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A special case of ear barotrauma caused by hyperbaric oxygen therapy

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Abstract. A special case of ear barotrauma caused by hyperbaric oxygen therapy
The problem: A 52-year-old male with prior ear surgery on the right side presented at our department for the first time with a crushing pain behind his right ear, unsteady gait, blurry vision, and confusion. These symptoms occurred in our hospital, during the last 10 min of a hyperbaric oxygen therapy session for chronic posttraumatic tinnitus.
M*ethodology:* Audiometry and a CT scan of the brain were performed.
Results: The CT scan showed a pneumocephalus, with a dehiscent tegmen as the port of air entry.
Conclusions: We recommend careful consideration of the risks of hyperbaric oxygen therapy before treating patients with a history of ear surgery. This is particularly important in cases of chronic tinnitus, because there is currently no definitive evidence of treatment efficacy.

Introduction

Hyperbaric oxygen therapy has been advocated for treating tinnitus and idiopathic sudden sensorineural hearing loss, because it may improve the oxygen supply to the inner ear, which could promote the production of growth factors and ameliorate post-ischemic and post-inflammatory changes.[1,2] Normally, hyperbaric oxygen therapy is considered safe and well tolerated, but recent ear surgery is described as a relative contraindication.[3] Currently, the routine use of hyperbaric oxygen therapy for treating chronic tinnitus is not recommended, because there is no evidence of benefit. Only a few trials, which were methodologically flawed, showed some improvement in an acute setting (therapy started within 2 weeks of onset).[1]

This report describes a unique case of barotrauma caused by hyperbaric oxygen therapy in a patient with chronic tinnitus.

Case report

In February 2017, a 52-year-old Caucasian male consulted our ENT department with urgency for the first time. He had experienced tinnitus and consulted an external ENT specialist, who prescribed 10 sessions of hyperbaric oxygen therapy. No problems occurred during the first 9 sessions. However, during the final 10 min of the last session (when pressure was being reduced), he experienced a sudden, crushing pain behind the right ear, radiating to the occiput and face. He described a sensation of air bubbles behind the ear, accompanied by dizziness and nausea.

On presentation, the sensation of pressure persisted behind the right ear, but not in the ear. His hearing had not changed. He also reported blurry vision in his right eye and tingling in the right half of his face. His gait was very unsteady, but there was no lateralization to one side. No otorrhoea had occurred. He reported a general feeling of malaise, and he was sweating. This behaviour evoked a strange impression: he could not sit still, and he acted confused and agitated.

His national medical file (CoZo, a digital collaboration platform) was rather incomplete. Based on the medical file and the patient interview, a prior history was revealed. In 1979, in another hospital, he underwent ear surgery on the right side. He had been in two traffic accidents, one in 1984 and another in 2011. After that, he had experienced tinnitus on the left side, accompanied by various orthopaedic complications and a cervical hernia.

This study required no financial support or funding.

The medical file did not mention a cranial trauma, concussion, or temporal bone fracture. Surgery was planned for the cervical hernia in the near future. After the last accident, he underwent a psychiatric follow-up for stress-related problems caused by the insurance company's refusal to provide full coverage of all consequences of the accident. The psychiatric report stated that he showed signs of narcissism, but no signs of psychiatric problems that could be immediately related to his behaviour upon presentation. In his free time, the patient was an avid diver, but no diving accidents were mentioned.

The clinical examination showed a retro-auricular scar on the right side. Otomicroscopy showed an intact neotympanic membrane without haemotympanum. On the left side, the tympanic membrane was normal and intact, and the middle ear was properly ventilated. The facial nerve function was normal. The range of neck movement was strongly limited, and palpation of the right neck musculature and sternocleidomastoid muscle elicited severe pain. Eye movements were normal. Romberg's test demonstrated an irregular swaying with the eyes open and closed. There was no rotation with Unterberger's test, but he was very unstable. The head-thrust test and finger-to-nose test were normal. There was no dysdiadochokinesia. The pure-tone audiometry results were within normal limits on both sides.

These results indicated that the patient's complaints were not due to an otological/vestibular dysfunction. Therefore, the patient was urgently referred to a neurologist, with suspicion of an intracerebral aetiology.

A CT scan of the brain was performed (Figures 1 and 2). On the right side, the CT scan showed a large amount of intracranial air located subdurally. and a state after a canal wall up attico-antrotomy with a very thin to absent bony lining to the middle cranial fossa. There was no opacification of the middle ear or the air cells of the mastoid bone. These findings led to the conclusion that a dehiscent tegmen tympani – particularly in the medial and anterior portions - served as port for the entry of intracranial air.

