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Case Report

Cerebral Air Embolism during Open Heart Surgery with Cardiopulmonary Bypass
- A Case Report -

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Cerebral air embolism is an unusual event that is mainly an iatrogenic cause, such as open heart surgery. We present a case of cerebral air embolism in a patient undergoing ASD patch repair with cardiopulmonary bypass. He had a status epilepticus, loss of consciousness and marked left limb weakness immediately after the operation. Diffusion-weighted MRI with angiography showed acute infarction in right entire hemisphere with patent internal carotid and intracranial arteries, and glucose PET brain scan showed severe decreased uptakes in right hemisphere. He recovered markedly with mild motor impairment of left upper and lower limbs in the 6 months after onset.

Key Words Cerebral air embolism, Cardiopulmonary bypass

INTRODUCTION

Cerebral air embolism (CAE) occurs very rarely, but is mostly caused by iatrogenic problems that arise in the process of open heart surgeries, hemodialysis, angiography, pulmonary barotrauma and venous catheterization. Even a tinny gas bubble may be fatal to brain tissue vulnerable to hypoxia if it flows into the arterial blood vessel,[1-3] wherefore heart surgeries with cardiopulmonary bypass (CPB) have a higher possibility to cause CAE.

In practice, however, there are very few cases where CAE occurs in the process of a heart surgery with CPB. Stoney et al.[4] reported that the incidence of CAE stood at approximately 0.1% and also tended downward as CPB had been advanced. Nevertheless, there is always a chance that the heart surgery with CPB will cause CAE, and that neurologic disorders occur as a result of it. But in reality, CAE tends to be overlooked in many cases.[1,4]

This paper is to report a case of CAE that occurred after an open heart surgery with CPB.

CASE REPORT

A 26-year-old male was hospitalized with the chief complaint of a chest pain and palpitation. The patient underwent reconstructive surgeries for patent ductus arteriosus and patent foramen ovale at the age of 8 months and 9 years respectively. On electrocardiography (EKG) an atrial septal defect (ASD), trisupid regurgitation and severe pulmonary insufficiency were detected, whereupon ASD repair was performed under a general anesthesia. CPB was adhibited for about 1 hour. The right atrium was exposed to air in the process of being dissected, and the operator reported that there was a strong possibility that air might flow into the

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right atrium.

Immediately after undergoing the operation, the patient had a seizure, shaking his upper limbs and face. The blood pressure was 163/98 mmHg, and the heart rate and the respiratory rate were 160/min and 16/min respectively. In arterial blood the potential of hydrogen (pH) was 7.235, and the partial pressure of carbon dioxide (pCO2) and the partial pressure of oxygen (pO2) were 30.7 mmHg and 81.9 mmHg respectively. Even considering the fact that the patient was being under postanesthetic sedation, stimuli got no response at all. Pupils were different in size from each other (right: 3 mm, left: 1 mm). Since status epilepticus could not be controlled with 10 mg of Lorazepam, Midazolam and Valproic acid were intravenously injected at loading doses. Nonetheless, the patient continued having a seizure, shaking his legs and twisting his head to the left. The symptoms were eliminated 16 hours later. Computed tomography (CT) was performed on the brain 20 hours after the onset of symptoms, whereon a swelling, sulcal effacement and the manifestation of an ischemic brain injury were detected in the right hemisphere (Fig. 1). After endotracheal intubation, 100% oxygen was supplied through a mechanical ventilator, during which the patient was in a supine position with proper blood pressure.

Even two days after the onset of symptoms, the left upper limb and lower limb did not show avoidance response to stimuli. A magnetic resonance imaging (MRI) scan was performed on the brain, whereon the manifestation of cerebral infarction was detected in the whole right hemisphere. On magnetic resonance angiography (MRA) there were no findings suspicious for the occlusions of the right middle cerebral artery, the internal carotid artery and the posterior cerebral artery (Fig. 2). The patient had been alert 8 days after the

