

Case Reports

Resolution of Neurological DCI after long treatment delays.

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D. D. WEISHER

St. Thomas Neurology, PLLC, P.O. Box 7307, Paragon Medical Building, Suite 209, St. Thomas, U.S.V.I. 00801

Weisher D D. Resolution of Neurological DCI after long treatment delays. *Undersea Hyperb Med* 2008; 35(3):159-161. We report two interesting cases in which both divers sustained a very serious Type II decompression sickness. This involved substantial neurological impairment which was successfully treated despite having a delayed treatment time 12 hours or more. The treatment used hyperbaric oxygen recompression therapy with the addition of lidocaine i.v. drip. This first case was in November 2007 and the second was in December 2007 and both patients made excellent recoveries.

CASE 1

We report a 54-year-old right-handed female with a history of hypertension and lupus. She was also status post surgery for abscess of colon and was using a colostomy bag. She is a highly experienced diver and at this time, holds a Master Diving rating. The patient had no previous history of decompression sickness.

The patient report two dives on the day of the illness. The first was a 84 foot depth for 40 minutes and the second was a 50 foot depth for 45 minutes with a dive interval of 90 minutes.

Within an hour, following the second dive, the patient sustained numbness in both lower extremities and ataxia that gradually progressed to severe weakness including both upper extremities and lower extremities. The

neurological deficit progressed to the point where she was not able to stand and could just barely have use of both upper extremities.

As the incident occurred in St. Croix, and the hyperbaric chamber is located on St. Thomas at the Schneider Regional Medical Center, there is often a logistic problem of transportation. Because of this, when we received the call from St. Croix, I decided to start the patient on a Lidocaine drip using a bolus of 100 mg and a rate of 2 mg/minute i.v. in addition to i.v. fluids and 100% oxygen via nonrebreather mask. Both oxygen and lidocaine were started approximately 1-2 hours post onset of injury.

At the time, the patient's current medication included Toporal, aspirin, Procardia, Plaquenil, and Prednisone which she has been taking for her pre-existing condition.

The patient finally arrived at the facility on St. Thomas, via Aerojet Air Ambulance and recompression therapy was begun 12 hours post onset of symptoms (around midnight.) At that time, prior to the first recompression therapy schedule, on exam, the patient was alert and oriented x3. Speech demonstrated only a slight dysarthria of doubtful clinical significance. Pupils were 2/3 and 2/3 and equally reactive. Optic fundi were benign. There was no facial asymmetry. Ocular range of motion was full. The patient developed a sensory gradient to pin prick and at about T4 level and below. Motor exam was roughly grade 1-2 / 5 to the left lower extremity and -4 / 5 to the right lower extremity. Weakness to both upper extremities improved substantially after starting lidocaine and oxygen. Deep tendon responses were 1 bilaterally. Plantar responses were clearly extensor with fanning on the left and flexor on the right. There was no clonus.

Because of the concerns regarding the possibility of oxygen toxicity seizure, Lidocaine drip was stopped just prior to the first recompression therapy. The patient underwent initially a Table 6 recompression dive treatment table. Shortly after arriving at 60 feet, the patient started to feel significantly better.

Afterwards, the patient was transferred to the medical floor on nasal canula oxygen 4 liters per minute.

The patient underwent a second Table 6 on hyperbaric oxygen treatment which further resulted in significant improvement. On the following day, she felt 95% better. She further underwent two Table 5 recompression treatments (total of 4 treatment dives) and continued to improve. 72 hours after the last dive, she flew home.

The patient, overall, achieved at least 98% improvement in neurological recovery in her own words.

Case #2

A 44-year -old healthy female (not on any medication) from England vacationing at the Bitter End, Virgin Gorda, British Virgin Islands who underwent an 85 foot dive for 45 minutes and a second 45 food dive for 30 minutes with a 2 hour interval. Within an hour from the second dive, the patient began to notice paraparesis and numbness to both lower extremities. She had hoped that symptoms would go away and therefore did not seek medical attention until the following day when she became weaker. While in my office in St Thomas, I received the urgent call from Virgin Gorda. At that time (approximately 24 hours post onset of symptoms) I ordered 100% oxygen via non-rebreather mask and immediately transported to Schneider regional medical center in St. Thomas. Upon arrival at the hospital, via helicopter a few hours later, she was given a 100mg bolus of lidocaine and started on lidocaine drip at 2mg / min.

Her exam demonstrated a soft touch and pin prick deficit to T-5 dermatome and below. With a grade 4/5 motor strength to both lower extremities. She also had urine retention. She was able to ambulate but only with assistance.

She was maintained on lidocaine during the HBO Table 6 treatment and sent to ICU in lidocaine and oxygen nasal canula at 4 lt / min. 12 hours post HBO treatment she sustained a full 100% recovery and was sent back to her hotel. No commercial flight for 72 hours.

DISCUSSION

These clinical case histories are remarkable not only for the fact that there was significant improvement in neurological condition despite a prolonged HBO treatment latency 12 hours and 24 hours after the event, but also because of the use of adjunctive

Lidocaine i.v. therapy which we believe might have assisted in their recovery. In the first case, the patient was on lidocaine for at least 12 hours prior the HBO treatment but was stopped before recompression because of concerns of lowering seizure threshold. The second case is remarkable for starting lidocaine therapy 12 hours after symptom onset and was maintained on lidocaine throughout treatment. Both patients did extremely well despite their delay in treatment and prolongation of symptoms.

Currently, UHMS guidelines classify Lidocaine for neurological injury as a Class 2B level C recommendation for treatment. There is very little research in Lidocaine therapy for decompression Type II sickness. Most of this research involved cats and dogs and demonstrated some degree of significant efficacy (1) (2). Hirayama et al reported 17 critical cases in which lidocaine was used to control life threatening edema in the posterior fossa in humans. They report that lidocaine successfully provided time for observation to select 5 correct candidates out of 17 indicated for aggressive decompression surgery (3).

The mechanism for the benefit of lidocaine therapy is not clearly established and may simply be to effect reduced edema, but also, could have a positive benefit on the ischemic penumbra by virtue of decreasing metabolic demand.

Nonetheless, these clinical cases suggest that further research is needed to determine the efficacy of lidocaine therapy in Type II decompression sickness. However because of my experience with success in using lidocaine in type II decompression sickness and significant success in serious brain and cervical cord trauma, I will continue to use it in all my serious neurological decompression cases.

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