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Clinical report

Cerebral air embolism complicating percutaneous thin-needle biopsy of the lung: complete neurological recovery after hyperbaric oxygen therapy

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Introduction

Percutaneous thin-needle biopsy of the lung is a well-established method for obtaining pulmonary tissue for histological examination because of its minimal invasiveness and excellent diagnostic accuracy [1,2]. It is generally safe, but some complications have been recognized. The most frequent complication is pneumothorax [3]. Systemic arterial air embolism is a very rare but sometimes fatal complication. Here we report a case of cerebral air embolism after percutaneous thin-needle biopsy in a patient who showed complete neurological recovery after hyperbaric oxygen therapy.

Case report

A 75-year-old male patient with a recent body weight loss (5 kg in a month) was admitted because of an abnormal chest radiograph. A chest computed tomographic (CT) scan showed a mass lesion (5 cm in diameter) in the left lower lobe (S10) and a cystic lesion (1.2 cm in diameter) in the right lower lobe (S9) of the lung. A specimen from the mass lesion, obtained via flexible fiberoptic bronchoscopy, revealed squamous cell carcinoma, and radiation therapy was chosen because surgical resection was not possible because of his impaired pulmonary function. During the radiation therapy, the cystic lesion gradually grew and the cystic wall became thinner, suggesting overinflation caused by obstruction of air outflow. In the last 3 weeks of therapy, the cystic lesion grew so rapidly that CT-guided percutaneous needle biopsy of the lesion was scheduled. The patient's past history included diabetes mellitus and familial tremor, which had been successfully treated with glibenclamide (2.5 mg/day) and clonazepam (2 mg/day), respectively. Routine laboratory tests before the biopsy showed normal coagulation function; normal blood chemistry; normal red blood cell, white blood cell, and platelet counts; and impaired pulmonary function (percent vital capacity 79.7% and forced expiratory volume in 1 s/forced vital capacity ratio 62.8%). An electrocardiogram showed a sinus rhythm with a rate of 65 beats/min.

CT-guided percutaneous thin-needle biopsy was performed for the cystic lesion (S9 in the right lung). With the patient in a prone position, an 18-gauge thin-walled Greene needle (Cook Critical Care, Bloomington, IN, USA) was vertically inserted into the lesion under CT guidance by two radiologists (Fig. 1). During this procedure, the patient was instructed to breathe quietly, and he was able to maintain suspended inspiration easily without coughing or discomfort. Immediately after the biopsy procedure, the patient suddenly became unresponsive and apneic, with seizures involving the left side of the body. The radial artery was not palpable; however, the carotid artery was palpable and tachycardic. Immediate ventilatory support was provided with a bag and mask. Approximately 5 min later, spontaneous breathing returned; however, the patient remained in a coma and presented with left hemiplegia. His pupils were moderately dilated and reactive to light, with conjugate movement of the eyes to the right. His blood pressure was 120/75 mm Hg, and his pulse was irregular, with a rate of 65 to 72 beats/min. Chest and brain CT scans were obtained after recovery of spontaneous breathing (15 min after the event). The brain CT scan with a window width of 300 HU and a level of 60 HU, demonstrated a small intravascular air bubble in the

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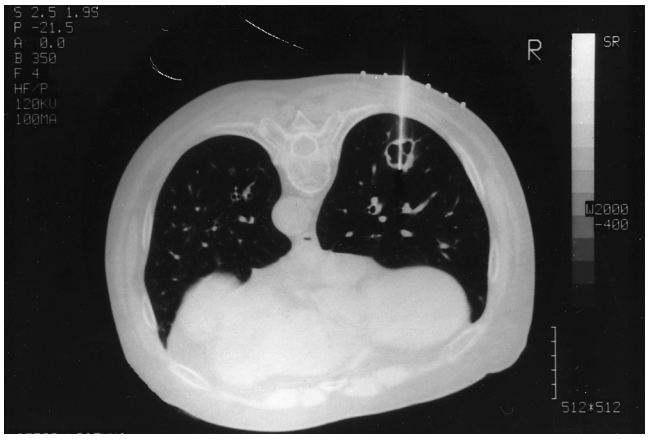

Fig. 1. Chest computed tomography scan during percutaneous needle biopsy. An 18-gauge needle was successfully inserted into the cystic lesion in the right lower lobe of the lung (S9) with computed tomographic guidance

