Pneumoperitoneum in Association with Pneumothorax*

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The association of pneumoperitoneum with pneumothorax is considered relatively rare. Over a two-year period five cases of this syndrome were seen in a general hospital setting. Three of the patients had the following in common: (1) ventilatory support; (2) severely noncompliant lungs and/or severely obstructed airways; (3) very high peak inspiratory pressures (greater than 40 cmH2O). In these cases alveolar or tracheal rupture led to the development of pneumoperitoneum. In the remaining cases the pneumothorax and pneumoperitoneum were secondary to (1) diagnostic peritoneoscopy and (2) high pressure compressive blunt trauma to the chest cage.

CASE REPORTS

CASE 1

This 16-year-old boy had a history of severe bronchial asthma since he was two years of age. For the past several years prior to admission he had been dependent on steroid therapy, requiring the equivalent of 25 mg of prednisone per day. One month prior to admission he began to inhale cromolyn sodium and had experienced such marked relief of his symptoms that he had discontinued taking steroids.

Twelve hours prior to the latest admission the patient began experiencing some increase in his cough and shortness of breath. One hour prior to admission he was in severe respiratory distress, necessitating hospitalization. In the emergency room his arterial pH was reported as 6.9, intubation was instituted, and the patient was transferred to the intensive care unit.

On admission to the ICU his respiratory rate was 40-50/minute. Positive physical findings included tracheal tug, hyperresonant chest sounds with diffuse bilateral inspiratory and expiratory wheezes, and a loud and widely split pulmonary second sound (P2).

On a volume ventilator the patient's tidal volume (TV) was 900 ml; peak inspiratory pressures (PIP) 70-80 cmH2O; arterial carbon dioxide pressure (Paco2) 65 mmHg, arterial oxygen pressure (Pao2) 67 mmHg, pH 7.25 and concentration of inspired oxygen (FiO2) 0.45. To facilitate ventilation, the patient was paralyzed with intravenous administration of a curarelike drug.

Twelve hours after admission he was noted to develop subcutaneous emphysema. His TV was 950 ml; PIP 73 cmH2O; Paco2, 76 mm Hg; Pao2, 123 mm Hg; pH, 7.16; and FiO2, 0.5. Chest x-ray film revealed a pneumothorax on the left side (Fig 1a). A chest tube was placed with good expansion of the lung and the Paco2 decreased to 55 mmHg, with the PaO2 increasing to 180 mmHg.

Nineteen hours after admission a routine upright portable chest x-ray film revealed air under the right side of the diaphragm (Fig 1b). Nasogastric suction returned guiac + aspirate. His abdomen was soft, but he was still under the influence of the curarelike drug. Twenty-eight hours after admission his white blood cell count increased to 18,900, with a shift to the left. A diatrizoate methylglucamine 76 percent (Gastrografin) study was negative.

The 30th hour after admission the patient underwent exploratory laparotomy for a possible perforated viscus. At operation retroperitoneal emphysema and air bubbles in the omentum were noted. No perforated viscus was found. At the close of the operation a no. 36 straight tube was placed in the left side of the chest. Immediately after operation the TV was 700 ml; PIP was 49 cmH2O; Paco2, 40 mmHg; Pao2, 85 mm Hg; pH, 7.42; and FiO2 0.35.

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Case 2

This 39-year-old woman entered a local hospital on Sept. 20, 1972 for correction of a hammer toe condition. On the fourth day after operation the patient’s temperature was 38.5°C, and a chest x-ray film revealed bilateral basilar and perihilar alveolar infiltrates. On the fifth day after operation the patient’s condition continued to deteriorate, an ET tube was inserted and mechanical ventilation was begun. On the sixth day after operation a tracheostomy was performed with difficulty. Immediately following this procedure, massive subcutaneous emphysema was noted. The tracheostomy tube was realigned and the subcutaneous emphysema stopped accumulating. She was suspected of having a left pneumothorax, and a chest tube was placed.

On the eighth hospital day the patient was transferred to Orange County Medical Center. She was noted to have a bubbling chest tube, bilateral bronchial breath sounds, moist râles and a markedly protuberant and hyperresonant abdomen. On a volume ventilator the TV was 800 ml; PIP was 50 cmH2O; Paco2 39 mm Hg; PaO2 85 mm Hg; pH, 7.50 and Fio2 0.8. A chest x-ray film revealed a persistent 15-20 percent left pneumothorax and massive subcutaneous emphysema (Fig 2a).

