Presumed Arterial Gas Embolism After Breath-Hold Diving in Shallow Water

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Dive-related injuries are relatively common and mostly occur in recreational or scuba diving. We report 2 children with acute central nervous system complications after breath-hold diving. A 12-year-old boy presented with unilateral leg weakness and paresthesia after diving beneath the water surface for a distance of ~25 m. After ascent, he suddenly felt extreme thoracic pain that resolved spontaneously. Neurologic examination revealed right leg weakness and sensory deficits with a sensory level at T5. Spinal MRI revealed a nonenhancing T2-hyperintense lesion in the central cord at the level of T1/T2 suggesting a spinal cord edema. A few weeks later, a 13-year-old girl was admitted with acute dizziness, personality changes, confusion, and headache. Thirty minutes before, she had practiced diving beneath the water surface for a distance of ~25 m. After stepping out, she felt sudden severe thoracic pain and lost consciousness. Shortly later she reported headache and vertigo, and numbness of the complete left side of her body. Neurologic examination revealed reduced sensibility to all modalities, a positive Romberg test, and vertigo. Cerebral MRI revealed no pathologic findings. Both children experienced a strikingly similar clinical course. The chronology of events strongly suggests that both patients were suffering from arterial gas embolism. This condition has been reported for the first time to occur in children after breath-hold diving beneath the water surface without glossopharyngeal insufflation.

CASE REPORTS

Patient 1

A previously healthy 12-year-old boy presented with unilateral leg weakness and paresthesia. Two days earlier, he had practiced repeated diving beneath the water surface for a distance of 25 m. He reports that, after ascent, he suddenly felt extreme thoracic pain that resolved spontaneously. During the following hours, he subsequently experienced progressive sensory changes affecting his right side. This was followed by acute right leg weakness later the same day. A cerebral and lumbar spinal MRI that was ordered by the community neurologist on the next day was reported normal and he was discharged from the hospital. Two days later, the patient was referred to our hospital with persistent right leg weakness and paresthesia and an inability to walk. Neurologic examination revealed right leg weakness and sensory deficits with...
a sensory level at T5. Pain sensation was intact; however, he reported a decreased sensation to pin-prick and temperature below the mid thorax. Proprioception and vibration sense were intact. Reflexes were positive for upper extremities, exaggerated bilaterally at the patellar level with sustained clonus at the right ankle. In addition, there was an upgoing toe on the right. Blood tests and electrocardiogram results were normal.

A complete spinal MRI revealed a nonenhancing T2-hyperintense lesion in the central cord at the level of T1/T2 suggesting a spinal cord edema (Fig 1). Together with the clinical course, this led to the diagnosis of a presumed AGE. Because of the delayed diagnosis, the patient was not considered a candidate for hyperbaric oxygen treatment. He was treated with intensive physiotherapy and over a course of several weeks he subsequently regained normal sensorimotor functions.

**Patient 2**

A few weeks later, a 13-year-old previously healthy girl was admitted to our hospital with acute dizziness, personality changes, confusion, and headache. Thirty minutes before, she had practiced diving beneath the water surface for a distance of ∼25 m. After ascent, she felt sudden severe thoracic pain and lost consciousness over 1 to 2 minutes. Afterward she reported headache and vertigo, and numbness of her complete left side. Neurologic examination on arrival in the emergency department revealed reduced sensibility on her left side to all modalities, a positive Romberg test, and vertigo. A cerebral MRI, including diffusion weighted imaging (DWI) was normal. Further tests, including blood test, electrocardiogram, chest radiograph, and echocardiography, were all without abnormalities. On spirometry mild airway obstruction was detected, as indicated by reduced forced expiratory volume in 1 second and forced expiratory volume percentage in 1 second. She had just recovered from an acute airway infection, and an ear, nose and throat (ENT) investigation revealed a mild laryngitis.

She was treated with acetylsalicylic acid (2 mg/kg) and a colloidal infusion. In addition, 12 and 36 hours after her injury, hyperbaric oxygen treatment was initiated in a decompression facility. Within 24 hours she recovered from her neurologic symptoms.

