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Case Report

Cerebral Air Embolism After Air-Powder Abrasive Dental Treatment: A Case Report

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HIGHLIGHTS

- A patient who developed cerebral air embolism (CAE) after air powder abrasive treatment.
- This is the rare case of CAE.
- Delayed hyperbaric oxygen therapy aided neurological symptom recovery in our case.

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ABSTRACT

Cerebral air embolism (CAE) occurs in various clinical situations such as surgery, angiography, and hemodialysis; most are iatrogenic. Here we report the case of a 57-year-old man who developed CAE immediately after air-powder abrasive treatment, which is commonly used in dentistry. The patient underwent air-powder abrasive treatment for peri-implantitis, and immediately after the treatment, cardiac arrest occurred and cardiopulmonary resuscitation was performed. After resuscitation, brain computed tomography performed in the emergency room showed scattered dark density presumed to be air. The day after admission, the patient showed right hemiplegia and a multifocal cerebral infarction was observed on brain magnetic resonance imaging. Therefore, CAE was strongly suspected. After hyperbaric oxygen treatment (HBOT), which started 4 days after the incident, the patient regained consciousness and showed improvement in cognitive impairment, and only grade 4 muscle weakness was observed in the right lower extremity on the manual muscle test. This case highlights the importance of considering CAE as a possible cause of neurological symptoms occurring during clinical procedures involving air, and adds to the accumulation of evidence of therapeutic effects of delayed HBOT.

Keywords: Dental Air Abrasion; Air Embolism; Iatrogenic Disease; Cerebral Ischemia; Hyperbaric Oxygenation

INTRODUCTION

Air-powder abrasive treatment in dentistry is mainly performed to treat peri-implantitis [1]. It treats peri-implantitis by subgingivally spraying compressed air along with abrasive powder to remove biofilms that cause inflammation [1]. However, side effects such as pneumomediastinum and pneumothorax have been reported due to pressurized air flowing into the gingival defect during treatment [2].

Air embolism, caused by the inflow of air into the blood vessels, rarely occurs but has a high mortality rate [3]. Air introduced into the blood vessels can cause ischemic damage to the end organs by blocking the blood circulation [3]. In particular, air introduced into the

Conflict of Interest
The authors have no potential conflicts of interest to disclose.

Author Contributions
Conceptualization: Seong ST, Kim GC; Data curation: Seong ST, Lee JH; Formal analysis: Seong ST, Sim YJ; Writing - original draft: Seong ST, Kim GC; Writing - review & editing: Seong ST, Kim GC, Jeong HJ

cerebrovascular circulation causes neurological symptoms similar to those of acute cerebral infarction [3,4]. However, unlike acute cerebral infarction caused by thrombosis, cerebral air embolism (CAE) requires acute hyperbaric oxygen treatment (HBOT) [4,5]. Therefore, a fast and accurate diagnosis is required.

CAE can occur in various clinical situations such as open surgery, angiography, and hemodialysis, but there are no reports of it occurring due to air-powder abrasive treatment, which is commonly used during dental treatment [4]. Here we report a case of CAE after air-powder abrasive dental treatment and the effect of delayed HBOT.

CASE DESCRIPTION

A 57-year-old man with no remarkable medical history visited a private dental clinic for pain in the mandibular anterior region. The patient underwent subgingival curettage from the 1st premolar to the 2nd molar of the right maxilla and mandible under local anesthesia. Twenty minutes after curettage, air-powder abrasive treatment was applied to the right lower second molar for 5 minutes to treat the peri-implantitis. Immediately after the procedure, the patient complained of dyspnea with face and neck edema, and cardiac arrest occurred 1 minute later. Cardiopulmonary resuscitation was performed immediately and after 2 cycles, the patient's spontaneous breathing and pulse returned, but he remained unconscious and was transferred to a tertiary hospital.

