Persistent Nocturnal Enuresis as a Symptom of Type II Decompression Sickness Successful Treatment with Delayed Hyperbaric Oxygen Therapy

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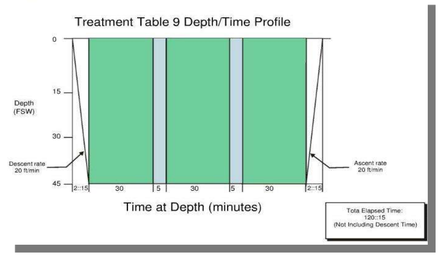
BACKGROUND / CASE PRESENTATION

Nocturnal Enuresis (NE) is a heterogeneous disorder with various underlying pathophysiological mechanisms causing a mismatch between the nocturnal bladder capacity and the amount of urine produced during sleep. This occurs with a simultaneous failure of conscious arousal in response to the sensation of bladder fullness. *Nocturnal Enuresis in the Absence of Daytime Incontinence* (NEADI) is rarely reported as an adult onset symptom occurring in only 0.02% of all cases of NE. New onset NEADI is predominantly seen in men with prostatic disease; 100% in one retrospective review of 3,277 cases. NEDI due to decompression sickness (DCS) or air gas embolism (AGE) has never been reported. A 42 y/o female recreational diver with NEADI following treatment of Type II DCS was evaluated. NEADI persisted post initial HBO_2 treatment (USN TT6 then TT5) and continued on the divers return to the United States. The diver presented to our facility fourteen days after her last HBO_2 treatment and underwent a detailed dive history and physical exam. She had no prior history of NE or any urological pathology. NEADI was diagnosed and began on the night of her diving accident and first HBO_2 treatment. Upon further questioning she revealed that not all original post dive symptoms had resolved after her first USN TT6. The patient agreed to be treated at our facility daily with a USN TT9 (Figure 1) as a delayed (trailing) treatment protocol. She refused post void residual urine and urodynamic evaluation, but agreed to MRI evaluation and Air Contrast Bubble Study.

Figure 1



Hypothesis / Rationale for Treatment

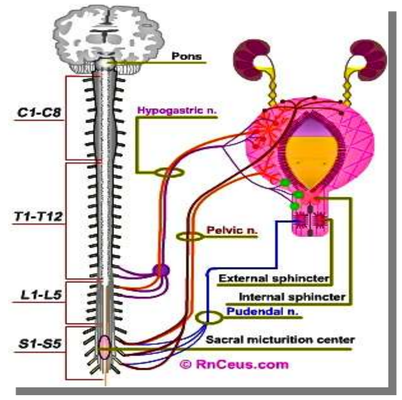
This patient had a provocative dive profile causing Type II DCS with Cutis Marmorata. The possibility of AGE was entertained with or without Patent Foramen Ovale (PFO) despite the provocative dive profile. The inflammatory response and edema associated with endovascular gas bubbles has been well described. We postulated this patient had residual inflammation, edema, and resultant local hypoxia of the neurological tissue involved with normal micturition. The normal micturition pathway can be seen in Figures 2 & 3. Considering the suspected under treatment at the time of the acute injury, ongoing inflammation, edema, ischemia and hypoxia were considered more likely. HBO_2 treatments have been successful at decreasing inflammation by down-regulating intracellular adhesion molecules (ICAMS), decreasing edema, and restoring normoxia to hypoxic tissues. Normoxia will favor return of physiologic function and ultimately return the micturition pathway to normal.

Figure 2

Potential Areas of Neurological Inflammation, Edema, Hypoperfusion & Hypoxia Associated with NEADI

- Cerebrum
- Pons
- Spinal Cord
  - Cervical
  - Thoracic
  - Lumbar
  - Sacral

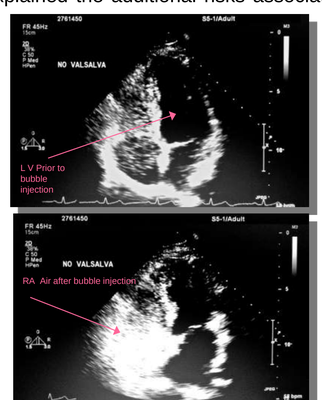
Figure 3



RESULTS

MRI's of the brain, brainstem, and spinal cord were negative for areas of damage or infarct. Other urological testing to determine another cause of the symptoms other than DCS were refused. Trials of no fluid ingestion two hours before sleep over two days failed to resolve the NE.

The NEADI improved the evening of the first hyperbaric treatment as measured by the decrease in the amount of urine volume lost while sleeping. Symptoms gradually improved with each consecutive treatment. The NEADI was completely resolved following the fifth treatment. The diver received a total of 8 trailing treatments and remains asymptomatic at the time of preparing this presentation (12 weeks following the last trailing treatment). Following improvement the patient agreed to be evaluated with a bubble study. A moderate sized right to left shunt was present (Fig 4) indicating the presence of a PFO. The patient was advised against any further scuba diving activities and explained the additional risks associated with having a moderate sized right to left shunt and PFO.



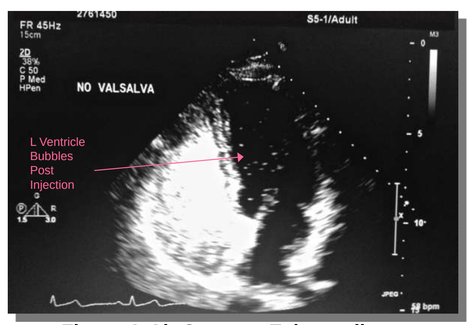


Figure 4 Air Contrast Echocardiogram Depicting a Moderate Right to Left Shunt

Discussion

NEADI is prominently a disease associated with prostatic hypertrophy in men and extremely rare (0.02%). Our patient and diver was a young female. To our knowledge this is the first reported case of NEADI reported as a consequence of DCS II or AGE in our peer reviewed literature. The neurologic control of micturition is complex. This patients type II decompression symptoms most likely were associated with arterial embolism. We based this on the complex neurological control of micturition and the various levels of the central and peripheral nervous system potentially effected by bubbles. The possibility of AGE in this diver was further supported with a positive bubble study post treatment and following resolution of symptoms. The use of "trailing" or delayed hyperbaric oxygen treatments for persistent symptoms of DCS is controversial. However, this patients improvement suggests that there are complex pathophysiologic changes in the nervous tissue causing reversible but persistent damage. These changes occur following the initial hypoxic insult causing inflammation, edema, hypoperfusion, and hypoxic neurological lesions similar to the ischemic "Penumbra" seen in certain cerebral neurologic dysfunction including radiation brain damage. It is here that delayed treatment with hyperbaric oxygen may have its benefit with chronic and persistent symptoms of Type II DCS and AGE.

ABBREVIATED BIBLIOGRAPHY

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