CASE REPORT

Acute psychosis associated with diving

R. O. HOPKINS and L. K. WEAVER

Psychology Department and Neuroscience Center, 1122 SWKT, Brigham Young University, Provo, Utah; and Departments of Pulmonary and Critical Care Medicine, LDS Hospital, Eighth Avenue and C Street, Salt Lake City, Utah

Hopkins RO, Weaver LK. Acute psychosis associated with diving. Undersea Hyper Med 2001; 28(3):145–148. — There are only a few reported cases of psychiatric disorders presenting as decompression sickness (DCS). Previous reports indicate that DCS can result in personality change, depression, Munchausen’s syndrome, and pseudo stroke. We report two cases of acute psychoses that occurred following diving as suspected DCS and were treated with hyperbaric oxygen, which did not improve the psychotic features. One patient had symptoms of DCS including myalgia, weakness, and fatigue; however the symptoms were inconsistent. The symptom onset and nitrogen loading from his dive profiles made the diagnosis of DCS unlikely. The second patient exhibited mild joint pain, fatigue, and psychosis that was temporally associated with diving but no other symptoms of DCS. Following a detailed medical evaluation we determined that these two patients did not have DCS or arterial gas embolism (AGE). Although it is highly unlikely that a pure psychotic episode will arise as a result of DCS, physicians caring for divers with symptoms of DCS or AGE and acute psychosis may consider a trial of recompensation therapy while completing the medical evaluation. Divers with acute psychosis without signs and symptoms and benign dive profiles are unlikely to have DCS or AGE.

decompression illness, diving, and psychosis

Decompression sickness (DCS) may result in cerebral injury, including neuroanatomical abnormalities (1), cerebral perfusion defects (2), cognitive impairments, personality change (3), mood changes (4), and encephalogram abnormalities. Patients may also experience detachment from reality, disorientation, and affective disorders (5). Elliott and Kindwall (1999) state that until proven otherwise neurologic symptoms or deficits following diving should be considered a manifestation of DCS (6). Rozsahegyi (7,8) reported pseudo-neurothemia, hysteria, and personality change associated with neurologic deficits and EEG abnormalities following DCS in divers and caisson workers. In a study of 37 sport divers treated for neurologic decompression illness, 19% reported psychiatric difficulties and significantly more symptoms of depression compared to sport divers who did not have a history of neurologic decompression illness (9).

Although personality and affective changes have been reported following DCS (6,10), it is unknown how many participants in recreational diving have a history of psychiatric disorders. According to the Diagnostic and Statistical Manual of the American Psychiatric Association (4th ed.), little systematic information is available regarding prevalence of psychotic disorders in the general population but all available evidence indicates that they are “uncommon" (11). The 2001 Divers Alert Network report shows that 3% (n = 2,520) of the Project Dive Exploration divers reported antidepressant medica-

