, scleroderma, and procedures such as sigmoidscopy, polypectomy or barium enema, which cause a break in the continuity of the mucosa, are conductive to the development of pneumatosis intestinalis. The relatively low frequency of pneumatosis intestinalis, despite a high rate of disorders producing mucosal disruption, may be due to rupture of the cysts and a rapid absorption of the air^{38}.

A combination of the mechanical and permeability theories would seem to be necessary to explain the development of pneumatosis intestinalis and resultant occasional appearance of pneumoperitoneum. These theories also seem to be appropriate to explain the onset of pneumoperitoneum in the clinical course of jejunal diverticulosis. The distended diverticular mucosa may function as a semipermeable membrane allowing transmural gas equilibration. In the presence of false diverticula, the mucosa may be acting as a semipermeable membrane which allows equilibration of gastrointestinal and blood gases across the intestine in the absence of any true anatomic "leak". Since gas is absorbed from the peritoneal cavity, the presence of persistent pneumoperitoneum implies a continuous or recurrent source of air. Wright and Lumsden^{39} reported a recurrent case of pneumoperitoneum in chronic jejunal diverticulosis.

Among other theories concerning the development of pneumatosis cystoides intestinalis, Smith and Walter^{35} suggest that air is forced into the tissue spaces around an obstructing pyloric lesion during vomiting. The intestinal cysts associated with obstructive lung disease are,
on the other hand, thought to result from rupture of pulmonary blebs with infiltration of air into the mediastinum and retroperitoneal space. This route resembles one of the intrathoracic causes of pneumoperitoneum. The results of gas analysis of the intestinal cysts showed the following; 70% to 90% nitrogen, 3% to 20% oxygen, 0% to 15% carbon dioxide, and traces of methane.

Felson and Wiot reviewed the causes of pneumoperitoneum and peptic ulcer disease was the most common. They presumed that one of the causes of pneumoperitoneum may be secondary to a tiny perforation. Koss postulates that an intermittent air leak through a peptic or malignant ulcer may cause pneumatosis intestinalis. However, this has not been verified.

Iatrogenic pneumoperitoneum is usually asymptomatic and often follows laparoscopy or laparotomy requiring larger incisions. Almost all of the causes of iatrogenic pneumoperitoneum are clearly understood because of the history of preceding diagnostic or therapeutic procedure. Although the causes had been frequently included in intrathoracic, gynecologic, etc., the precipitating factors of this pneumoperitoneum during medical management were various. Reported causes in the literature were simple dental extraction, inadvertent needle puncture of diaphragm for the treatment of pneumothorax and resultant communication between the peritoneal cavity and the pleural space, manual Ambu bag ventilation, mechanical ventilator therapy, closed-chest cardiac massage, deliberate percutaneous injection of air bolus, gastroscopy, percutaneous cholangiography, percutaneous biopsy of liver or kidney, routine pelvic examination, culdoscopy, Rubin test, and rectal perforation due to injudicious use of rectal thermometers in neonates.

In our case, neither the technique of esophagogastric fiberscopy nor insertion of catheter for intravenous hyperalimentation would seem to be responsible for development of pneumoperitoneum. The patient has had no obstructive lung disease, denied the gynecologic causes of pneumoperitoneum, and no intestinal cysts or diverticulosis were found at the surgery. The macroscopic specimen of the carcinoma demonstrated no perforation despite the serosal surface of the stomach was already invaded.

Papp and Sullivan reported a case of spontaneous pneumoperitoneum due to aerophagia. Although the presence of air cysts in the stomach of pneumatosis intestinalis was recorded, postoperative histologic examination demonstrated no gas cysts in the gastric wall in our case. However, the location and involvement of carcinoma in the esophagogastric junction of the patient in our case developed the progressive disturbance of eructation simulating so-called gas-bloating syndrome, and this condition of the gas-filled stomach of intraluminal high pressure may suggest the resemblance to the condition of aerophagia. Although the true process of producing pneumoperitoneum in our case is still unclear, resultant pneumoperitoneum may be evoked by the expanded stomach.

To establish the diagnosis of nonsurgical pneumoperitoneum is important in order to prevent needless laparotomy and to improve the condition of debilitated patients. Zerella and McCullough suggests that the absence of abdominal distention prior to the occurrence of pneumoperitoneum often means nonsurgical pneumoperitoneum, and they advocates following
diagnostic plans; multiple x-ray views of the abdomen for determining the necessity for laparotomy, paracentesis, and instilling water-soluble contrast material into the gastrointestinal tract through a nasogastric tube. Repeated careful examinations are necessary to differentiate nonsurgical pneumoperitoneum from usual surgical pneumoperitoneum which should be operated as soon as possible. Whether to remove the intra-abdominal air of nonsurgical pneumoperitoneum or to remain it in situ depends upon the causative factors, but generally emergency laparotomy is needless.

Summary

A case of nonsurgical pneumoperitoneum developed during the preoperative management to carcinoma in the esophagogastric junction was described. Several precipitating factors concerning the development of nonsurgical pneumoperitoneum were discussed with review of the literature. Among these cases, so-called pure spontaneous pneumoperitoneum was relatively rare because intrathoracic, abdominal, gynecologic, iatrogenic, or mixed causes were generally noted preceding to the occurrence of this phenomenon. The gas-filled stomach occurring in so-called gas-bloating syndrome was thought to be responsible for the appearance of pneumoperitoneum in this patient. But true process of air leak was not detected.

References


和文抄録

特発性の非手術適応の気腹像，食道胃境界部癌に合併の1例

近畿大学医学部第二外科学教室（主任：久山 健教授）
笠原 洋，田中 茂，山田 幸和，園部 鳴海，松本 博城
須藤 竦章，梅村 博也，白羽 誠，久山 健

若草第一病院外科（院長：川合弘穂博士）
川 合 秀 治

通常気腹像が開腹術施行後以外にみられる場合は，消化管穿孔またはガス産生菌によることが多く，緊急手術の適応となる，しかし非外科的，非手術適応，内科的気腹などと呼ばれる気腹像の報告も少数ながらみられる。この非手術適応の気腹はその発生原因により胸部由来，腹部由来，婦人科的由来，医原性，およびこれらの混合型がみられるが，まったく誘因ないし原因不明の例は少ないとあるのである。自験例においては食道胃境界部癌の進展により腹下気室の排出困難，胃膨満，我々がもってきた，偶然術前の胸腹部レ線撮影において発見された高強度の気腹像の成因として，腸管囊状気室と同様な胃壁の変化，あるいは極小の癌部の穿孔などが疑われたが，組織的検索ではいずれも否定的であり，胃膨満と気腹発生の関連性を示唆するに止めた。