Air Embolism Complicating Percutaneous Needle Biopsy of the Lung

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Radiographically and pathologically documented air embolism occurred following percutaneous needle aspiration of the lung. While air embolism is uncommon, it is more likely to occur when the needle is inserted into a rigid portion of the lung as occurs with extensive consolidation, abscess formation, or extensive pleural disease.

Percutaneous needle biopsy and aspiration have gained wide acceptance for the diagnosis of pulmonary lesions. Minor complications such as pneumothorax and hemoptysis are common, but reported fatalities are rare. Only six cases have been found in which needle biopsy or aspiration resulted in the death of the patient.1-5 The deaths were caused by bronchial hemorrhage (three cases), tension pneumothorax (one case), probable air embolism (one case), and unknown causes (one case).

In the past, air embolism was a well recognized complication of thoracentesis and therapeutic pneumothorax,6-12 but there have been no prior reports of confirmed fatal air embolism following diagnostic aspiration or biopsy of the lung. The purpose of this presentation is to report a case of radiographically and pathologically documented fatal air embolism which probably occurred during percutaneous needle aspiration of the lung. The mechanisms of pulmonary embolism and possible ways to minimize its occurrence are also presented.

CASE REPORT

A 62-year-old man had had chronic lymphocytic leukemia for 11 years. He entered the University of Utah Medical Center in November of 1971 with right lower lobe pneumonia which later progressed to abscess formation. The infection initially improved but then worsened and progressed, and the patient was eventually treated with antimicrobial agents. He continued to have fever and increased WBC count and eventually required assisted ventilation. Just prior to death, the patient coughed up a frothy mixture of blood and sputum. Bronchopneumonia was present in the lungs, liver, spleen, kidneys, lymph nodes, adrenals, and bone marrow. Bronchopneumonia was present in the lungs, and cultures revealed only rare Enterobacter and Enterococci.

FIGURE 1. Lateral skull roentgenogram obtained immediately after death. Note air in basilar, posterior cerebral and middle cerebral arteries (arrows). Air in many smaller middle and anterior cerebral branches does not visualize on reproduction. Contrast material in subarachnoid cisterns is from previous myelography.

The patient coughed approximately 2 to 3 tablespoons of blood and was instructed to move his head to the side. He did not respond, and immediately urinated and defecated. His pulse was weak, and within a few seconds it was not palpable. External cardiac massage, assisted ventilation, and later defibrillation and resuscitative measures were all unsuccessful, and the patient was pronounced dead approximately 30 minutes later.

Roentgenograms of the skull were obtained immediately and showed air in the basilar artery and in numerous large and small branches of the middle cerebral arteries (fig 1). Postmortem examination revealed approximately 350 ml of bloody fluid in the left pleural cavity. There was extensive bilateral bronchopneumonia, and there was an old abscess cavity in the right lower lobe. There was a small puncture wound in the superior segment of the left lower lobe which was approximately 3 cm deep, and there appeared to be a communication between the puncture wound, a small peripheral bronchus, and a peripheral pulmonary vessel. There was some blood in the bronchi adjacent to the puncture. A frothy mixture of air and blood was present in the cardiac chambers, and numerous air bubbles were seen in the coronary arteries (fig 2).

Histologic examination revealed lymphocytic infiltration of the lungs, liver, spleen, kidneys, lymph nodes, adrenals, and bone marrow. Bronchopneumonia was present in the lungs, but cultures revealed only rare Enterobacter and Enterococci.

DISCUSSION

In a discussion of air embolism, it is important to...
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Separate peripheral venous from pulmonary venous air emboli. Although fatalities from venous air embolism have been reported, small quantities of air in the right heart and pulmonary arteries cause no difficulty, and large quantities have also been well tolerated, especially if injected slowly. The amount of air required to produce injury and death is much less when air embolism occurs from the pulmonary veins. Experimental studies in dogs have shown that as little as 2 to 3 ml of air injected in the pulmonary veins may be fatal. Death from pulmonary venous air embolism has been described in many circumstances, including thoracentesis, therapeutic pneumothorax, pleural irrigation, pulmonary and cardiac surgery, status asthmaticus, lung biopsy and aspiration, scuba diving, and even during trumpet playing. Some authors contend that the syndrome of pleural shock occurring with thoracentesis is also due to air embolism.

Air embolism should be considered if a sudden central nervous system catastrophe or sudden shock occurs during chest surgery or during needle puncture of the lungs, pleura, or pulmonary veins. The diagnosis is further suggested or confirmed by the presence of frothy blood from bleeding vessels, air in the retinal vessels, and by roentgenograms of the chest and skull showing air in the heart, great vessels, or cerebral circulation (present case).