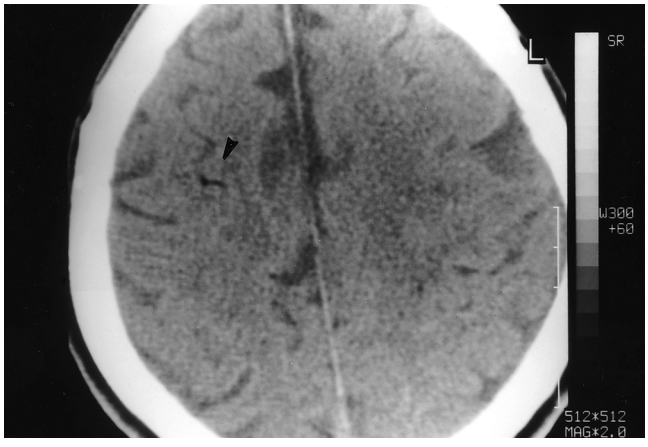

Fig. 2. Cerebral computed tomography scan 15 min after onset of signs and symptoms. A small intravascular air bubble was seen in the right parietal lobe

right parietal lobe (Fig. 2). The lung and mediastinal CT scan did not show any abnormal changes compared with the findings on the preceding scan. The patient was immediately transferred to the intensive care unit. On arrival at the intensive care unit about 30 min after the event, the patient remained in a coma and had left hemiplegia with hyperreflexia. His pupils were not dilated and were promptly reactive to light, with right conjugated deviation of the eyes. Vital signs were stable (blood pressure, 140/60 mm Hg; pulse, irregular with a rate of 56–86 beats/min; breathing rate, 16 breaths/min; and axillary temperature, 36.7°C). An electrocardiogram showed atrial fibrillation and no evidence of myocardial ischemia. Hyperbaric oxygen therapy (100% oxygen, 3 atm for 60 min) was initiated within 60 mins after the initial event. After the hyperbaric oxygen therapy, the patient's level of consciousness improved, and he was able to open his eyes in response to vocal commands. He recovered completely from the hemiplegia of the left lower extremity and the right conjugate deviation of eyes. However, hemiplegia of the left upper extremity remained. On the next day, after a second session of hyperbaric oxygen therapy, he became cooperative and was able to speak, with dysarthria. The remaining hemiplegia had resolved. A brain CT scan showed disappearance of the intravascular air bubble and no evidence of cerebral infarction. On the third day, the dysarthria had resolved. The patient was transferred to the general ward, with normal neurological function, other than the previously present tremor. Histological examination of the needle biopsy revealed squamous cell carcinoma, and radiation therapy was initiated.

Discussion

Percutaneous needle biopsy of the lung is a commonly used diagnostic procedure with good accuracy for histological diagnosis [1,2]. It is generally regarded as a safe procedure that has limited morbidity and extremely rare mortality [2]. The most frequent complications are pneumothorax (27%), pulmonary bleeding (11%), and hemoptysis (7%) [3]. These minor complications are generally self-resolving and do not require clinical intervention [2]. Rare complications include systemic arterial air embolism, tumor implantation, and empyema [2]. Systemic arterial air embolism is extremely rare (0.5–0.8 per 1000) [3–5], but it is serious and often fatal [6–10]. To our knowledge, there have been only nine documented case reports of systemic arterial air embolism related to this biopsy technique over the past 30 years (Table 1) [4–12].

Air in the pulmonary venous system embolizes mainly to coronary and cerebral arteries, with catastrophic consequences [13]. Only 2 ml of air directly injected into the cerebral circulation can be fatal [13]. Similarly, 0.5 to 1.0 ml of air injected into the pulmonary vein can cause cardiac arrest arising from coronary air embolism and myocardial ischemia [13].