The following day her abdomen was noted to be more distended. X-ray films revealed a massive pneumoperitoneum (Fig 2b). Paracentesis with removal of the air was performed successfully, and a tube was placed in the abdomen for continuous evacuation of air.

On the 15th hospital day the patient underwent repair of a tracheal-esophageal (TE) fistula, which had been noted the previous day. The remainder of her hospital course was highlighted by slow but progressive recovery.

Comment

This patient’s pneumoperitoneum was probably secondary to the high inspiration pressures used (eg, 55 cmH2O and greater), while she was on the volume ventilator. Her primary trauma was probably encountered during the tracheostomy when her mediastinal integrity was breached.

Case 3

This 42-year-old woman was admitted to OCMC on March 11, 1973 after taking an unknown quantity of tranquillizers. On admission she was comatose and unresponsive to deep pain. Due to her clinical state an ET tube was inserted and she was transferred to the respiratory intensive care unit (RICU).

By the fourth hospital day she was awake but confused, and the ET tube had been removed. Her temperature was 39.4°C. Physical examination revealed bilateral pulmonary râles. Chest x-ray films showed bilateral alveolar infiltrates. The Paco2 was 28 mm Hg, PaO2 45 mm Hg and Fio2 0.2.

Comment

Although the pneumoperitoneum was not confirmed until eight hours after a diagnosis of pneumothorax was made, the two were probably directly related. The cause of the abdominal air was alveolar rupture due to the high inspiratory pressures (up to 73 cmH2O) required for ventilation of the patient. Dissection of air into the retroperitoneal space with subsequent rupture into the free peritoneum was the etiologic cause, since the presence of omental and retroperitoneal air was noted at laparotomy.

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Over the ensuing 24 hours her condition worsened and intubation was instituted. She was paralyzed with intravenous administration of a curarelike drug and mechanical ventilation was begun.

On the seventh hospital day a tracheostomy was performed. During the following week the patient's condition continued to deteriorate. On the 21st hospital day the patient was noted to have a massively distended abdomen. X-ray films revealed air in the intestinal tract. Bronchoscopy was performed and a TE fistula noted. On the following day, operation was undertaken for repair. Immediately after operation the TV was 600 ml; PIP was 45 cmH₂O; PEEP, 5 cm; PaCO₂, 24 mm Hg; PaO₂, 53 mm Hg and FIO₂, 1.0.

On the 27th hospital day she was noted to have bilateral 30-40 percent pneumothoraces, probably due to the breakdown of the TE fistula repair and further leakage. The following day a KUB showed an associated pneumoperitoneum.

The patient's condition continued to deteriorate and on the 45th hospital day she died.

Comment

This patient had pulmonary insufficiency related to a low cardiac output state from overdosage and possible aspiration. This necessitated relatively high ventilatory inspired pressures (45 cmH₂O) and the use of PEEP. These elevated pressures probably contributed to her tracheal leak, which led to the pneumothorax and pneumoperitoneum.

CASE 4

This 40-year-old woman was admitted to OCMC after driving her car over a cliff. Examination revealed no abnormalities except for fractured ribs and a right sided pneumothorax, which resolved following chest tube placement. Chest x-ray films revealed subcutaneous and mediastinal emphysema. The following day another chest x-ray film revealed air under the diaphragm. The patient was asymptomatic and had no evidence of a ruptured viscus. The remainder of the hospital course was uneventful, and she was discharged within the week.

Comment

This patient's pneumothorax was secondary to fractured ribs. These were probably caused and accompanied by high pressures applied to the thoracic cage. These high intrathoracic pressures forced air into the abdomen either through a diaphragmatic defect or retroperitoneally.

CASE 5

This 50-year-old man was admitted for evaluation of elevation of the right side of the diaphragm, etiology unknown. Routine tests disclosed no abnormalities. On the third hospital day bronchoscopy was performed without any evidence of tumor. Due to a history of heavy alcoholic intake, a peritoneoscopy was performed and multiple liver biopsies obtained. Following the procedure the patient complained of right shoulder pain and shortness of breath. Chest x-ray films revealed air under the right diaphragm and a 30 percent right pneumothorax. Paracentesis for removal of air was unsuccessful. The patient signed out of the hospital against medical advice.