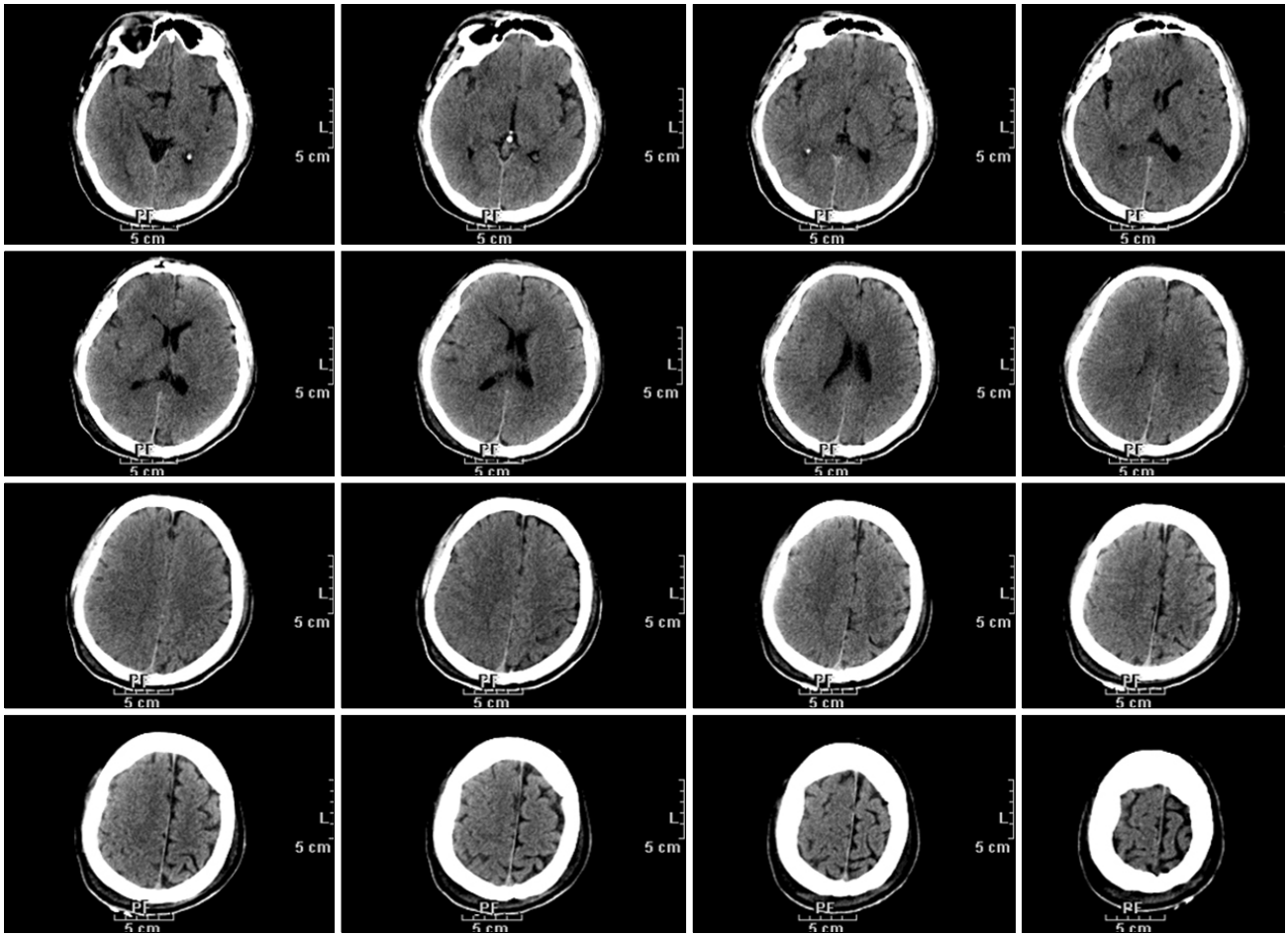

Fig. 1. Brain CT scan showed the swelling of right hemisphere with effacement sulcal space and suspected ischemic damage.