At the time of emergency room admission, the patient was in a semi-comatose state, and the Glasgow Coma Scale score was 6 (eye opening, 2; motor response, 2; and verbal response, 2). His initial vital signs were as follows: blood pressure, 100/70 mmHg; pulse, 138 beats/min; respiration rate, 24 breaths/min; body temperature, 35.4°C; oxygen saturation, 60%. Intubation and ventilator care were immediately performed. Electrocardiography showed ST elevation in V1–V4 leads, while no significant stenosis or specific findings in the spasm test were noted on coronary angiography. Neck and chest computed tomography (CT) showed pneumomediastinum with extensive subcutaneous emphysema over the face and neck. Brain CT (**Fig. 1A and B**) showed scattered dark density on both frontoparietal sulci at 50 minutes after the onset of the cardiac arrest. After 4 hours, a follow-up brain CT (**Fig. 1C and D**) showed resolution of the dark density, but brain edema was observed. Initial brain magnetic resonance image (MRI) (**Fig. 2A and B**) taken 4 hours after the onset of the cardiac arrest showed no definite diffusion restriction in the diffusion-weighted image. However, on the day after admission, the patient was evaluated as grade 4 for right upper-extremity strength and grade 2 for right lower-extremity strength on a manual muscle test (MMT). And follow-up brain MRI (**Fig. 2C and D**) was performed and showed multifocal cerebral infarction with high signal intensity on diffusion-weighted image in the left temporal/parietal lobe and the bilateral frontal/occipital lobes. CAE was strongly suspected due to neurological symptoms occurring after air-powder abrasive treatment, scattered dark density presumed to be air on the brain CT, and multifocal cerebral infarction findings on brain MRI; however, the hospital was lacking an HBOT center.

After 4 days of acute treatment in the intensive care unit, the patient recovered consciousness to a drowsy mentality, with a Glasgow Coma Scale score of 13 (eye opening: 3, motor response: 6, and verbal response: 4), and the right lower-extremity strength improved to grade 3 on MMT. Subsequently, the patient was transferred to another hospital for HBOT.

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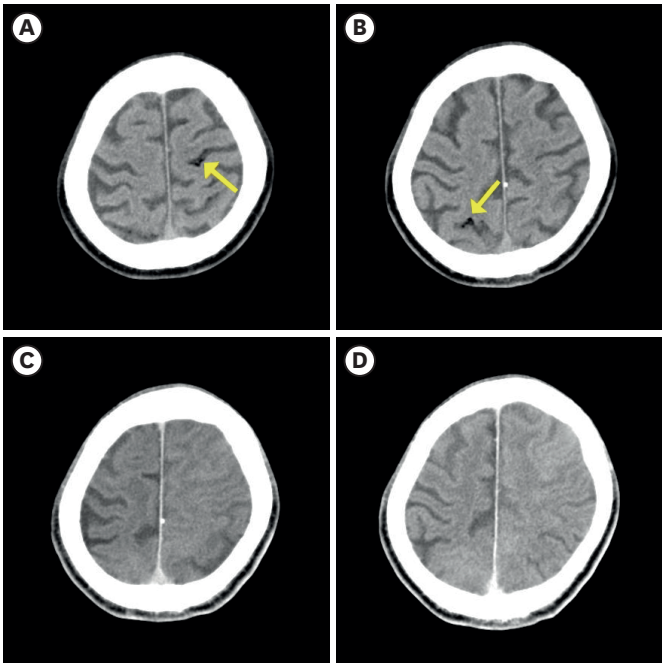

Fig. 1. Initial and follow-up brain CT image.
Initial non-contrast-enhanced brain CT (A and B) performed 50 minutes after cardiac arrest onset showing scattered dark densities (yellow arrows) at both frontoparietal sulci. Follow-up non-contrast-enhanced brain CT (C and D) performed 5 hours after cardiac arrest onset showing non-visualization of the sulcal dark densities at both frontoparietal lobes but effacement of the cerebral sulci.
CT, computed tomography.