The next day, after spontaneous resolution of the intracranial air, the patient had fully recovered, and he left the neurology department in good condition. No new CT scan was performed. There was no further follow-up at our clinic, but the neurologist

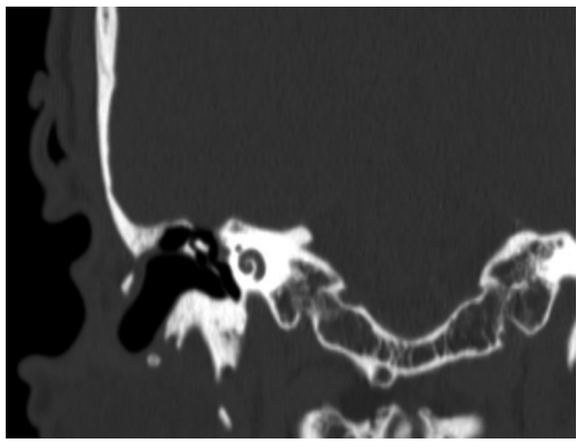

Figure 1

CT scan of the brain, coronal view. After a canal-wall-up attico-antrotomy, the middle cranial fossa had a very thin-to-absent bony lining.

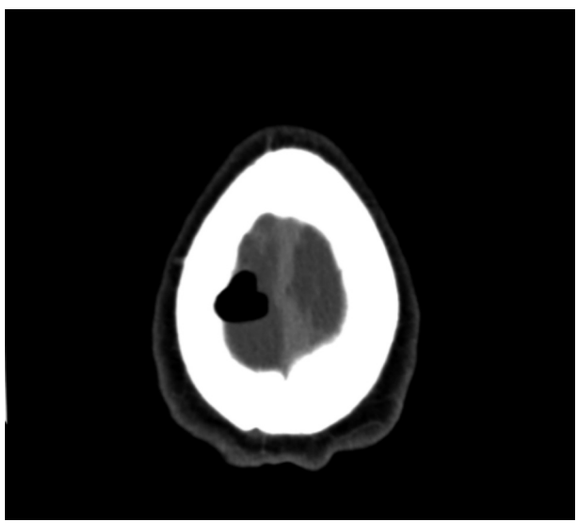

Figure 2

CT scan of the brain, axial view. Intracranial air, viewed as a black area.

referred the patient to the original ENT physician. Two months after the barotrauma occurred, the original ENT physician ordered a new CT scan, which confirmed a dehiscent tegmen tympani.

Discussion

Hyperbaric oxygen therapy can cause *barotrauma*. Although it is fairly straightforward to diagnose and manage a middle ear barotrauma, the ENT physician might be confronted with several potential forms of barotrauma and other complications that might arise from hyperbaric oxygen therapy.

An **inner ear barotrauma,** as described by Yamamoto, is an extremely rare complication of hyperbaric oxygen therapy.[4] This condition arises due to a difference in pressure between the middle ear and the inner ear, which can cause a rupture in the oval and/or round window and/or a secondary rupture in Reissner's membrane and/or bleeding within the inner ear.[4-7] These disruptions can lead to sudden onset of sensory neural hearing loss, tinnitus, vertigo, aural fullness, and dizziness.[4-7] The patient described in the present study did not experience any hearing deterioration; therefore, a diagnosis of inner ear barotrauma was quite unlikely.

A differential diagnosis should also consider a pulmonary barotrauma and decompression sickness, both of which can give rise to a **gas embolism**. An **arterial gas embolus** will break up when it passes a vascular branch point, and it can get stuck in a small blood vessel, which causes distal ischemia. When the ischemia occurs in the brain, it manifests as dizziness, and it can lead to focal motor, sensory, or visual impairments, seizures, loss of consciousness, apnoea, and death.[3]

Barotrauma (pressure-induced tissue damage) occurs when the pressure in an air-filled body space fails to equilibrate with the environment after changes in ambient pressure. According to Boyle's law, the absolute pressure of an ideal gas is inversely proportional to its volume, given constant temperature and gas mass, within a closed system.[6] Thus, when the pressure drops in a closed system, the gas volume increases. In the lungs, this can cause alveolar rupture. When the pulmonary veins tear, and the alveoli rupture, gas bubbles can pass into the pulmonary veins to the left heart, then become gas emboli in the systemic arterial circulation.[6] In these cases, a thoracic X-ray is routinely performed before starting therapy, to exclude a pneumothorax. Arterial gas bubbles can also arise when venous gas emboli exceed the filtering capacity of the pulmonary capillaries, and they are carried into the systemic arterial circulation. Or alternatively, venous gas bubbles carried to the heart can pass directly through a right-to-left shunt (e.g., through a patent foramen ovale).[1,6]