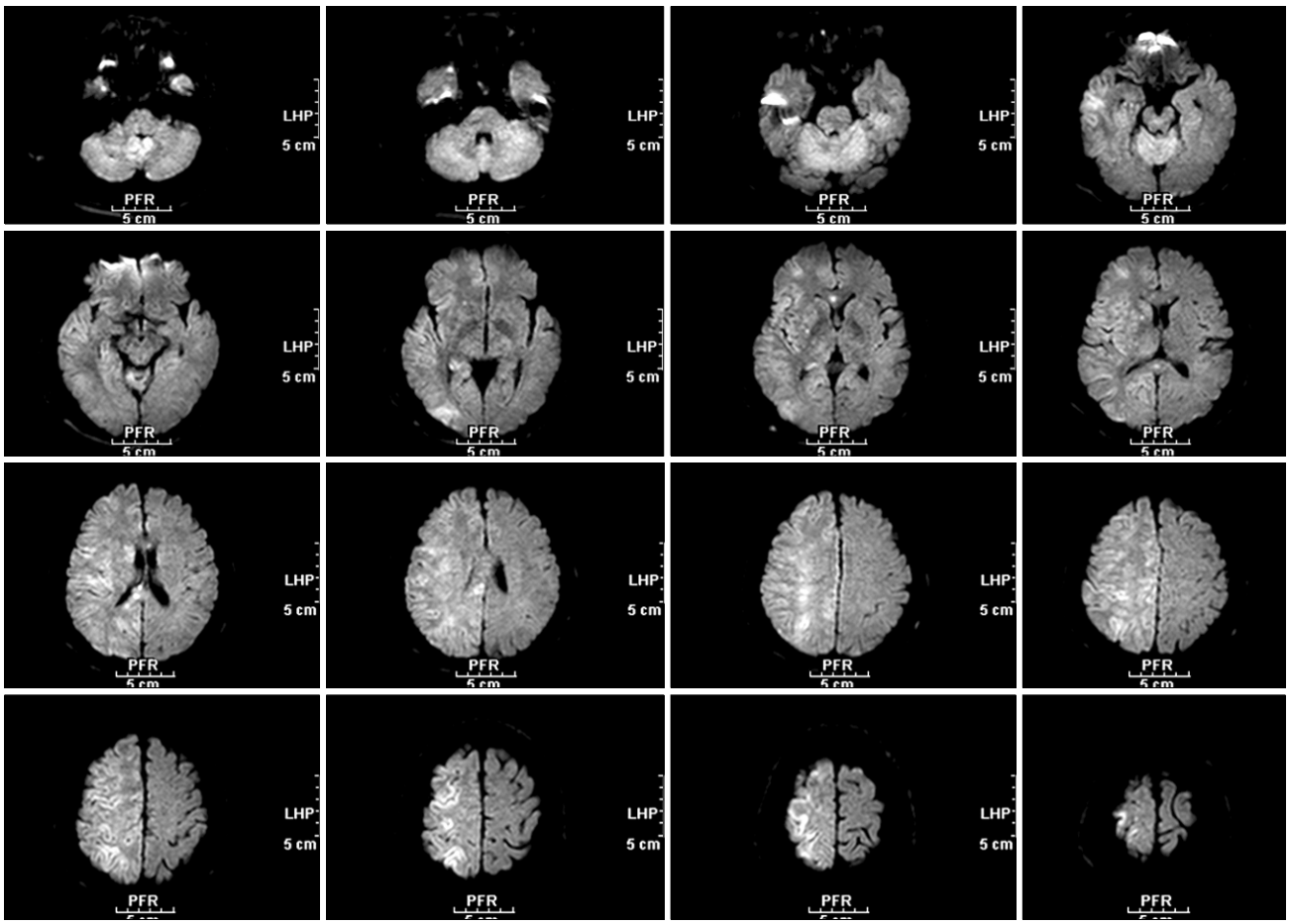

Fig. 2. Brain MRI diffusion-weighted image revealed the acute cerebral infarction in the entire right hemisphere.

onset of symptoms, whose vital signs and cardiopulmonary function had been stable. Twenty days after the onset of symptoms, the patient was transferred to the general ward, when he looked as though he had no problems with cognition and speech. On the mini-mental state examination (MMSE) the patient got 25 points, especially low grade in the category of attention and calculation. Also, he showed the symptoms of left hemineglect. On the manual muscle test (MMT), the right upper limb and lower limb were within the normal range. The left upper limb was graded 1 out of 5. In the case of the left ankle, dorsal and plantar flexions were graded 1 and the other regions were graded 3. On the Fugl-Meyer assessment, upper limbs and lower ones scored 2 and 21 respectively, 23 in all. The patient could walk 10 meters indoors by himself under the watchful eye of his protector, but showed abnormal gaits such as foot dragging and hip hiking on left lower limb. On functional independence measure (FIM), the patient scored 87 and needed help in relation to self-care and mobility. On positron emission tomography (PET) performed on the brain 23 days after the onset of symptoms, severe metabolic decrease was observed in the right hemisphere, especially in the right middle cerebral artery area. Also in the left cerebellum, somewhat metabolic decrease was observed (Fig. 3).

After 7-week rehabilitation, left hemineglect was improved. The muscle strength of left upper and lower limbs were generally improved as they were graded 3 and 4 respectively on MMT. On Fugl-Meyer assessment, upper limbs and lower limbs scored 44 and 29 respectively, 73 in all, especially, the motor function of upper limbs was conspicuously improved. The patient could walk and go up and down stairs by himself. In addition, he could walk with near-normal gait pattern. The MMSE score and the FIM score were improved to 28 and 126 respectively, which mean that the patient can lead his daily life by himself.