There are two possible ways in which air can be introduced into the pulmonary venous system during percutaneous needle biopsy of the lung [6]. First, air may directly enter the pulmonary venous system through the needle. If the tip of the needle is placed into a pulmonary vein while the base of needle is exposed to the atmosphere, and the atmospheric pressure exceeds the pulmonary venous pressure, as may occur during deep inspirations, then air embolism ensues. However, in our patient, this process was unlikely to have occurred because the base of the needle had been carefully

Table 1. Summary of published case reports of systemic air embolism occurring during percutaneous needle biopsy of the lung

Authors	Lung lesion	Detected air	Risk factor	HBO (time to HBO)	Outcome
Westcott [6] (1973)	Cavity	Brain and heart	None	Not performed	Lethal
Aberle et al. [7] (1987)	Cavity	Not detected	Cough	Not performed	Lethal
Tolly et al. [10] (1988)	Solid	Heart	Cough	Not performed	Complete recovery
Cianci et al. [11] (1987)	Consolidation	Brain[a]	None	Performed (11 h)	Left hemiplegia
Baker and Awwad [8] (1988)	Consolidation	Brain[a]	IPPV	Not performed	Lethal
Worth et al. [4] (1990)	Solid	Heart	IPPV	Not performed	Complete recovery
Wong et al. [5] (1995)	Solid	Aorta[a]	Vasculopathy	Performed (4 h)	Left hemiplegia
Regge et al. [12] (1997)	Consolidation	Aorta and pulmonary vein[a]	Pneumothorax	Performed	Complete recovery
Kodama et al. [9] (1999)	Cavity	Brain and heart[a]	None	Not performed	Lethal
Present patient	Cavity	Brain[a]	Overinflation	Performed (1 h)	Complete recovery

HBO, Hyperbaric oxygen therapy; IPPV, intermittent positive pressure ventilation
[a] Indicates air bubbles detected by computed tomography

occluded by the fingers of the radiologist. Second, a needle may penetrate an air-containing space such as the alveolar space, bronchus, cavity, or air cyst and a nearby pulmonary vein at the same time, which could create a communicating fistula. Under those circumstances, if the lung is rigid, the vein does not collapse, and the pressure in the air-containing space exceeds the venous pressure; thus, air may enter from the lung to the pulmonary vein. Cough, straining, or the Valsalva maneuver can increase the pressure in an air-containing space, resulting in an air embolism [6]. There were no such episodes in our patient. However, over-inflation in the cystic lesion was present, which could possibly have contributed to air embolism.

Diagnosis of a systemic air embolism is difficult. It is essential for the physician to suspect it based on clinical symptoms such as abrupt deterioration of neurological and/or cardiovascular status. Brain and chest CT scans can provide a definitive diagnosis by showing air bubbles in cerebral vessels [9–11], the aorta [5,12], the left atrium and ventricle [10], or the pulmonary vein [12]. However, in some instances patients, air bubbles are not readily detected on a CT scan taken immediately after the suspected incident [7]. In such instances, air may be more detectable on CT scan by changing the level of the window to negative. Coronary air embolism may cause electrocardiographic evidence of ischemia, arrhythmias, depression of myocardial function, myocardial infarction, and sudden death. Our patient showed transient hypotension and atrial fibrillation rhythm immediately after the event, presumably caused by coronary air embolism. Cerebral air embolism may cause focal defects, seizure, and coma, which primarily depend on the exact location of the arterial occlusion and the volume of air disseminated to the brain. An ophthalmoscopic examination may be diagnostic by showing air bubbles in retinal vessels [5].

Initial treatment of systemic air embolism consists of the immediate administration of 100% oxygen, placing the patient in a left lateral decubitus position with lowering of the head [4,13]. The administration of an anticonvulsant to control seizures, a large single dose of steroid to reduce cerebral edema, and medications to antagonize platelet aggregation is helpful [4,13]. Hyperbaric oxygen therapy is a definitive therapy for systemic air embolism, and can reduce bubble volume, improve tissue oxygenation, and reduce biochemical reactions at the blood-gas interface that result in hemostasis, endothelial damage, and activation of leucocytes [14]. Thus, patients with systemic air embolism should be treated with hyperberic oxygen therapy as soon as initial resuscitation and diagnostics are completed [13]. In patients with cerebral air embolism, immediate hyperbaric oxygen therapy was reported to decrease the mortality rate to 7% [15]. However, delayed hyperbaric oxygen therapy may be also effective for survival and neurological recovery, even after many hours have elapsed, because air bubbles have been demonstrated up to 48 h after the initial event [16].

In our patient, cerebral air embolism was immediately diagnosed on the brain CT scan, hyperbaric oxygen therapy was initiated within 60 min after the onset, and the amount of air bubble was small, which was evidenced by a relatively stable cardiovascular status and no detectable air bubbles other than in the brain. These factors may have contributed to the patient's complete neurological recovery from air embolism.

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