Pneumoperitoneum associated with artificial ventilation

BRUCE SUMMERS

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Summary and conclusions

Four adults injured in serious road-traffic accidents developed pneumoperitoneum after artificial ventilation. No case could be attributed to injury or to perforation of a hollow viscus in the belly, but doubt about this in one patient resulted in exploratory laparotomy. In three patients the origin of intraperitoneal air was considered to be leakage through minute ruptures in alveoli subjected to the stress of artificial ventilation, with air tracking to the mediastinum, pleural space, subcutaneous tissues of the neck and chest, and peritoneal cavity. The fourth patient later developed herniation of the stomach through a rupture in the diaphragm, the presence of a pneumothorax on the same side suggesting direct passage of air through the diaphragm.

Pneumoperitoneum induced by artificial ventilation is probably more common than reports suggest and should be distinguished clinically and radiologically from that caused by rupture or perforation of a hollow viscus. This will reduce the number of needless laparotomies performed on patients who are already seriously ill.

Introduction

Pneumoperitoneum in patients who have not had recent laparotomy usually indicates rupture of a hollow viscus and necessitates surgical exploration. Rarer causes of pneumoperitoneum should be considered in patients who show no other clinical evidence of perforation. These "non-surgical" causes were well reviewed by

Gantt *et al.*¹ who classified them according to the source of gas. Thus abdominal causes include pneumatosis cystoides intestinalis, in which gas-filled cysts in the walls of the gastrointestinal tract burst into the peritoneum without leakage of intestinal contents.² The natural communication between the female genital tract and the peritoneal cavity has resulted in pneumoperitoneum secondary to orogenital insufflation,¹ knee-chest exercises,³ and pelvic examination in the postpartum period. Iatrogenic causes include laparotomy, endoscopic procedures, and tubal insufflation to determine patency of the Fallopian tubes.

Gantt *et al.*¹ discussed pneumoperitoneum in newborn babies ventilated for respiratory distress syndrome but failed to mention its occurrence in adults, which, though rare, presents the same diagnostic dilemma. Other American reports, however, have described its occurrence in adults ventilated for various reasons.⁴⁻⁷ I review here the cases of four adults admitted to this hospital after serious road-traffic accidents who developed pneumoperitoneum after artificial ventilation.

Case histories

The table gives the details of the four cases.

Case 1—A 29-year-old man was admitted with injuries including a left pneumothorax and an extensive fracture of the skull. The abdomen was normal. Treatment was instituted with intercostal drainage; dexamethasone and mannitol to reduce cerebral oedema; and endotracheal intubation and ventilation with oxygen at an inflating pressure of 30-35 mm H₂O. Later that day pronounced surgical emphysema of the neck and chest was noted and an erect x-ray film showed gas under the diaphragm and in the mediastinum. The pneumoperitoneum was both intraperitoneal and extraperitoneal (fig 1). Peritoneal lavage showed no evidence of contamination with gastrointestinal contents, but owing to an increase in gas below the diaphragm laparotomy was performed; no perforation was found. The pneumoperitoneum persisted, and the patient continued with ventilation until his death seven days later. Necropsy showed no lesion in the gut or diaphragm.

Case 2—A 20-year-old motorcyclist was admitted with a fractured femur. There was no evidence of injury to head, chest, or abdomen.

Birmingham Accident Hospital, Birmingham B15 1NA

BRUCE SUMMERS, MB, BS, senior house surgeon

Details of patients injured in serious road-traffic accidents who developed pneumoperitoneum as a result of artificial ventilation

Case No	Sex and age	Condition on admission and indications for ventilation	Treatment	Clinical course	Outcome
1	M 29	Conscious; signs of cerebral bruising; left pneumothorax; fractures of left zygoma and left clavicle; abdomen soft and bowel sounds present; extensive skull fracture seen on radiography	Intercostal drainage; dexamethasone and mannitol; and endotracheal intubation and ventilation at inflating pressure of 30-35 mm H ₂ O instituted on day 1	Surgical emphysema of neck and chest noted on day 1; gas under diaphragm and in mediastinum seen on erect radiography	Died seven days after exploratory laparotomy. Necropsy showed extensive cerebral bruising and bronchopneumonia. No lesion in gut or diaphragm
2	M 20	Fractured shaft of femur; no sign of injury to head, chest, or abdomen; fat embolism diagnosed on day 2	Ventilation at 7 cm H ₂ O (PEEP) and tidal volume 1.5 l instituted on day 2	Bilateral pneumothoraces, pneumoperitoneum, and pneumomediastinum seen on radiography	Recovered. No perforation of gut seen on radiography
3	F 20	Burns to face, neck, hands; hangman's fracture of pedicles of axis; fractured left clavicle. Fever and rigors on day 5, with heavy growth <i>Escherichia coli</i> isolated from blood. Pulmonary oedema and hypoxia on day 7	Ventilation at 25 cm H ₂ O (PEEP) instituted on day 7	Surgical emphysema of upper chest, neck, and face noted on day 8; pneumoperitoneum, pneumomediastinum, and pneumopericardium seen on erect radiography; right pneumothorax seen on day 9; disseminated intravascular coagulation diagnosed	Died on day 10. Necropsy findings consistent with disseminated intravascular coagulation. No defect in gut or diaphragm
4	F 23	Unconscious; fractures of mandible, maxilla, and ribs; left haemothorax	Intercostal drainage, and intubation and ventilation with oxygen at inspiratory pressure of 38 mm H ₂ O and tidal volume 1.0 l instituted on day 1	Surgical emphysema of right chest noted on day 2; gas under diaphragm seen on erect radiography	Tear in left dome of diaphragm discovered in 2nd week. Discharged seven weeks after admission