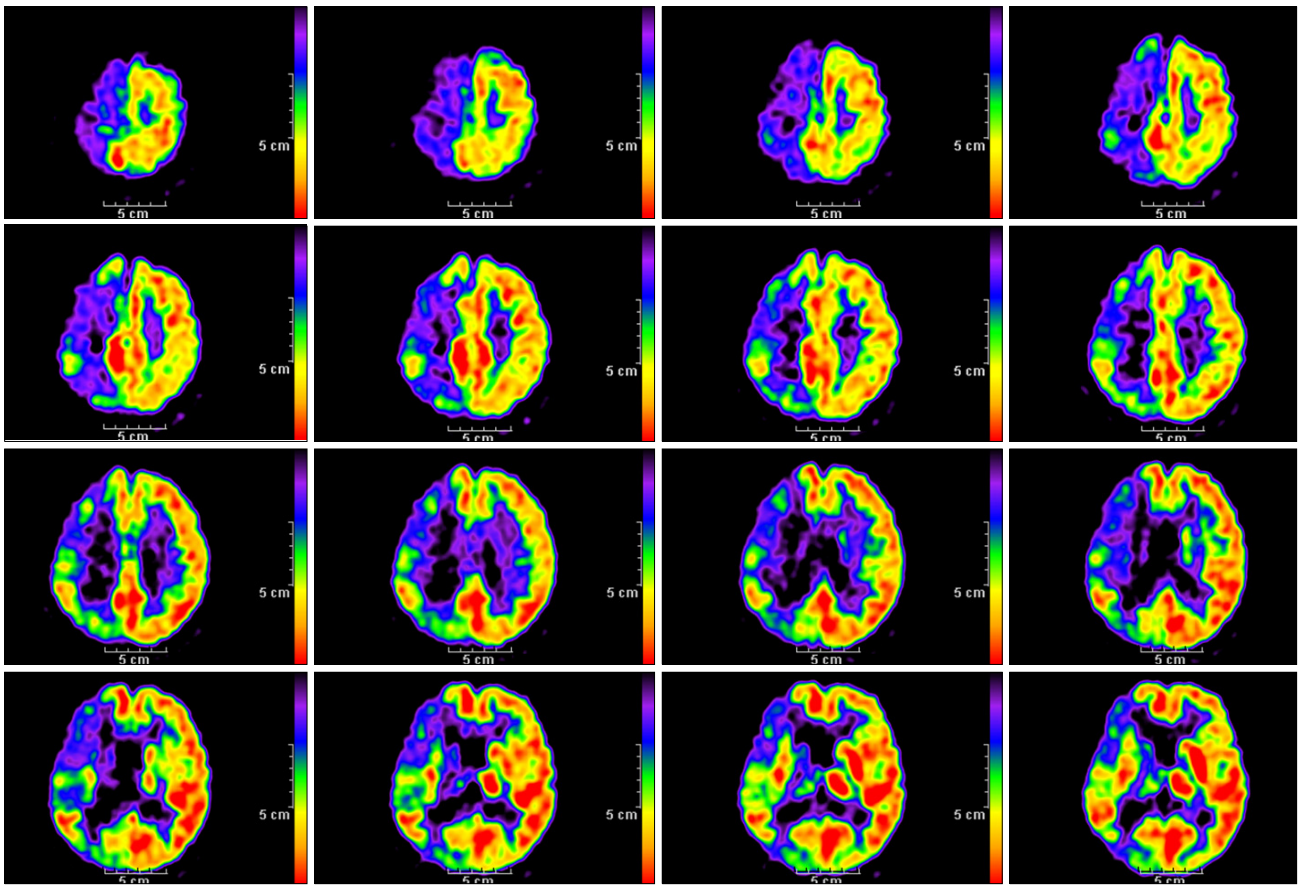

Fig. 3. 18F-FDG Brain PET showed decreased FDG uptake in entire right hemisphere, especially in right MCA territory.

After getting out of the hospital, he leaded his daily life normally and received fine motor training as a part of out-patient treatments. On Fugl-Meyer assessment performed 6 months after the onset of symptoms, upper limbs and lower limbs scored 52 and 31 respectively, 83 in all, and thus motor function was more improved. *Inter alia*, the motor function of upper limbs was remarkably recovered. The MMSE score was improved to 30, and left hemineglect was eliminated. At the initial stage it was impossible to measure grip power and to perform the box and block test, but 6 months later grip power reached 37% of the average. Moreover, the patient could achieve 54% of the box and block test.

