Pneumoperitoneum Complicating Bronchoscopy

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Since its introduction into clinical practice by Ikeda et al. [1], fiberoptic bronchoscopy has become one of the most frequently performed invasive procedures in pulmonary medicine, providing a wide range of diagnostic and therapeutic applications. However, FOB is not without danger; the most common hazards – secondary to instrumentation or biopsy procedures – are transient hypoxemia, arrhythmias, minor hemorrhage, and pulmonary infiltrates [2]. Pneumothorax is less frequently reported, while pneumomediastinum, pneumoperitoneum and air embolism are very rare.

Pneumoperitoneum not resulting from diaphragmatic or abdominal viscus rupture, but rather as an extension of pneumomediastinum has seldom been reported. We present this rare complication following bronchoscopy, culminating in complete recovery of the patient.

Patient Description

An 82 year old woman was referred to the pulmonary clinic at Barzilai Medical Center in January 2001 for evaluation of an incidental X-ray finding in an abdominal computed tomography scan done as part of a work-up of epigastric pain and a 10 kg weight loss from which the patient had suffered for 5 years prior to referral. Her past medical history included cholecystectomy and two breast biopsies, which revealed fibroadenomas. She had undergone several diagnostic procedures including upper and lower gastrointestinal tract endoscopies, the first of which was within normal limits and the latter revealed mild diverticulitic changes in the descending colon. As mentioned above, the abdominal

FOB = fiberoptic bronchoscopy

CT scan was within normal limits but the upper cuts revealed a solid homogeneous 2x3cm mass attached to the mediastinal pleura of the right lower lobe. Physical examination and laboratory work-up were within normal limits.

The patient was prepared for FOB by an intramuscular injection of pethidine, and after anesthetizing her throat with lidocaine spray she was sedated by an intravenous midazolam injection. During FOB, the only gross pathologic finding was a concentric narrowing of the right lower lobe apical segment orifice. Bronchoalveolar lavage was performed. During the second transbronchial biopsy the patient suddenly became stuporotic and bradycardic to 60 beats per minute. Vital signs changed from an initial blood pressure value of 135/90, respiratory rate of 20 and arterial O_2 saturation of 92%, to blood pressure 115/80, respiratory rate 34/min and O_2 saturation 91%. The patient received preventive oxygen supplementation via nasal cannula and an intravenous injection of flumazenil. Since no change in consciousness was observed and a cerebrovascular event could not be ruled out, nasotracheal intubation was performed for airway protection. Because air embolism was a possible explanation for her sudden mental deterioration, the patient was placed in a left lateral and Trendelenburg position. She was ventilated manually with an ambu-bag for a short time only, to check placement of the endotracheal tube. Her oxygenation, ventilation and hemodynamic status remained stable thereafter. A plain X-ray revealed a small left pneumothorax, pneu-

momediastinum and a large pneumoperitoneum [Figure]. An upper and lower abdominal contrast-enhanced CT scan ruled out a traumatic esophageal or other viscus rupture, and esophagoscopy was therefore not done.

In the operating room a diagnostic abdominal laparoscopy showed no evidence of a perforated viscus. The patient was transferred to the internal medicine department and recovered uneventfully in 48 hours. She was discharged from hospital on the third day after FOB.

Comment

FOB complications can be classified into those occurring secondary to: a) premedication and anesthetics (i.e., hypoventilation, bronchospasm, laryngospasm, etc.), b) technical problems (trauma to airways during endotracheal tube insertion or false route formation), and c) effects of instrumentation or biopsy. Minor complications secondary to the latter two procedures occur in less than 0.2–10% of cases [2], and include hypoxemia, arrhythmia and bleeding of less than 50 ml. Major compli-

Pneumoperitoneum (upper part) following transbronchial biopsy.

cations reported in 0.08–1.7% of cases include pneumonia, aspiration of gastric contents, septic shock, and acute pulmonary edema [3]. Pneumothorax has been reported in 1–5% of cases, major hemorrhage (more than 50 ml) in less than 0.5%, and mortality in 0.01–0.5% of cases [3]. Pneumomediastinum appears mostly after transbronchial needle aspiration as a primary event and is a rare sequela of pneumothorax [4]. Since intraabdominal pressure exceeds intrathoracic pressures by an average of 20–30 cmH_2O both during inspiration and expiration, simple spontaneous pneumothorax, even when associated with mediastinal and subcutaneous emphysema, should not cause pneumoperitoneum via air leakage under the diaphragm. In the rare cases of spontaneous pneumomediastinum causing pneumoperitoneum described in the literature, the risk factors were high airway pressures (peak inspiratory pressure exceeding 40 cmH_2O and positive end-expiratory pressure exceeding 20 cmH_2O), large tidal volumes during mechanical ventilation, non-compliant lungs, and preexisting pulmonary disease [5]. None of these factors was relevant to our patient.

The relationship of pneumothorax, pneumomediastinum and pneumoperitoneum in general can be explained by the following theories:

- They are directly caused by transbronchial biopsies performed during the bronchoscopy either by direct passage of air through pleural and diaphragmatic defects or by the classically described passage of air via the mediastinum along perivascular connective tissue or major diaphragmatic portals to the retroperitoneum and finally to the peritoneal space. Macklin and Macklin [5] were the first to report the transport of air along vascular sheaths from the alveoli to the mediastinum and then to the retroperitoneum and peritoneum. Subsequent reports suggest an interesting alternative to the classic pathway, i.e., air passage through microperforations or parallel pillars of the diaphragm in areas of demonstrated weakness, such as posterolateral or parasternal regions, which correlate with hernias of Bochdalek and Morgagni respectively. These passageways may alternatively simply represent natural microscopic passages analogous to the proposed mechanism of cirrhotic fluid traversing the diaphragm. This was most probably the mechanism of pneumomediastinum and pneumoperitoneum formation in our case.
- Another possibility, although not present in our case but worth mentioning, is that pneumoperitoneum was the trigger. It has been reported that pneumoperitoneum resulting from peritoneoscopy might lead to pneumothorax via direct thoracic spread of air from the abdomen.
- Abdominal viscus perforation can also occur, causing simultaneous pneumothorax and pneumomediastinum. The laparoscopy ruled out such a possibility in our patient.

In conclusion, although pneumoperitoneum results mostly from abdominal catastrophes, in our case it probably derived from the few ambu blows administered immediately after preventive intubation, forcing air from pneumomediastinum into the abdominal cavity. The clinician should always keep in mind that any change in the patient's mental status may be due to the abdominal distension (causing both decreased venous return and decreased oxygenation via basal lung areas compression) and not a result of the hemodynamic catastrophe [4,5], and that this early neurologic manifestation can sometimes be the sole clinical manifestations of pneumoperitoneum. A high index of suspicion should always be held to the possibility of pneumoperitoneum being a complication of an extraabdominal procedure like FOB, tracheostomy or blunt chest trauma.

In most cases of pneumoperitoneum, non-surgical treatment is indicated, as in our case, and had the preventive intubation not raised the possibility of esophageal perforation, explorative laparoscopy would not have been necessary and the patient would have recovered with bed-rest and supplemental oxygen only.

References

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Capsule

Rat-to-human transmission of cowpox infection

Woifs et al. isolated cowpox virus (CPXV) from the ulcerative eyelid lesions of a 14 year old girl, who had cared for a clinically ill wild rat that later died. CPXV isolated from the rat (*Rattus norvegicus*) showed complete homology with the girl's virus. This case is the first proven rat-to-human transmission of cowpox.

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