Spontaneous hydrocele resolution after hyperbaric oxygen treatment: A clinical case report

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ABSTRACT
Hyperbaric oxygen is considered an adjunctive treatment to medical and surgical care. We present a unique case in which a male patient with decompression illness affecting inner ear and spinal cord presented a worsened unilateral hydrocele synchronously with the neurological pathology. At the Diving and Hyperbaric Medicine Department, the patient was initially recompressed using a modified United States Navy Treatment Table 6A; on the following days he was treated for decompression illness using hyperbaric oxygen. Hyperbaric oxygen treatment has not been used for the treatment of hydrocele, but disappearance of the hydrocele occurred during the time he was treated with hyperbaric oxygen for decompression illness. He was discharged on Day 8, free of symptoms, having a normal neurological examination.

INTRODUCTION
Hyperbaric oxygen is considered an adjunctive treatment to medical and surgical care. We present a unique case, in which a unilateral hydrocele that worsened after a diving accident spontaneously resolved using hyperbaric oxygen treatment.

CASE REPORT
Patient
A 29-year-old male was delivered to the hospital outpatient department complaining of vertigo, dizziness, gait disorder and muscle weakness in the lower extremities. Symptoms appeared 10 minutes after completing a scuba dive, starting with dizziness and voiding.

Description of the dive
The diver executed a 27-minute scuba dive, using air as his breathing gas, to a maximum depth of 55.8 meters of sea water (msw). He stayed at maximum depth for 12 minutes, ascent rate was 10 msw per minute and he made decompression stops at 6 and 3 msw for a total of eight minutes. During the previous three days he performed similar dives, one dive every day, with maximum depths ranging from 44.6 to 55 msw. The critical dive took place 25 hours after the preceding one.

Clinical examination
On arrival, he was examined by the Diving and Hyperbaric Medicine Department staff. At that time, symptoms were progressing and included vertigo, tinnitus, fatigue and muscle weakness in the lower extremities. Clinical examination revealed motor deficit and abnormal tendon reflexes of the limbs and nystagmus – consistent with decompression illness affecting the inner ear and spinal cord.

Blood tests
Blood tests showed increased white blood cells (WBC), hemoconcentration and increased levels of creatine phosphokinase (CPK) and glucose (GCB) [WBC 28, 570/uL-polys 89.6%, Hct 49.9%, Hb 16.5g/dL, PLT 334,000/uL], Glu 175.8 mg/dl, CPK 350 U/L].

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Treatment
He was taken immediately to the hyperbaric chamber (four hours after surfacing from the dive) and treated with recompression using a modified United States Navy Treatment Table 6A. A Foley 16F catheter was placed, and a unilateral scrotal swelling was revealed. The patient reported that inside the recompression chamber, the hydrocele worsened and had increased in volume since the dive accident. Hydration (intravenous normal saline), steroid therapy, plasma expanders and gastroprotection were part of his initial treatment. A marked improvement was evident after the session concerning symptoms (mainly vertigo) and results of neurological examination. Scrotal swelling was recorded at that point by us, appearing unaffected by the initial treatment.

Hospitalization
Following this first hyperbaric oxygen therapy (HBO₂) session a urologic consultation was held; the scrotal ultrasound performed revealed a right hydrocele. The patient’s medical history reported a right hydrocele due to a previous accidental injury during a football match several years ago. He had undergone a scrotal Doppler test at that time that revealed a mild testicular edema. Since then he had experienced scrotal swelling over a period of years after the original injury.

During the following days, the patient was treated at the Diving and Hyperbaric Medicine Department for decompression illness, using hyperbaric oxygen. His treatment schedule included three sessions on the day following the accident, two sessions daily for the next three days and one session per day for the last three days. All sessions involved 90 minutes of 100% oxygen breathing at 1.8 atmospheres absolute (ATA), in the multipurpose chamber of our hospital. A Foley catheter was removed on Day 5, with normal bladder function. Pure-tone audiometry on Day 4 revealed a light high-frequency sensorineural hearing loss that was consistent with the diagnosis and the side affected.