**DISCUSSION**

We here report 2 children who experienced a strikingly similar clinical course after breath-hold diving. The chronology of events (diving, ascending, sudden thoracic pain, followed by acute onset of neurologic symptoms) strongly suggests that both patients were suffering from AGE, possibly as a result of a pulmonary barotrauma.

We are only aware of a single comparable case in the literature, a 21-year-old man who developed severe neurologic symptoms immediately after ascending from a long-distance dive at a depth of ∼4 feet.³ He suffered from sudden onset headache, dizziness, and generalized tingling, and reported chest pain. Within minutes he developed a generalized tonic-clonic seizure and eventually died of cardiopulmonary arrest. On autopsy “large amounts of air were noted in the cerebral arteries and veins.” The authors concluded that AGE secondary to local barotrauma was the likely cause.

AGE, a well-known complication of scuba diving, is generally described together with DCS under the global term DCL.⁴ AGE is defined as the introduction of gas emboli into the arterial circulation. Cerebral AGE may occur by rupture of the alveolar membrane from pulmonary barotrauma or from shunting of venous air via a left-to-right shunt such as a patent foramen ovale or through a transpulmonary capillary bed. Pulmonary barotrauma is the most severe form of barotrauma in scuba divers and occurs during ascent, either due to breath holding and/or local air trapping (eg, due to chronic lung disease, acute lung infection, laryngospasm).¹ As the ambient pressure is reduced during ascent, gas inside the lungs will expand in volume. If this expanding gas is not allowed to escape by exhalation, the alveoli and surrounding tissues may tear. Air is released into the pulmonary capillaries, and hence the arterial circulation. In scuba divers most often the target organ is the thoracic spinal cord.² Typically, clinical symptoms of AGE develop within minutes of reaching the surface.

AGE can occur after a brief compressed gas dive to a depth of as little as 1 m.⁴ Even a mildly elevated transpulmonary pressure from 95 to 110 cm (10 kPa/0.1 bar) can be sufficient to lead to a pulmonary rupture with subsequent AGE.

However, the question is if this mechanism is plausible in a shallow breath-hold diving scenario? It has long been believed that DCI due to breath-hold diving is literally nonexistent. After several well documented cases (eg, in Ama divers
in Japan), there is now growing evidence that severe neurologic events due to DCI after breath-hold diving can occur. These events are significantly correlated with the severity of dive exposure, including dive depth, dive time, and short surface intervals.

For unknown reasons a sparing of the spinal cord in cases of breath-hold diving related DCI has been observed. Another characteristic of DCI after breath-hold diving was that neurologic symptoms typically resolved within several hours, even without treatment. Possible pathomechanisms have been suggested with DCS rather than AGE being the most plausible etiology. However, explanatory models do not satisfactorily apply to breath-hold diving in shallow water. Therefore, different mechanisms or additional risk factors need to be assumed in our patients.

Glossopharyngeal insufflation has been described as a mechanism for AGE after single breath hold dives. It is commonly used by breath-hold divers to prolong their time under water by increasing their oxygen stores. However, none of our patients has reported using this technique.

Other predisposing factors such as underlying pulmonary (due to air trapping mechanisms) or cardiac diseases (right-to-left shunts) may increase the risk for AGE in breath-hold divers. Our first patient’s history did not point to any preexisting lung disease and no cardiac abnormalities were found. However, he did not undergo a formal pulmonological workup, including bronchial provocation challenges (eg, to rule out exercise induced bronchoconstriction). Contrary, the second patient was recovering from a recent airway infection, and we found evidence for airway obstruction. Theoretically, airway obstruction leading to trapped air could cause a critical increase of the alveolar pressure and a pulmonary barotrauma in breath-hold divers. Bronchospasm can develop in asthmatic patients rapidly, commonly induced by exertion. Airway obstruction may be localized and uncontrolled expansion of the distal airway can result in pulmonary barotrauma and a gas embolism.