Comment

In this case the pneumothorax was clearly secondary to the pneumoperitoneum. A diaphragmatic defect had to be present for air to move from the abdominal cavity into the thoracic cavity.
DISCUSSION

The relationship of pneumoperitoneum to pneumothorax may be categorized as follows:

1. The pneumothorax may cause the pneumoperitoneum directly. An example might be that of a ruptured bleb or bullae leading to the development of tension, which then causes air to leak through defects in the diaphragm. None of our cases would fit into this category.

2. Both the pneumothorax and pneumoperitoneum may have a common etiology. Case 1 was secondary to alveolar rupture with subsequent dissection of air into the mediastinum and finally into the pleural and peritoneal spaces. Cases 2 and 3 were slightly different, as tracheal leakage led to the same sequence of events. Case 4 is more puzzling as the thoracic and abdominal air may have been secondary to trauma with rupture of the lung either leading to retroperitoneal dissection or leakage directly through a defect in the diaphragm.

3. The pneumothorax may be caused by the pneumoperitoneum. Case 5 is a relatively common example of this type.

4. A ruptured abdominal viscus may occur simultaneously with a pneumothorax. We felt case 1 belonged in this category, but laparotomy proved us wrong.

Three of the four patients in category 2 had the following in common: (1) very stiff noncompliant lungs and/or severely obstructed airways; (2) mechanical ventilatory treatment; and (3) very high peak inspiratory pressures (40 cmH₂O or greater). Since intraabdominal pressures exceed intrathoracic pressures by an average of 20-30 cmH₂O, both during inspiration and expiration, simple spontaneous pneumothorax, even if associated with mediastinal and subcutaneous emphysema, should not be associated with air under the diaphragm. This would seem to be confirmed not only by the lack of case reports in the literature but also by our experience. In the past four years 42 patients with spontaneous or traumatic pneumothoraces have been admitted to our hospital. Twelve patients had evidence of mediastinal or subcutaneous emphysema by chest x-ray film. None of these patients had air under the diaphragm. Even patients with obvious tension pneumothoraces did not develop this complication. This may be due to either the rapidity of treatment of the tension or inadequate buildup of intrathoracic pressure. Grosfeld et al.⁹ using cats, found that when intratracheal pressures exceeded 40 cmH₂O pressure, interstitial emphysema routinely developed. When pressures exceeded 50 cmH₂O pressure, pneumomediastinum occurred. At pressure above 60 cmH₂O, subcutaneous emphysema and pneumoperitoneum were observed. These findings confirmed the results found in rats by Donahoe et al. and in dogs by Lenaghan et al.¹⁰ These latter investigators found that lung rupture occurred at lower intratracheal pressures when "shock lung" conditions were produced. This type of condition was present in patients 2 and 3.

The route of dissection to produce most cases of pneumoperitoneum secondary to pneumothorax would seem to be as follows: under pressure, air dissects from ruptured alveoli along the sheath of adjacent vessels to the mediastinum. As pressures increase, dissection can occur both into the pleural space and along the thoracic great vessels and esophagus. This air then moves into the retroperitoneal space where rupture finally occurs. Case 1 would seem to fit these details, as retroperitoneal air was present at operation and no diaphragmatic defects were apparent.

That air can also move directly across the diaphragm through defects is apparent from the literature¹¹ and our case (no. 5) in which diagnostic peritoneoscopy led to pneumothorax. Since minimal amounts of air, low pressures and no retroperitoneal sites are involved, direct diaphragmatic extension seems the only reasonable explanation for the route of air movement from the abdomen to the thorax in these cases.

The hazards of pneumoperitoneum in association with pneumothorax are several. Firstly, increased air under the diaphragm may add to respiratory pressures decreased from 60 cmH₂O to approximately 50 cmH₂O after a laparotomy and removal of the air. Secondly, pneumoperitoneum may raise the question of a perforated viscus. Once again, patient 1 was subjected to operation due to this consideration. Patient 2 was also felt to have a possibility of perforated viscus. To rule out this diagnosis the following two measures may be used: (1) Diatrizoate methylglucarnine 76 percent studies of the gastrointestinal tract should be made. If no perforation is seen, the possibility of early closure or an overlooked area of perforation must still be considered. (2) Paracentesis should be repeated for removal of air and determination of any relationship between the inspiratory pressure and the inspiratory pressure of the thorax.