PEEP = Peak end-expiratory pressure.

FIG 1—Case 1. Erect chest x-ray film showing pneumoperitoneum, subcutaneous emphysema, and intercostal chest drain.

FIG 2—Case 2. Erect chest x-ray film showing pneumoperitoneum and left pneumothorax.

The next day he became unconscious and cyanotic; fat embolism was diagnosed. Treatment included ventilation with a pressure of 7 cm H₂O at the end of expiration (PEEP) and a tidal volume of 1.5 l. On day 3 radiography showed bilateral pneumothoraces, pneumoperitoneum, and pneumomediastinum (fig 2). Bilateral intercostal drains were inserted and both lungs re-expanded. The pneumoperitoneum persisted until artificial ventilation was stopped five days later, when the chest drains were removed.

Case 3—A 20-year-old woman car driver was admitted with injuries including burns to face, neck, and hands, amounting to 18% of the surface area. She made good progress till the fifth day. On day 7 she developed pulmonary oedema and hypoxia necessitating ventilation with an end expiratory pressure of 25 cm H₂O. Next day gross surgical emphysema of the upper chest, neck, and face was noted. Erect x-ray films showed pneumoperitoneum, pneumomediastinum, and pneumopericardium. Reducing end expiratory pressure to below 15 cm H₂O resulted in severe hypoxia. On day 9 radiography disclosed a right pneumothorax, which was treated with intercostal drainage. Coagulation studies showed disseminated intravascular coagulation. Despite treatment with heparin and clotting factors, however, she

died the next day. Pneumoperitoneum, pneumomediastinum, pneumopericardium, and subcutaneous emphysema persisted till death. At necropsy no defect was found in the diaphragm or gut.

Case 4—A 23-year-old woman was admitted unconscious and with fractures of the mandible, maxilla, and ribs on both sides. Radiography showed a left haemothorax. An intercostal drain was inserted and the patient intubated and ventilated with oxygen at an inspiratory pressure of 38 mm H₂O and a tidal volume of 1.0 l. Next day surgical emphysema of the right chest was noted, and an erect x-ray film showed gas under the diaphragm. These signs lasted for one day. Ventilation was discontinued on the second day but the endotracheal tube was left in place. Recovery was complicated by herniation of the stomach through a tear in the left dome of the diaphragm. This was repaired and the patient discharged seven weeks after admission.

Discussion

These four patients developed pneumoperitoneum after artificial ventilation. Two (cases 1 and 3) also developed pneumothoraces, pneumomediastinum, and subcutaneous emphysema. One (case 2) developed bilateral pneumothoraces and pneumomediastinum. The coexistence of air in these areas suggests a common cause. In 1929 Joannides and Tsoulos⁸ reported that introducing air into the lungs of dogs under suddenly increased pressure caused bilateral pneumothoraces, pneumomediastinum, subcutaneous emphysema, and pneumoperitoneum. Macklin⁹ then showed in animals that overinflation of the lungs resulted in minute ruptures of the alveoli, allowing leakage of air into the interstitial tissue of the lung, where it "can travel along the sheaths of pulmonary vessels in artificial channels which it dissects for itself . . ." to the mediastinum. From there it tracks to the cervical fascial layers and produces surgical emphysema; to the pleural space, causing a pneumothorax; and to the retroperitoneal space along para-aortic and paraesophageal tissues, later rupturing into the peritoneal cavity and giving rise to pneumoperitoneum.¹⁰ In 1971 Grosfield *et al.*,¹¹ conducting similar experiments to those of Macklin, found pneumopericardium in one-third of their animals.

One patient in my series (case 4) was initially thought to have developed pneumoperitoneum in this way. The herniation of her stomach through a tear in the diaphragm, however, made it more likely that air took a direct route through the diaphragm.

Case 1 presented a difficult diagnostic problem. The patient was unconscious, paralysed, and on a ventilator when gas was found in his belly. Although a clear outflow after running one litre of saline into the peritoneal cavity was at first taken as evidence that there had been no intra-abdominal injury, the pronounced subsequent increase in air was thought to warrant exploration. Out of 12 similar cases in adults reported elsewhere, four underwent needless laparotomy.

