Nonfatal Cerebral Air Embolism After Dental Surgery

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After removal of four impacted third molars under general anesthesia, our patient developed subcutaneous emphysema, pneumothorax, pneumopericardium, and pneumomediastinum. Soon thereafter, coma with generalized epileptic status ensued. A cerebral magnetic resonance and single photon emission computed tomography showed hypoperfusion of the right thalamus and parietal, temporal, and frontal cortices. The likely mechanism was injection of air by the high-speed dental drill through the soft tissue adjacent to the roots of the lower molars. We were unable to find any previous report of systemic air embolism after oral surgery.
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Pneumomediastinum, pneumothorax, venous air embolism, and even death, have been reported as complications after dental surgery; most of these cases were associated with the use of dental equipment that directed pressurized air or water into gingival defects produced by dental procedures.[1–3] Systemic air embolism has been reported as a complication of various surgical operations and invasive maneuvers and is, more often than not, an iatrogenic complication.[4,5] Coma ensuing from cerebral air embolism after dental surgery has not been described.

CASE REPORT

A 21-yr-old male ASA I patient, was scheduled for the removal of four impacted third molars under general anesthesia. His preoperative chemical and hematological profiles, electrocardiogram and chest radiograph were normal. General anesthesia was induced with propofol and fentanyl; neuromuscular block was achieved and maintained with vecuronium. After nasotracheal intubation, the patient's lungs were ventilated with an air–nitrous oxide mixture. Anesthesia was maintained with propofol and fentanyl. Intraoperative monitoring included a 3-lead electrocardiogram, pulse oximetry, capnography and noninvasive arterial blood pressure. The surgical procedure was uneventful. Spontaneous ventilation was restored 5 min before the end of the procedure and the trachea was extubated when the patient responded to verbal commands, during a moderate fit of coughing; cognitive recovery was complete and satisfactory pain control. After 45 minutes the patient became progressively agitated. Physical examination showed moderate anterior neck and lower facial swelling with crepitus. His arterial blood pressure was normal as was his blood gas analysis. A chest radiograph showed bilateral small pneumothorax, pneumopericardium, and pneumomediastinum. A cerebral computed tomography (CT) scan showed no edema or hematoma and magnetic resonance imaging did not show any sign of ischemic lesions. One hour later, because of his rapid loss of consciousness the trachea was intubated and he was transferred to the neurosurgical intensive care Unit. Generalized tonic-clonic convulsions were treated with a lorazepam infusion (16 mg/24 h) and the electroencephalographic recording showed generalized epileptic status. A CT of the thorax confirmed the presence of a small pneumothorax, pneumopericardium, and pneumomediastinum. Air drainage was not recommended because of the small air volume. Fiberoptic bronchoscopy revealed no lesions of the trachea and mechanical ventilation was continued without increasing the size of the pneumothorax that recovered in 3 days.

Twenty-four hours later, a magnetic resonance imaging showed bilateral hyperintensity of cortical and subcortical temporomesial areas, more pronounced on the left and the presence of an ischemic lesion in the left portion of the troncus. A contrast multiplane transesophageal echocardiogram with the Valsalva maneuver excluded the presence of a cardiac defect including patent foramen ovale.

After suspension of sedatives, the patient recovered consciousness and was tracheally extubated on the fifth day. He was able to obey simple and complex verbal commands and did not show any motor or sensory deficits. He was discharged from the intensive care unit on the tenth day to a neurological ward. One month later he underwent a single photon emission CT (SPECT) examination that showed hypoperfusion of the right thalamic region and the parietal, temporal and frontal cortices (Fig. 1). After 1 month, the patient was discharged from the hospital with moderate impairment of short-term memory and he enrolled in a long-term neurorehabilitative program.

DISCUSSION

In our patient, there was recognizable evidence of subcutaneous emphysema, pneumomediastinum, pneumothorax, pneumopericardium, and the absence of tracheobronchial lesions. Since no obvious cause related to anesthesia maneuvers could be found, it is likely that air was forced into soft tissue by an exogenous source. The modern high-speed dental handpiece operates by compressed air at a pressure of 2.2 kPa and this drives a