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Paper

In social groups of ancient times, the urge of speedy and detailed communication prompted search for new means so as to get away from records carved in stone or cast in clay. The best known information concerning the history of paper making is related to the use of *Cyperus papyrus*, the common reed from the name of which the word "paper" originates. Paper reed growing in abundance on the marshy banks of the lower Nile was the source of paper from about 2200 BC to 400 AD. Its stalks were split into thin slices which were pressed into sheets. Another way of producing paper was invented by the Chinese, Ts'ai Lun, in 105 AD. He matted into sheets fibers of bamboo and the inner bark fibers of mulberry tree. Nearly 800 years later a paper mill employing this Chinese technology was established in Bagdad, and one in France about 400 years later. On the American continent, Germantown, Pennsylvania, became the site of a paper mill in 1690. Continuous sheets of paper were the invention of Nicolas Louis Robert of France in 1799. The manufacture of cardboard was discovered, through the accidental production of strawboard, by the American George Shyrock. Paper manufacturing was radically changed and its cost substantially reduced since the invention of a machine by F Keller and H Volter of Germany for grinding wood into pulp, in 1867. Although raw material for paper is available in other vegetable fibers, such as cotton, bagasse, bamboo, manila rope, esparto, cereal straws, flax straw, it is stated by Thode, E F (Grolier Encyclopedia International, New York, Grolier, 1964) that "wood has almost entirely replaced rags and other vegetable fibers as a raw material for paper. In the United States, wood annually supplies more than 97 percent of the raw material for paper production. There are over 800 paper and paperboard mills in the United States. In fact, more than 400 lbs of paper is consumed by or for every American each year." As a foreign body wood dust may cause irritation of the air passages and alveoli. Almost half a century ago Bahn, K (Klin Wochenschr 7:1963, 1928) wrote on allergy in woodsawers. Shortly thereafter others reported on sensitivity to wood and on the consequent occurrence of asthma-like disease. An early report related to the paper industry was published by Towey, J W et al (JAMA 99:453, 1932). It described granulomatous interstitial pneumonia caused by the fungus *Cryptosoma corticale*. The latter was found under the bark of maple logs. The disease was found in laborers engaged in stripping barks from maple logs prior to cutting and chipping in preparation of wood pulp. Sosman A J et al (New Engl J Med 281:977, 1969) distinguished two types of sensitivity reaction to wood dust: 1. the acute type with wheezing and dyspnea within minutes of exposure and with prompt cessation of symptoms after exposure ceases; 2. the delayed-onset type is slow and has a gradual appearance of wheezing which is of a longer duration. They confirmed the view of Pepys, J (J R Coll Phys London 2:42, 1967) that inhalation challenge with appropriate wood dust is the most expedient method of diagnosis in these patients inasmuch as no specific skin reaction can be elicited. In their opinion, "immunologic mechanisms other than those mediated by IgE are involved in the pathogenesis of this illness". Schluetter, D P et al (Ann Int Med 77:907, 1972) reported two cases of wood-pulp workers' disease: hypersensitivity pneumonia resulting from prolonged exposure to air contaminated with the mold Alternaria. The dust originated from debarking logs, cutting them into chips and delivering the latter on conveyors to bins and digestors for making wood pulp. Bleaching chemicals are used extensively in the manufacture of white paper, particularly sulphur dioxide and chlorine. In addition to the deleterious effect of sulphur dioxide upon lung structures, it causes cough, wheezing and increased resistance to air flow when inhaled in concentration of 20ppm. Chlorine is not only a respiratory irritant which may cause tracheobronchitis, bronchospasm, cessation of ciliary function and pulmonary edema but also, as pointed out by Johnstone, R T et al (Occupational Diseases and Industrial Medicine, Philadelphia, Saunders, 1960) it damages the lung tissue through its intense oxidizing action, during which ozone, a potent protoplasmic poison, is produced.

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