The patient was discharged on Day 8, free of symptoms and having a normal neurological examination. Interestingly, at the conclusion of the hyperbaric oxygen therapy schedule, the hydrocele was resolved.

Follow-up
The patient was advised to undergo an appropriate cardiological examination to exclude the presence of patent foramen ovale.

When seen at one month after discharge, he had a normal neurological examination and no indication of hydrocele. Doppler ultrasound transthoracic and transeosophageal echocardiography did not reveal any structural abnormalities of the heart. On follow-up at six months, results of clinical examination were normal, too.

DISCUSSION
It is well known that the key pathophysiologic event in decompression illness is the formation of bubbles in body fluids, originating from dissolved inert gas (1). Venous bubbles are detected more frequently than arterial ones in divers. Whatever their origin, these intravascular bubbles have been shown to interact with elements of blood and endothelial cells and initiate the inflammation cascade, giving rise to decompression illness. Finally, microcirculation disturbance with capillary leakage occurs. These systemic reactions, coupled with localized phenomena that accompany decompression illness, are responsible for the heterogeneity of its clinical course and the varieties of its manifestations.

Tissue swelling has been identified in the severe forms of the disease as cerebral, spinal cord or pulmonary edema (2). Other than those, swelling of muscle, skin, parotid glands, breast and extremities have all been reported as lymphatic manifestations. We are not aware of any previously published report of hydrocele associated with decompression illness or having a causal association with scuba diving.
Obstruction of lymphatic drainage after interaction with bubbles is widely accepted as the cause for tissue edema in mild forms of decompression illness. This could be the case in our patient, as judging from his dive profile and subsequent illness, his nitrogen load was high. Nitrogen bubbles could have obstructed lymph nodes involved, leading to hydrocele, although this is questionable. However, hyperbaric oxygen has been shown to be beneficial in other cases of disturbed lymphatic circulation.

Our patient had a definite diagnosis of decompression illness involving the central nervous system and inner ear. The activation of the inflammatory cascade undoubtedly led to fluid extravasation as also shown by his biochemistry (hemoconcentration, marked increase of white blood cell count). However, medical examination did not detect other areas of profound tissue edema in his body. In our experience of treating more than 600 cases of decompression illness, we have not seen such a presentation. In some cases, in the course of their disease, we have seen edema formation in body areas not directly related to organs affected (personal experience). Concerning this case, the hydrocele worsened synchronously with the neurological pathology.

HBO₂ involves pure oxygen breathing in conditions of increased ambient pressure. This is accomplished in chambers specially constructed for that reason. It was around the 1960s when hyperbaric oxygen started being studied extensively; the biochemical and physiological effects HBO₂ produces have led to its therapeutic use in various diseases and conditions (3). Among its various effects, it produces significant vasoconstriction, which enhances recovery in crush injury, compartment syndrome and other cases of post-traumatic tissue edema (4).

Our patient was treated for his main disease – decompression illness – using hyperbaric oxygen. The therapeutic schedule was determined by the patient’s response to treatment: He underwent HBO₂ treatment for eight consecutive days and a total of 12 sessions (excluding the very first recompression session). By the time he was discharged from the hospital, hydrocele was resolved, as demonstrated by the follow-up urologic consultation. We cannot determine whether hyperbaric oxygen therapy led to that result. HBO₂ therapy has not been used for the treatment of hydrocele, but disappearance of hydrocele occurred during the time the patient was treated with HBO₂ for decompression illness. Existing extensive knowledge of the effects of hyperbaric oxygen (5) gives a plausible explanation for this result.

In conclusion, we report a case of hydrocele in a diver brought to the hospital for neurological decompression sickness, confirmed with ultrasound study. The hydrocele disappeared during hospitalization at the time he was treated for decompression illness with the use of HBO₂. To our knowledge, this is the first reported case of hydrocele worsening in a patient suffering from a dive accident that resolved by the end of hyperbaric oxygen therapeutic sessions.

REFERENCES


