Cerebral air embolism.

O Iwami, J Kawamura, S Hashimoto, M Nakamura and T Suenaga

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Cerebral Air Embolism

Cerebral air embolism often is produced either accidentally or iatrogenically [1]. It rarely is caused by lung cancer.

Case Report

A 62-year-old man had a lobectomy and then chemotherapy for a squamous cell carcinoma of the right lung. Two years later he had radiation therapy for lymphadenopathy of the right hilus. Four months after the radiation therapy, he suddenly became comatose with generalized convulsions. On admission at this time, he was unresponsive to painful stimuli and had occasional myoclonic movements in the right upper extremity and hyperactive muscle stretch reflexes in the left lower extremity. Hemothorax was confirmed by thoracocentesis. Bronchoscopy showed a small amount of bleeding at the right B3 segment and a portion of necrotic tumor at the opening of a bronchiolar fistula.

CT 3 hr after the onset of coma and convulsions showed multiple low-density lesions of various sizes, which were localized mainly in the subcortical areas of both cerebral hemispheres (Fig. 1A). The attenuation of the lesions ranged from -249 to -351 H. The periventricular low-density areas had been noted on a previous CT scan performed before the patient’s lobectomy and was unrelated to chemotherapy. CT 1 day after the onset of coma and convulsions showed scattered air bubbles and obscured sulcal markings, suggestive of brain edema. No lesions were seen on contrast-enhanced CT (Fig. 1B). Unenhanced CT on the fourth hospital day showed a residual air bubble and increased edema with narrowed cortical sulcal markings (Fig. 1C). The patient did not regain consciousness and died 6 days after the onset of coma and convulsions. No autopsy was performed.

Discussion

In this patient, the air could have entered the systemic circulation through the fistula between bronchus, tumor cavity, and pulmonary veins. CT scans seemed to show the sequential events. At first, multiple air bubbles were present in subcortical areas. The next day, the bubbles were smaller and the cortical sulcal width had decreased. On the subsequent CT scan, the bubbles were fewer and smaller, and decreased sulcal markings indicated increased compression of the cortex.

Experimentally, air injected into the carotid system is distributed initially throughout the territory of the system [2]. It then might be propelled by hydrostatic forces to the watershed or distal zones of the cerebral vasculature [3]. Three to 24 hours later, pathologic changes similar to those caused by ischemia produced by unilateral carotid ligation can appear [4]. The size of the air embolus also may affect the distribution and extent of the brain lesions. Solid emboli up to 15 to 17 μm in diameter produced lesions usually confined to the white matter of the brain, and larger ones involved the gray matter alone or both the gray and white matter [5]. CT is valuable to show the air in the brain vasculature at the early stage of cerebral air embolism [6].

REFERENCES


Fig. 1.—Cerebral air embolism caused by lung cancer.

A, CT scan obtained 3 hr after onset of coma and convulsions shows multiple air bubbles, mainly in subcortical arterial boundary zones of both cerebral hemispheres.

B, Contrast-enhanced CT scan obtained 2 days after onset shows multiple air bubbles and reduced cortical sulcal markings, indicative of cerebral edema. No abnormally enhanced lesions were seen.

C, Unenhanced CT scan obtained 4 days after onset shows edema involving subcortical white matter more widely and loss of sulcal markings. Air bubble is still visible in right frontal subcortex.