Cerebral air embolism after transthoracic aspiration with a 0.6 mm (23 gauge) needle

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ABSTRACT: A 54 yr old man experienced weakness in his legs, ataxia and subsequent urinary retention after a percutaneous fine-needle aspiration of a tumour in the right lower lobe. Clinical neurological examination and electroencephalography revealed signs of a brain stem lesion, probably due to an air embolism to the basilar artery. The symptoms and signs gradually disappeared prior to, as well as after, hyperbaric oxygen treatment. We believe this to be the first case of air embolism after transthoracic puncture with a 23 gauge needle to be reported in medical literature.

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Air embolism is a serious complication following percutaneous lung puncture and several cases have been reported with needles less than 20 gauge [1-3]. We report a case where cerebral air embolism occurred after transthoracic aspiration with a 23 gauge (0.6 mm) needle.

Case report

A 54 yr old man was admitted for examination of a lesion in the right lower lobe discovered on a routine chest roentgenogram. Seven years earlier he had undergone unilateral orchidectomy and radiation therapy for stage II testicular seminoma. He had smoked 20 cigarettes daily for 31 yrs, but had stopped smoking at the age of 47 yrs.

On admission, clinical examination revealed a normal respiratory, cardiovascular and nervous system. Forced vital capacity (FVC) and forced expiratory volume in one second (FEV₁) were 88% and 83% of predicted, respectively [4]. The coagulation status was normal. Bronchoscopy showed an open central bronchial tree, and bronchial brushings and biopsies did not provide a cytological or histological diagnosis.

Percutaneous fine needle aspiration was performed under fluoroscopic guidance with a Philips Diagnost ARC A and a Philips angio Diagnost 3 table with the patient in a prone position. 0.6 mg atropine sulphate, 10 mg diazepam and 10 mg hydrokone bitartrate was given 60 min prior to the investigation. A needle guide (Gillette 19 gauge and 40 mm) was inserted at the upper margin of a rib after local anaesthesia (2% Lidocaine) and advanced to the front of the parietal pleura [5]. A 23 gauge and 15 cm long needle with inner stylet (Chiba Depts of Thoracic Medicine', Neurology'', Surgery''' and Radiology', University of Bergen, and Royal Norwegian Navy, Medical Dt[†], 5078 Haakonsvern, Norway.

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biopsy needle W. Crook, 4632 Bjaeverskov, Denmark) was inserted to a depth of 12 cm of the lower lobe. The fluoroscopic control showed that the needle was located within the tumour. The inner stylet was removed and a 20 ml syringe was attached. During aspiration the needle was moved back and forth in the lesion. Suction was then released and the needle with the syringe attached was withdrawn. Immediately after the withdrawal the patient started coughing and had a haemoptysis of 5–10 ml. He then complained of chest pain lasting 4–5 min. No seizures or disturbance of consciousness were observed by the two attending physicians and two nurses. A supine chest roentgenogram showed no signs of pneumothorax and the electrocardiograph (ECG) was normal.

Thirty-six hours after the procedure the patient made the staff aware of a weakness in his legs and unstable walking. On interrogation he had retrograde amnesia of the procedure. When the patient returned to the nursing floor after the puncture, he noticed blurred vision and complex formed visual hallucinations.

Further examinations were delayed due to misinterpretation of the symptoms. However, a clinical neurological examination seven days after the puncture revealed symmetrical moderate weakness and hyperreflexia in the lower limbs, extensor plantar reflexes and abolished abdominal reflexes. No spasticity, brain nerve malfunction or ataxia were found. Urine retention developed and urodynamic evaluation showed an elevated maximum bladder capacity and underactive detrusor function. Intermittent catheterization of the bladder was started. Electroencephalography (EEG) showed significant pathological findings with slow theta activity in both hemispheres, mainly in the temporal and occipital regions. Computed tomography (CT) of the cerebrum did not reveal pathological changes. It was then concluded that the patient had a brain stem injury, probably caused by an air embolism to the basilar artery ("top of the basilar syndrome") [6].

Eight days after the procedure the patient was recompressed in a hyperbaric oxygen chamber at 50 m for almost 5 h according to standard naval procedures [7] reducing the bubble size to approximately 1/6. The neurological symptoms gradually disappeared prior to, as well as after, the hyperbaric treatment. The intermittent catheterization was discontinued after six months when residual urine volumes were appoximately 50 ml on repeated measurements. The patient subsequently demonstrated a normal uroflowmetry.