DISSCUSION

Pathologically, cerebral air embolism (CAE) is caused by the distal hypoperfusion in the blood vessel occluded by the gas bubble and the vessel wall's inflammatory response to the gas bubble.[3] Since approximately 20% of cardiac output is supplied to the brain, a gas bubble has the high possibility to be formed in the cerebral blood flow. In case a gas bubble touches the endothelium of the blood-brain barrier, polymorphonuclear leukocytes are activated and stick to the injured region and as a result a cerebral edema, an inflammation and various neurologic symptoms may occur.[5]

Ordinarily, CAE is diagnosed on the authority of clinical history. If neurologic symptoms such as motor paralysis, seizure and mental deterioration occur after an invasive procedure, a check should be made on CAE without delay.[3] Griese et al.[6] who conducted a retrospective study on patients who contracted cerebral cortical infarction after undergoing CPB, reported that there was a strong possibility that CAE might cause the infarction if a cerebral cortical lesion in a hemisphere was observed on the brain diffusion MRI scan and the corresponding hemiplegia was observed in the opposite hemisphere and seizure occurred early after the operation. According to previous case reports, it is difficult

to detect the gas bubble even on CT and MRI, and besides, it is absorbed within hours after being formed.[7] Furthermore, tiny bubbles, formed when a big bubble burst, may occlude cortical arteries with narrow bores, which mostly occurs at the boundary between the middle cerebral artery and the anterior cerebral artery.[6,8] Also in this case, the gas bubble could not be detected on CT, but CAE was diagnosed in the light of an operator's report that air flew into the right atrium in the process of the operation, neurologic symptoms that occurred after the high-risk procedure, infarction covering the right hemisphere cortex, and early seizure.

In the case of CAE, hyperbaric oxygen therapy is adhibited as the primary treatment.[1] The problem is that there are few medical institutions equipped with such facilities in Korea. Also, the therapy may not be adhibited to patients in the acute phase inasmuch as their vital signs are not stable. Also in this case, vital signs were unstable with status epilepticus and thus conservative treatments were performed exclusive to the exclusion of hyperbaric oxygen therapy, which were essential measures to minimize secondary injuries. Conservative treatments are to perform endotracheal intubation and to supply 100% oxygen through a mechanical ventilator, which are known to treat hypoxia and to remove the gas bubble by forming diffusion gradient. In this case, the patient should be in supine position in order that buoyancy may not make the gas bubble worsen the cerebral edema. In addition, it is important to maintain proper blood pressure so that promote the circulation of the blood.[5]

Few studies have been conducted on the prognosis of CAE. Hyperbaric oxygen therapy, *i.e.,* the primary treatment for CAE, is closely related to the prognosis of patients, but there has been still controversy as to the appropriate time. Some maintain that it should be adhibited within 3 hours after the onset of CAE, some assert 48 hours.[5] A retrospective analysis, made of patients who contracted cerebral infarction after undergoing open heart surgeries, *i.e.,* a high-risk factor to CAE, showed that patients who underwent hyperbaric oxygen therapy showed more satisfactory prognosis compared to ones who did not so.[9] Benson et al.[7] observed the progress of 19 patients with iatrogenic CAE for 2 months, and reported that neurologic symptoms were completely eliminated in 42% of patients; in 32%, such symptoms were partially relieved, and the other 26% expired. Trytko and Bennett[10] evaluated 26 patients with CAE who underwent hyperbaric oxygen therapy right before they got out of the hospital, and reported that 4 showed slight neurological sequelae and only 2 showed serious sequelae. In result, most patients with CAE showed favorable outcome neurologically. Jeon et al.[8] reported that the larger infarction became on the brain diffusion MRI scan, the worse prognosis got. Most of studies on the prognosis of patients with CAE have been focused on whether the patients underwent hyperbaric oxygen therapy, and besides, such studies have been conducted only on small groups and also have not evaluated neurologic injuries.

In this case, infarction was observed in the right hemisphere on the brain MRI scan. On brain PET, hypofunction was observed in the right hemisphere, especially in middle cerebral arteries. In the early stage, the left hemiplegia occurred but was almost recovered 2 and 6 months later to the exclusion of a slight disorder in the left upper limb. It is problematic to compare this case to previous cases, as hyperbaric oxygen therapy was not adhibited to this case. But obviously, findings suspicious for CAE were detected early on and as a result favorable prognosis could be derived.

It is rare that CAE occurs, but once it occurs, it gives rise to serious neurological disorders. Thus in case neurologic symptoms occur after a high-risk operation, it is advisable to suspect CAE and to perform treatments with diagnosis. As shown in this case, appropriate treatment may derive noteworthy recovery, hence the need for early treatments and rehabilitation.

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