Copyright © 2001 Undersea and Hyperbaric Medical Society, Inc. 145
CASE STUDIES

Case 1: Patient 1 (P1) was a 39-yr-old white male, Search and Rescue diver searching for the body of a drowning victim. The rescuers dove once to 40 ft [119 kPa (g)] for 45 min at an altitude of 6,000 ft [81 kPa (g)], in fresh water that was 58°F (14.4°C) with poor visibility. After surfacing mid-dive, without a decompression or safety stop, he developed a severe headache, blurred vision, lightheadedness, and was cold with chills, followed within 2 h by joint pain. He was taken to a local emergency department, where his mental status deteriorated to the point where he did not respond to verbal commands or questions. By pressurized air medical transport he was flown to a tertiary care center, where on admission he had severe pain, acute mental status change, and was mostly non-communicative. Medical history was obtained from the medical records from the referring hospital and from telephone conversations with his wife. His medical history was negative for substance abuse, neurologic disease, and psychiatric disorders. His mini-mental status exam was 16 out of 30, indicating severe cognitive impairment. He was oriented to person, place, year, and season, but not month, day, or date. He recalled two out of three objects but recalled only one of the three objects after 3 min. He could not do serial sevens. He could follow simple commands but when asked to write the sentence “I like to dive”, he wrote a scribbled response. He was severely disoriented, had decreased motor abilities including dysmetria and dysdiadochokinesia. His gait was slow and ataxic, and he was unable to maintain his balance during the Romberg test. His neurologic exam, laboratory tests, drug screen, and a chest radiograph were normal; however, he had a mildly elevated serum creatine kinase level (normal = 20–400 U · liter⁻¹) of 473 U · liter⁻¹ (MB isoenzyme, 2% of the total). The admission differential diagnosis included DCS, arterial gas embolism (AGE), carbon monoxide poisoning, contaminated gas in the diving tank, multiple sclerosis, hypoxic encephalopathy, meningitis or encephalitis, postictal state post seizure. He received two HBO₂ treatments over the next 36 h. The first treatment was a Navy table 6 (17), 10 h after diving. Following the first HBO₂ treatment his mental status improved to where he was able to respond slowly to verbal commands, but his mental status remained significantly impaired. He complained of severe headache, tingling, and joint pain. The second HBO₂ treatment was at 2.4 atm abs [243.2 kPa (a)] for 90 min. Neuropsychological test performance was inconsistent and abnormal. He exhibited marked psychomotor slowing, could not remember his wife’s name, his age, misspelled his last name, but remembered the day of the week and year. He could repeat only 2 digits forward and was unable to recall the alphabet or count to 10.

Following HBO₂ treatment on hospital Day 2, P1’s condition deteriorated and he was unresponsive to all stimuli. He had symmetric weakness with increased catatonia. He was unable to raise his arms on command; however he could raise them to indicate where he was experiencing pain. He was able to walk when unaware of being observed but was unable to walk on command. A neurologist noted probable factitious weakness or conversion catatonia or both. Brain computed tomography (CT) was normal but a brain magnetic resonance (MR) scan showed several small subcortical white matter hyperintensities thought to be due to age; however, there was no acute bleeding or ischemic changes.

Since there was no significant improvement following HBO₂ treatments, a psychiatric consult was obtained. Psychiatry felt the acute mental status changes were of psychiatric origins; however, a lumbar puncture and EEG were performed to rule out meningitis or encephalitis. Both were normal. During a psychiatric interview with amobarbital sodium, P1 started talking and moving spontaneously. He stated that he found a decomposed body during the dive. He tried to pull the body to the surface but the body came apart in his hands. He was very upset, returned to the boat, and did not tell anyone about the body. He was able to articulate an intense and emotional conflict during the dive and it was thought that the patient had a dissociative episode during the dive. After the amobarbital sodium interview, P1 reported that he was amnesic for the 3 days after the dive. After discharge from psychiatry his cognitive, affective, and motor function returned to normal. His thought processes were coherent and he had no delusional or paranoid thoughts. P1’s discharge diagnosis was dissociative disorder, psychosis NOS, and acute distress disorder.

Case 2: P2 was a 22-yr-old Japanese male scuba diver, who was diving off the California coast with multiple dives to 85 ft [260 kPa (g)] for up to 30 min (according to friends and the referring physician). On Day 1 he completed one dive of 32 ft [98 kPa (g)] for 28 min. On Day 2 he performed two dives at 50 ft [153 kPa (g)] for 30 min and 40 ft [122 kPa (g)] for 30 min. The surface intervals between dives are unknown. In the evening his dive buddy noted that P2 was behaving abnormally, was unfriendly and occasionally incoherent, and withdrew from the group when two WWII veterans teased him about being Japanese. On Day 3 he completed four dives: 50 ft [153 kPa (g)] for 23 min, 80 ft [245 kPa (g)] for 21 min, 50 ft [153 kPa (g)] for 21 min, and 40 ft [122 kPa (g)] for 25 min. P2 was unable to recall specific episodes from these dives but reported that following diving he
had elbow and knee pain and he was extremely tired. Within 12 h of diving he had auditory hallucinations and abnormal behavior, including erratic driving. He did not attend college for 2 days post-diving. He was taken to a local hospital for evaluation and was flown by pressurized air medical transport to a tertiary care center for evaluation of possible DCS. Neurologic exam, toxicology, laboratory tests, brain CT, and MR scans were normal. He reported delusions and hallucinations: (e.g., "The TV told him to go visit the President of the United States"). He described himself as a social drinker, smoked one-half pack of cigarettes per day, and occasionally used marijuana. He denied a history of psychiatric or neurologic illness. Because of the temporal nature of the psychosis, abnormal behavior, and mild joint pain to diving, the possibility of DCS was considered.