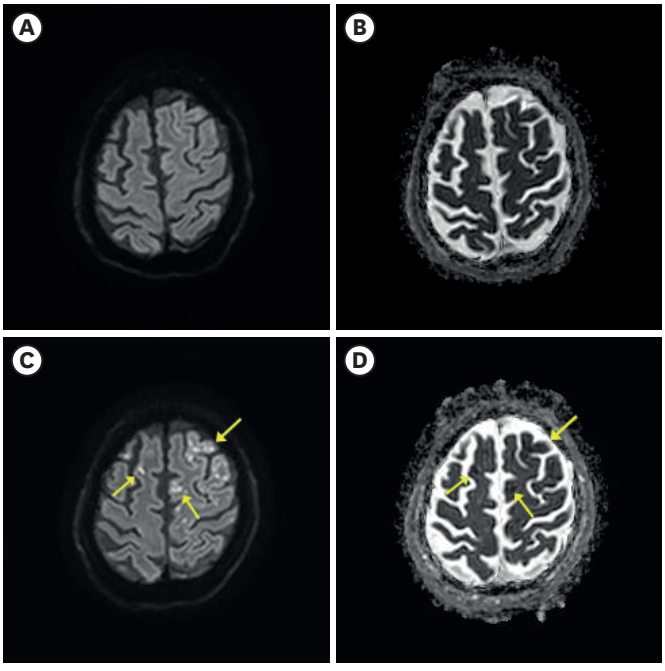

Fig. 2. Initial and follow-up brain MRI.
Initial brain MRI (A and B) taken 4 hours after cardiac arrest onset showing no evidence of definite acute cerebral infarct. Follow-up diffusion-weighted MRI with a B value of 1,000 (C) and an apparent diffusion coefficient map (D) performed 22 hours after cardiac arrest onset showing an acute multifocal cerebral infarction (yellow arrows) at both cerebral hemispheres.
MRI, magnetic resonance image.

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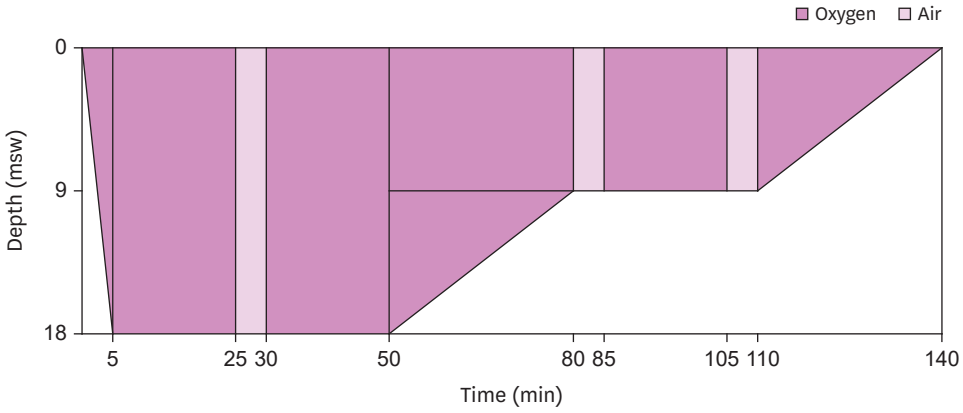

Fig. 3. United States Navy Treatment Tables 5.
The United States Navy Treatment Tables 5 are used for cerebral arterial gas embolization when prolonged treatment is required. It consists of a compression phase that lasts about 5 minutes, reaching a depth of 18 msw under 100% oxygen, followed by 2 oxygen cycles lasting 20 minutes each, with short air intervals. Afterward, the patient is decompressed to about 9 msw and exposed to an oxygen cycle lasting 20 minutes before slowly returning to surface pressure.
msw, meter sea water.

Four days after the onset of cardiac arrest, the patient underwent HBOT seven times over 11 days according to the United States Navy Treatment Table 5 program (**Fig. 3**). The day after the end of HBOT, the patient's consciousness recovered to alert mentality, with a Glasgow Coma Scale score of 15, and he obtained a score of 27 points on the Mini-Mental State Examination. In addition, the right upper extremity muscle strength improved to grade 5 and right lower extremity muscle strength improved to grade 4 on the MMT.