Pulmonary barotrauma leading to pneumomediastinum has been repeatedly reported in the context of noncompetitive breath-hold dives in shallow water, including children. Even simple breath-holding as well as extreme respiratory efforts (eg, during pulmonary function testing or exercise) have been reported as causes of spontaneous pneumomediastinum. The predominant clinical symptom of pneumomediastinum is a stabbing precordial or retrosternal chest pain, which was the initial clinical presentation of both our patients shortly after ascending. Although routine chest radiographs were normal in both our patients, it is important to stress that this test may miss up to 50% of pneumomediastinum cases. In addition, our first case presented with much delay, and clinical as well as imaging signs could have normalized in the meantime.

More importantly, it is possible that alveolar rupture has occurred in the absence of pneumomediastinum. After barotrauma, there are several routes for extrapulmonary gas to follow, 1 of which is systemic gas embolism. Air may enter the pulmonary capillaries, and hence the arterial circulation.

We speculate that, in our patients, gas emboli entering the cerebral and spinal vessels caused the neurologic events.

Various other mechanisms or differential diagnosis were taken into consideration in our patients, none of which sufficiently explained the clinical history.

We excluded DCS as an explanation in both patients. It is generally assumed that there is no risk of DCS during a single breath-hold dive in shallow water.

There exist a limited number of other differential diagnoses.

In the first patient, the clinical course suggested an acute spinal cord injury. Spinal cord injury in children and adolescents can be due to a wide range of etiologies. An MRI would have identified space occupying lesions such as tumors, arteriovenous malformations, or hemorrhagic cord infarction. In the absence of a trauma, spinal cord injury without radiographic abnormality was not considered. Sudden onset leg weakness and a sensory level may also result from a spinal thromboembolic occlusion. However, no embolic source could be identified, and the close temporal relationship to the diving session together with the complete recovery led us to conclude that an air embolus was the likely explanation.

Minor trauma has been associated with fibrocartilaginous embolism (FCE), a cause of childhood spinal cord infarction. Patients usually experience sudden back pain with subsequent onset of rapid neurologic deterioration. FCE most often affects the anterior spinal artery territory. Typically, MRI demonstrates T2 hyperintense area that is linear or pencillike and extending multiple spinal levels. In the absence of a preceding trauma and typical MRI features, FCE was excluded in our patient.

Another differential diagnosis to consider is transverse myelitis. The clinical presentation can be similar to spinal cord infarction. Additional diagnostic criteria for transverse myelitis include MRI findings not corresponding to a vascular territory, gadolinium enhancement, and progression of clinical symptoms within hours to days after onset. We concluded that the clinical course in
that girl was not suggestive of transverse myelitis.

In our second case, the symptomatology would best be described as a strokelike event. It would be compatible with an arterial ischemic stroke, a transient ischemic attack, or a hemorrhagic stroke. However, imaging findings, including a complete stroke protocol (DWI, apparent diffusion coefficient [ADC], T2, magnetic resonance angiography) were normal. The clinical features were also not suggestive of migraine, the most common differential diagnosis of strokelike episodes in this age group. Another differential diagnosis to consider is “shallow water blackout.” However, unlike in our patient, this typically happens when breath-hold divers attempting a distance swim lose consciousness while still in the water, presumably from hypoxia.

Finally, various significant physiologic changes can occur during dry breath-holds as well as breath-hold diving, referred to as “diving response.” Among others, this includes hypercapnia, reduction of cardiac output, peripheral vasoconstriction, increase of blood pressure, increase of cerebral blood flow, and involuntary diaphragmatic contractions. In addition, it has been demonstrated recently that cerebral autoregulation is acutely impaired during maximal apnoea. In extreme situations is very difficult to be finally proved, AGE after a pulmonary barotrauma is the most plausible explanation for the reported symptoms in these patients.

**CONCLUSIONS**

AGE after breath-hold diving near water surface might be a rare but important condition, possibly in the setting of preexisting diseases or risk behavior. The fact that we have seen 2 independent patients within a few weeks period might suggests that there are further yet unidentified cases. Even though the clinical course in our patients was favorable, hyperbaric oxygen treatment should be considered in similar cases.

**ABBREVIATIONS**

AGE: arterial gas embolism
DCI: decompression illness
DCS: decompression sickness
FCE: fibrocartilaginous embolism

**REFERENCES**

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