Cognitive testing revealed impaired attention and verbal memory. His Verbal Intelligence Quotient was 81; Performance Intelligence Quotient, 94; Verbal Memory Index, 72; Nonverbal Memory Index, 110, and a Full Scale Memory Index of 88 (mean = 100, standard deviation = 15). P2 received two treatments with HBO₂, a Navy table 6 (17) followed by 2.4 atm abs [243.2 kPa (a)] for 90 min the following day, with some improvement in memory. Due to the delusions, he was admitted to Psychiatry, where he became intermittently erratic with periods of more organized behavior. His thought processes were perseverative, tangential, and circumstantial with loose associations. His affect was somewhat disconnected from the content of his current situation. P2’s discharge diagnosis was psychosis NOS.

DISCUSSION

These two patients with acute psychosis that initially looked like “possible DCS” were treated with HBO₂. However, as further information became available, we concluded that neither patient had DCS. Psychosis has been reported as a manifestation of DCS (6,10), yet details of the cause and references were not provided, so we believe that it is unlikely that psychosis can be attributed to DCS in the absence of other neurologic symptoms.

P1 had pain, confusion, and fatigue suggestive of DCS, yet the nitrogen loading for this particular dive was low given his dive profile, making the diagnosis of DCS extremely unlikely. A more likely diagnosis for P1’s symptoms is AGE. In addition, stress associated with diving can be a contributor to diving accidents and resulting injuries and fatalities that occur in recreational divers (18). Any event while diving that results in increased apprehension or increases autonomic arousal, including cold water, can result in stress or panic or both. It is possible that P1 had AGE due to a panic ascent after finding the body, yet his initial presentation was atypical for AGE (18). If his psychosis was due to AGE he should have had other neurologic signs in addition to the severe headache (19). For example, one study found that patients with AGE had elevated serum creatine kinase levels with a median of 1218 U·liter⁻¹ due to release of creatine kinase from skeletal muscle following diving-associated AGE (20). Although P1 had a mildly elevated serum creatine kinase level, it was significantly lower than the median level reported to be associated with AGE (20). After two HBO₂ treatments his symptoms not only did not improve but became more pronounced. Following the amobarbital sodium interview his symptoms disappeared which confirmed the diagnosis of psychosis. P2 had symptoms of DCS including mild joint paint, fatigue, and cognitive impairments. He also developed psychosis that may have been coincidental or due to DCS and subsequent brain injury. However, his neurologic exam, MR, and CT scans of the brain were within normal limits and since there are no other reported cases of psychosis due to DCS, we discount DCS as the cause of his psychosis. The psychiatric consult made it clear that the psychosis was due to factors unrelated to diving.

The initial presentation of physical complaints, cognitive impairments, and psychosis that was closely associated with diving, initially lead us to believe that these divers had DCS. The incidence of psychosis as a presenting symptom of DCS is unknown, but given the mechanisms of DCS, it is unlikely that psychosis would be the only major symptom. After treatment with HBO₂ and extensive evaluations by neurologists and psychiatrists, the diagnosis of psychosis, not DCS, was confirmed. It is also possible that the psychosis these divers experienced was a direct result of psychological stress caused by diving (i.e., anxiety) (18).

A diver with psychosis that is temporally associated with diving, as the sole expression of disability should not be considered to have DCS or AGE. If the psychosis were due to intravascular bubbles and brain injury, we would expect the diver to have other neurologic manifestations. We recognize, as exhibited by our cases, information that is linked with medical decision making may not be available immediately. If a diver has acute symptoms of DCS or AGE, including psychosis, a trial of HBO₂ is indicated.

For divers with psychosis and symptoms of DCS or AGE after compressed air diving, DCS should be considered in the differential diagnosis. Acute psychosis as the sole manifestation following compressed gas diving is not DCS or AGE.
REFERENCES