Cytological examination of the fine needle aspirate showed an epidermoid carcinoma. The lower lobe of the right lung was resected five weeks after the puncture. Histopathological examination confirmed the cytological diagnosis. Six months after the puncture the patient is back at work as a full-time electrician and is completely neurologically recovered.

Discussion

Air embolism complicating percutaneous lung puncture is a rare event when using needles with diameters less than 0.9 mm. DAHLGREN and NORDENSTRØM [1] initially reported one possible case of air embolism in 574 punctures with needles from 19–20 gauge (1.1–0.9 mm). Reviewing the literature, SINNER [2] found two possible cases of air embolism in 2,726 patients using needles from 20–16.5 gauge (0.9–1.6 mm), whilst NORDENSTRÖM [8] reported the incidence of these complications to be 0.5 in 1,000. WESTCOTT [3] reported one fatal case using an 18 gauge (1.3 mm) needle. Risk factors for air embolism are cough, emphysema, a non co-operative patient, a large diameter needle and a centrally located tumour. In addition, bleeding into a bronchus after a puncture may act as an irritant and give rise to coughing.

The presumed mechanism causing air embolism is communication between a bronchus and a pulmonary vein. This might occur during fine needle aspiration, especially when a positive pressure difference between the airways and the pulmonary veins is created during coughing, Valsalva manoeuvre or deep inspiration. The risk of atmospheric-to-pulmonary venous air embolism can be reduced by occluding the needle at all times unless the respiration is suspended. The patient's cough reflexes should be depressed before the puncture by means of an antitussive agent.

The introduction of ultrathin needles in percutaneous lung puncture has reduced the risk of serious complications [9]. However, our report illustrates that air embolism may occur even when ultrathin needles are used. CIANCI *et al.* [10] reported a case using a 22 gauge needle (0.7 mm). Air embolism may be difficult to diagnose when presented with vague symptoms [11]. It should always be considered in patients who, after lung punctures, develop chest pain, dizziness, nausea, visual disturbances, seizures or altered consciousness. Neurological symptoms appearing, even hours and days after a percutaneous lung puncture, should alert the physician to consider the possibility of air embolism. Behavioural abnormalities including defects in memory, amnesia, apathy and lack of attention [6] may explain the late detection of air embolism in our case.

Immediate treatment includes 100% oxygen supply placing the patient with the head down [12]. Hyperbaric oxygen therapy is a well-established treatment to reduce bubble size, restore circulation, facilitate nitrogen resorption and oxygenate hypoxic tissue to maintain viability. Transfer to an oxygen chamber should be effected as soon as possible and not delayed for confirmative diagnosis as recovery is dependent on the speed of recompression. Air bubbles have been demonstrated in pial vessels up to 48 h after experimental cerebral air embolism in dogs [13]. Hyperbaric therapy may be useful even if this therapy is delayed by more than 24 h after the procedure producing the air embolism [14, 15].

In the present case the brain stem lesion was diagnosed seven days after the puncture. The clinical history, neurological signs and EEG showed changes, probably due to an air embolism to the basilar artery. This is compatible with the urodynamic findings. A critical neural circuit for normal voiding detrusor contraction comprises afferent nerves to the rostral pons, synapses at that point and efferent nerves that run back down the cord to sacral segments [16]. Minor vascular lesions in the cerebrum are often not discovered when examined by computed tomography [17]. Such cases will usually show complete clinical recovery. In our case the benefit of the hyperbaric oxygen treatment performed eight days after the episode was uncertain.

Physicians performing fine needle aspiration of the lung should acquaint themselves with signs and symptoms indicating cerebral air embolism, and should locate the nearest hyperbaric oxygen chamber in order to obtain immediate access if needed.

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Embolie gazeuse cérébrale après ponction transthoracique au moyen d'une aiguille de 0.6 mm (23 gauge). E. Omenaas, O. Moerkve, L. Thomassen, L. Daehlin, J. Larsen, S. Eidsvik, A. Gulsvik.

RÉSUMÉ: Un homme de 54 ans ressent de la faiblesse dans les membres, une marche instable et ultérieurement de la rétention urinaire, après ponction transthoracique à l'aiguille fine d'une tumeur du lobe inférieur droit. L'examen neurologique clinique et électroencéphalographique montre des signes de lésion du tronc cérébral, probablement par suite d'une embolie gazeuse de l'artère basilaire. Les symptômes subjectifs et objectifs ont disparu graduellement, avant et après traitement à l'oxygène hyperbare. Nous pensons que cette observation est la première rapportant une embolie gazeuse après ponction transthoracique au moyen d'une aiguille 23 gauge dans la littérature médicale. *Eur Respir J., 1989, 2, 908–910.*