This study was reviewed and approved by the Institutional Review Board (IRB) of Kosin University Gospel Hospital (IRB No. 2023-02-026). Informed consent was waived by the board.

DISCUSSION

CAE rarely occurs and is mostly iatrogenic [4]. Even if the blood vessels are exposed to the atmosphere, air does not flow into the veins because the venous pressure is higher than the atmospheric pressure, and a small amount of air that flows in is absorbed by the pulmonary filter [3,6]. However, when the patient is in an upright position, the inflow of air can cause cerebral venous air embolism by retrogradely ascending the venous circulation [3]. Also, if more than 0.35 mL/kg/min of air flows into the vein, it cannot be sufficiently absorbed by the pulmonary filter and may flow into the artery, causing arterial embolism [6]. In addition, if an atrial septal defect, foramen ovale, and pulmonary arteriovenous malformation are present, air passing through the right-to-left shunt can cause an arterial embolism [3,4,6].

In our case, it is believed that CAE is induced by a venous source, given that serpiginous air densities are visible on the initial brain CT (**Fig. 1A and B**), which is compatible with venous CAE [7]. Additionally, a pulmonary arteriovenous malformation was suspected on transesophageal echocardiography and shunt test performed 20 days after symptom onset. However, chest CT showed no suspicious findings of pulmonary arteriovenous malformation, and no additional tests, such as pulmonary angiography, were performed. Therefore, in this case, arterial air embolism is also possible as the air passes from the venous

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to the arterial circulation, either via a microscopic pulmonary arteriovenous malformation or by directly crossing the pulmonary capillary bed.

Subgingival curettage is a treatment for periodontal disease and in which inflammatory soft tissue is removed by scraping the tissue sidewall of the periodontal pocket using a curette [8]. After subgingival curettage, bleeding occurs in the area where the epithelial wall is removed, and epithelialization occurs 2–7 days later [8]. In this case, pressurized air was injected while the subgingival tissue was damaged after 20 minutes of subgingival curettage, raising the possibility of air inflow into the tissue and vessel and increasing the inflow amount.

HBOT should be performed as soon as possible when CAE accompanies cardiopulmonary damage, neurological defects, or end organ damage [4,9]. HBOT improves tissue ischemia by supplying 100% oxygen in 2–3 atmosphere in the chamber, reducing the size of air bubbles introduced into blood vessels and increasing oxygen delivery [4,5]. Starting HBOT within 6 h of onset reportedly has the best therapeutic effect, but cases of partial or complete recovery have also been reported in cases of delayed HBOT [4,10,11]. In this case, HBOT was performed 4 days after symptom onset due to delayed diagnosis and the absence of a HBOT facility. However, despite a 4-day delay, after HBOT, the patient's neurological symptoms such as level of consciousness and recovery of muscle strength, clearly improved. It is believed that the effect of HBOT in CAE is due to the existence of cellular and biochemical benefits in neurofunctional recovery including neurogenesis as well as reduction of intravascular bubble size and relief of local hypoxia [12].

CAE should be diagnosed early for rapid treatment. However, making a radiological diagnosis is difficult because air is rapidly absorbed from the circulatory system [11]. Therefore, a clinical history to suspect CAE is important. If cardiorespiratory or neurological symptoms occur in the patient during dental treatment, especially when using an air powder abrasive device, the possibility of CAE as the cause of cerebral infarction should be considered and HBOT performed as soon as possible if other diagnoses are excluded. In addition, based on the literature and this case, improvement in neurological symptoms can be expected even if HBOT is delayed for several days [10,11]. Therefore, HBOT should be performed as soon as possible if not immediately after symptom onset. In addition, to prevent CAE, it is necessary to consider the risk factors of air inflow into blood vessels such as tissue damage and bleeding during treatment, and use an air powder abrasive device appropriately.

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