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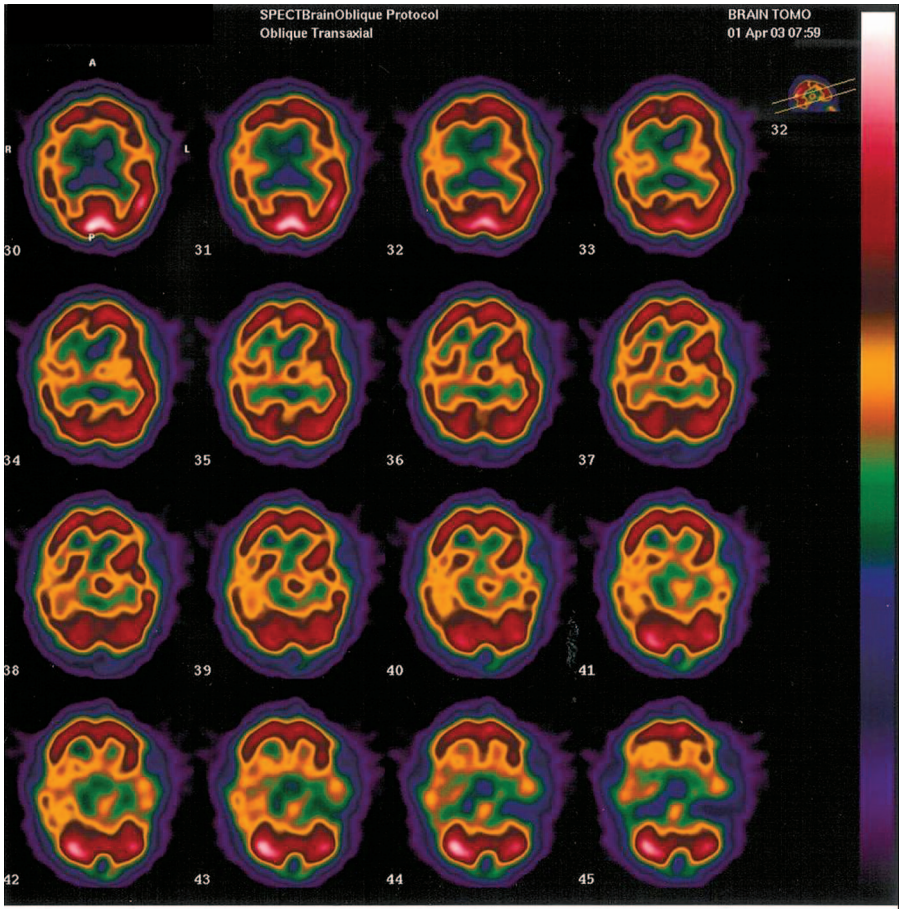

Figure 1. Single photon emission computed tomography (SPECT), brain oblique transaxial views showing hypoperfusion of the right parietal, temporal, and frontal cortex. The right thalamic region appears relatively hypoperfused compared to the contralateral portions.

turbine to rotating speeds more than 200.000 rpm. The tip of the burr rotating at this speed generates frictional heat and requires cooling to avoid tissue burns. Cooling is commonly achieved using a separate air jet and water directed onto the point of the burr. Air bubbles forced by the turbine burr drill may have entered through the mucosal breach and moved along tissue planes deep into the cervical fascia, through parapharyngeal and retropharyngeal tissues toward the mediastinum.

Recently, Sujeet and Shankar[6] presented a radiograph of the neck, clearly showing the presence of prevertebral air in the cervical soft tissues in a patient who had undergone a root canal therapy. The authors report that surgical procedures involving the lower third molar teeth may confer a predisposition to the development of soft tissue emphysema, and even fatal air embolism, when air-cooled dental turbines are used. The relationship between the cervical prevertebral space and the mediastinum illustrated by Sujeet and Shankar clearly explains how pneumomediastinum, bilateral pneumothorax, and pneumopericardium likely ensued in our patient. Moreover, the use of nitrous oxide during anesthesia may have increased the total volume of gas within distensible tissues. Pushed by a turbine burr drill and/or by pressure gradient, air bubbles penetrate the pterigoideal plexus and eventually enter the vena cava, right atrium, pulmonary vessels (by opening of the intra-extra alveolar shunts), left ventricle, aorta, right carotid artery, thereby causing ischemic lesions in the right temporal lobe, right frontal lobe, and right thalamus.

Gases from venous vessels may reach the arterial circulation by overwhelming the mechanisms that normally prevent arterial gas embolism. Studies in animals suggest that a large bolus of gas (20 mL or more) or small continuous amounts (11 mL/min) introduced into the venous system may generate intraarterial bubbles.[7] Moreover, paradoxical arterial gas embolism in the sitting position has been reported, although no cardiac defect could be demonstrated.[8,9] Butler and Hills described the concept of "critical rate of infusion of air:" at infusion rates below 0.30 mL/kg/min, venous emboli are trapped in the pulmonary vasculature, but infusion rates above 0.35 mL/kg/min exceed the "filtration threshold" and arterial spill-over occurs.[10] We suppose that the mechanism of paradoxical air embolism in our patient was an intrapulmonary shunt, because we were not able to identify any cardiac defect by contrast transesophageal echocardiography. Since we did not perform contrast transcranial Doppler, we were not able to demonstrate the presence of air inside the cerebral circulation. Nevertheless, we believe that the most important diagnostic criterion for air embolism is the patient's history; the clinical suspicion of arterial embolism is based on initial neurological symptoms and on the direct temporal relation between the onset of

symptoms and the performance of an invasive surgical procedure.[11] In addition, the preferential involvement of the right hemisphere, including the right thalamus, has been recognized as a distinctive feature of cerebral air gas embolism.[12]

The case reported here is unusual because the patient's symptoms started 45 minutes after restoration of consciousness and no signs or symptoms of pulmonary embolism were detected before the systemic cerebral symptoms occurred. Microbubbles may have been formed that did not cause clinically detectable signs of pulmonary air embolism, whereas symptoms of cerebral hypoperfusion became rapidly evident, perhaps reflecting the lower tolerance of cerebral tissue to reduced local flow. Prompt treatment with supplemental oxygen may have reduced the size of gas emboli by increasing the gradient for the egress of nitrogen from the bubbles. However, immediate hyperbaric oxygen treatment could not be provided because of the presence of pneuomthorax and pneumomediastinum.

In conclusion, although pneumomediastinum, pneumothorax and venous air embolism are not uncommon complications after dental surgery, systemic air embolism after dental surgery has not been reported. Anesthesiologists and dental surgeons should be aware of the possibility of this complication.

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