In view of the very large number of cases in which needles are introduced into the lungs or pleura, it is surprising that air embolism does not occur more frequently. Special circumstances apparently must exist before air embolism can occur. It is highly unlikely that puncture or laceration of a normal lung would result in air embolism. Chiu et al incised the lungs of dogs at varying levels of intrabronchial pressure. At normal breathing levels, no embolism occurred. With increasing positive pressure, the bronchial and pulmonary venous pressure both increased, but eventually the venous-bronchial pressure-gradient was reversed so that bronchial pressure exceeded that in the veins, and air embolism occurred. In the presence of a diseased and rigid lung or pleura, the vein cannot be pushed aside by the needle and once punctured, it cannot collapse. Most, if not all, cases of fatal air embolism following needle puncture of the lung have occurred in areas of advanced consolidation or pulmonary or pleural abscess formation.

Aside from situations where air is introduced directly into the lung, (as may accidentally occur with therapeutic pneumothorax) there are two ways in which air embolism can occur at the time of lung needle puncture: (1) Atmosphere to pulmonary vein. If the needle tip happens to lodge within a pulmonary vein and if there is no obstructing stylet or stopcock, and if atmospheric pressure exceeds pulmonary venous pressure (as might occur during rapid inspiration), then air embolism may occur. (2) Bronchus or lung to pulmonary vein. A needle may pierce a cavity, air cyst, or bronchus and a nearby pulmonary vein. If the lung is rigid and the vein does not collapse and if the pressure in the air cyst or bronchus or cavity exceeds venous pressure, air may pass from the lung to the pulmonary vein. It is of interest that the patient reported here coughed two to three times when the needle was inserted. Intrapulmonary pressure rises markedly to 180 cm of water or even higher during the early phase of coughing prior to glottic opening. Since the needle traversed both a small peripheral bronchus and vein, it seems likely that air was blown into the vein during the coughing episode. A similar sequence occurred in a case reported by Van Allen et al; the patient coughed during attempted needle puncture of a lung abscess, and fatal air embolism immediately followed.

It is conceivable that the air embolism episode in the present case was induced by the attempted cardiac resuscitation. However, the clinical sequence of immediate unresponsiveness, loss of sphincter control, and circulatory collapse immediately following the needle puncture would favor air embolism. Also, while air embolism could theoretically occur as a result of attempted cardiac resuscitation, no reports of this complication have been found.

While death from air embolism is uncommon, certain precautions could probably reduce its incidence even further. Embolism from the atmosphere to the pulmonary vein can be prevented by any mechanism which maintains intrathoracic pressure greater than atmospheric pressure but most of the maneuvers which accomplish this (positive pressure breathing and the Valsalva maneuver), also increase the likelihood of lung to pulmonary vein embolism. The simplest way to avoid atmosphere to pulmonary vein communication is to make sure that the needle is always closed with either a stylet or with the operator's finger, and that if the needle must be left open for a short period, that respiration and movement be completely suspended.

Avoidance of bronchopulmonary-venous air embolism is more difficult to prevent, but the patient should be thoroughly instructed to avoid coughing and straining.
and the procedure should probably be abandoned in the patient who is coughing frequently. Whenever possible, one should also try to avoid cystic and cavitary areas of the lung, and those areas that are maximally consolidated, or in which there is extensive pleural thickening.

References

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Endobronchial Polyp

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A case of benign endobronchial polyp is reported. The unusual feature is that the major signs and symptoms, consisting of dyspnea, absence of breath sounds in one lung, and massive atelectasis of the same lung at roentgenologic examination, occurred suddenly. A "polyp," exactly duplicating the structure of the benign nasal polyps, was removed at endoscopy, with immediate relief of symptoms and return to normal conditions. In the absence of any history of chronic inflammatory disease of the respiratory tract and in the light of the microscopic structure of the polyp showing prominent edema and congestion, it is assumed that the endobronchial lesion resulted from a progressive accumulation of edema fluid in the bronchial mucosa, ultimately giving rise to massive mucosal herniation and bronchial occlusion.

Carcinoma of the bronchus is by far the most common intrabronchial lesion causing obstruction of the respiratory passages. However, in a meager 5 to 10 percent of the cases, the bronchial obstruction is found to be underlined by a benign bronchial tumor. Jackson and Jackson's review of benign tumors and tumor-like conditions in the tracheobronchial tree extends over 20 different pathologic conditions.

Among the benign growths, the endobronchial polyps form a distinct category. With only a handful of cases recorded in the literature, the chance that the obstruction is produced by an inflammatory polyp arising from the bronchial mucosa is indeed very small; nonetheless, its existence is well substantiated and should not be ignored. The significant point is that once the nature of the process is ascertained at biopsy, the lesion can be eradicated completely by the endoscopic route, avoiding recourse to unnecessary major surgery. However, it must be made clear that the concept of benign lesion applied to the polyp of the tracheobronchial tree is not synonymous with innocence inasmuch as if the growth is not timely removed, it may cause obstruction of the air passages, a condition which can result in asphyxia, as well as in suppurrative lung disease by preventing normal ventilation and drainage. These aspects of the endobronchial polyp are well illustrated by our patient.

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