Pneumopericardium occurred only in case 3. In animal experiments only Grosfield *et al.*¹¹ recorded pneumopericardium, which occurred under the highest of pressures exerted in the lungs—namely, 70 cm H₂O; the patient in my series was receiving an end expiratory pressure of 25 cm H₂O.

How can pneumoperitoneum induced by artificial ventilation be distinguished from that caused by rupture or perforation of a hollow viscus? Absence of a history of peptic ulcer; absence of signs of peritonitis; inability to show distended loops of bowel with fluid levels in erect abdominal radiographs; and the coexistence of subcutaneous emphysema, pneumomediastinum, and pneumothoraces strongly suggest that ventilation has caused the pneumoperitoneum. Contrast radiography of the gastrointestinal tract may be used to prove the state of the gut. Paracentesis and examination of the effluent from peritoneal lavage may show contamination by gastrointestinal contents.

Does injury to the chest contribute to the development of pneumoperitoneum caused by ventilation? Of the 12 previously reported cases in adults, only one occurred after a chest injury. Glauser and Bartlett,⁴ however, described a case of pneumoperitoneum, pneumomediastinum, and pneumothorax that occurred after chest injury alone, artificial ventilation not being used. Macklin and Macklin¹⁰ stated that "atelectasis of part of

the lung . . . through crushing forces which collapse part of the lung . . . may act as a trip hammer to set off the train of events leading ultimately to the invasion of the interstitial tissues of the body by air from the lungs."

Apart from my series, only 12 cases of ventilator-induced pneumoperitoneum have been recorded in adults. The present cases were collected over three years at one hospital, two being seen in the last six months. The syndrome is unlikely to be an isolated occurrence and is probably more common than reports suggest. Awareness of artificial ventilation as a cause of pneumoperitoneum will reduce the number of needless laparotomies performed on patients who are already seriously ill.

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Unknown primary adenocarcinoma: incidence of overinvestigation and natural history

J F STEWART, M H N TATTERSALL, R L WOODS, R M FOX

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Summary and conclusions

Out of 1300 patients referred to a medical oncology unit, there were 87 with metastatic cancer in whom a primary tumour site was not evident from the history and after physical examination and chest radiography had been carried out. An analysis of the investigations performed in these patients and their results showed that in only eight of the 87 patients did non-surgical investigations at presentation determine the primary site. In two patients it was identified by diagnostic laparotomy, and in a further 13 clinical follow-up led to recognition of the primary tumour site before death.

Few investigations should be performed in patients in whom the primary site is not known since they have a low yield, and in our population identifying the primary tumour did not improve the outcome or alter management. Treatable tumours should be excluded, and this may be done in most cases by simple blood tests, particularly those measuring acid phosphatase activity and other tumour markers.

Introduction

Patients often present with metastatic malignant disease without an obvious primary tumour. The dilemma facing the clinician is how aggressively to try to identify the primary site. Traditionally, in medical wards large series of non-invasive investigations

are undertaken, while in surgical wards a more rapid progression to exploratory laparotomy is common. The argument for pursuing the primary site aggressively in these patients is usually based on two beliefs: that finding the primary site might, firstly, lead to specific antitumour treatment and, secondly, give a better guide to prognosis. A possible further reason may be the diagnostic challenge posed by such patients.

We have reviewed the clinical histories of 1300 patients with malignant disease referred to this newly established medical oncology department in a major teaching hospital. Patients who presented initially with metastatic adenocarcinoma and undifferentiated carcinoma (confirmed histologically or cytologically) in whom the clinical history, full physical examination including breast palpation and pelvic examination, and chest radiography did not identify the primary site were studied. Eighty-seven patients (6.7%) fell into this category, and the investigations in these patients and natural history of the disease constitute this report.

Patients and methods

The clinical staff of this institute saw 1300 patients with cancer between January 1977 and October 1978. All clinical histories were reviewed retrospectively, and patients who presented initially with metastatic cancer without a documented primary site on clinical history, examination, and chest radiography were studied further. Only patients with metastatic non-squamous carcinoma based on review of the initial histological specimens were included. All investigations were recorded, including those before referral and those that were not designed specifically to identify the primary tumour site. The unit's policy was to discourage exhaustive investigations to find the primary site, but in patients considered to be suitable for trials of chemotherapy staging investigations to assess the disease were encouraged. The radiological procedures were routine, and nuclear-medicine scans were performed in a single department where the bone-scanning radionuclide used is technetium-labelled pyrophosphate and the liver-scanning agent technetium-99m sulphacolloid. Technetium pertechnetate was used for thyroid and brain scans, and cerebral CAT scans were performed in certain patients. The haematological and biochemical investigations were by standard techniques.

The results of investigations designed to identify the primary sites

Sydney Cancer Therapy Unit, Ludwig Institute for Cancer Research, Royal Prince Alfred Hospital, Sydney, Australia

J F STEWART, MB, BS, registrar

M H N TATTERSALL, FRACP, director, professor of cancer medicine

R L WOODS, MRCP, assistant physician

R M FOX, FRACP, associate professor of cancer medicine