D*ecompression sickness* involves another mechanism. The law of Henry states that the quantity of a dissolved gas is proportional to the partial pressure of that gas (given a constant temperature).[6] Thus, during a diving descent, the pressure increases and more gas, particularly nitrogen, dissolves in the tissue. Conversely, during ascent, the sum of the gas tensions in the tissue may exceed the ambient pressure and lead to the liberation of free gas from the tissues into the blood stream, in the form of bubbles. Most diverse neurological manifestations of decompression sickness are thought to arise from gas bubbles forming and clotting within low pressure **venous plexuses**, where they obstruct venous blood flow, and – as described above –, they can eventually become arterial gas emboli.[6] In addition to cerebral emboli, is thought that **bubble formation within nervous tissue or otic fluid** might play a role in the neurological and vestibulocochlear symptoms associated with decompression sickness.[5,6] However, decompression sickness is a very rare event with hyperbaric oxygen therapy, because when 100% oxygen is administered, the nitrogen content in the bubbles is replaced with oxygen, which is rapidly metabolized in the tissues.[3]

Our patient harboured a large intracranial air bubble, which could not be explained by a gas embolism. Therefore, no further investigation was performed to explore a possible patent foramen ovale.

Oxygen toxicity can also give rise to neurological and vestibulocochlear symptoms associated with hyperbaric oxygen therapy. These symptoms include confusion, dizziness, sensory deficits, visual deficits, and seizures.[3] However, hyperbaric oxygen therapy sessions are carefully controlled to ensure that pressures do not fall below the threshold for central nervous system toxicity. Consequently, oxygen toxicity is a rare complication.[8]

This case study represents an extraordinary aetiology of barotrauma. Symptoms arose in the final 10 min of the hyperbaric oxygen session, during a reduction in the pressure, when the gas volume increased in the middle ear. Before the gas could be evacuated through a Eustachian tube, the excess of air found a way through the dehiscent tegmen tympani and the dura into the subdural space. The resulting pneumocephalus could explain the patient's complaints. In the absence of evidence that a CSF leak had occurred, we presumed that the air travelled subdurally, through a tear in a surgically weakened dural area, which served as a one-way valve. This type of one-way valve mechanism – where air enters in, but no CSF can escape, due to the presence of external pressure –

was previously described in a case of spontaneous pneumocephalus.[9]

Few previous studies have reported a pneumocephalus caused by barotrauma.[10-12] Two cases were described after air travel. In one case, the pneumocephalus developed when air entered through a semicircular canal dehiscence after a pneumolabyrinth had occurred. In another case, a spontaneous disruption in a weak spot of the roof of the mastoid was considered the route of entry.[11,12] The most common routes for air entry into the brain are through a skull base fracture, the nasal cavity/sinuses, or the ear. Another possibility is the presence of residual air in the skull after a craniotomy or skull-base surgery; in some cases, the air can expand and cause a tension pneumocephalus.[10] The latter possibility was unlikely in our patient, because he never underwent skull base surgery and no problems occurred during the first 9 sessions of hyperbaric oxygen therapy. Pneumocephalus after hyperbaric oxygen therapy caused by ear barotrauma has never been described before. In most cases, there is no need for any medical intervention, and the air resolves spontaneously. However, when a CSF leak or tension pneumocephalus occurs, surgical intervention is required.[11,13]

Conclusion

This study demonstrated that the risks and benefits of hyperbaric oxygen therapy should be carefully considered for treating tinnitus in patients with a history of ear surgery. Hyperbaric oxygen therapy is controversial as a treatment for tinnitus. A previous Cochrane database systematic review recommended against hyperbaric oxygen therapy in patients that have had tinnitus for a few years.[1] Some small studies have shown that hyperbaric oxygen therapy could improve hearing in patients with idiopathic, sudden sensorineural hearing loss, and possibly also tinnitus, but only when applied within two weeks of onset.[1,15] Consequently, the benefits of hypobaric oxygen therapy were doubtful for our patient, who had experienced tinnitus for several years. Moreover, the risk of complications, including pneumocephalus, are elevated at baseline for patients with a history of ear surgery.[3] We recommend that, when patients display confusion after hyperbaric oxygen therapy, pneumocephalus should be excluded with a CT.[14]

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