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Lidocaine as an adjunct to hyperbaric therapy in decompression illness: a case report

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Drewry A, Gorman DF. Lidocaine as an adjunct to hyperbaric therapy in decompression illness: a case report. Undersea Biomed Res 1992; 19(3):187–190.—A recreational scuba diver with nervous system decompression illness had a poor response to hyperbaric therapy. On the basis of available and supportive in vivo data, he was then given a continuous infusion of lidocaine (serum levels, low therapeutic range: 6.4–9.1 μmol/liter). Within 24 h of the start of this infusion he experienced a full resolution of his neurologic deficits. His symptoms recurred 3 days later, but again completely resolved after further hyperbaric therapy and concurrent administration of lidocaine (serum levels: 6.9–9.1 μmol/liter). This observation supports the need to conduct trials of lidocaine as an adjunct to hyperbaric therapy in decompression illness.

decompression illness
lidocaine
hyperbaric therapy

Decompression illness (DCI) involving the nervous system may not respond completely to hyperbaric therapy. An effective adjunct to recompression is needed. There are data to support lidocaine in neural injuries and perhaps in DCI (1–11), and we present here a case in which i.v. lidocaine was of apparent benefit in a diver with neurologic DCI.

CASE REPORT

A 34-yr-old, self-taught recreational scuba diver, who had dived infrequently over the preceding 5 yr, dived to 40 m in seawater for a duration of 28 min. This included 14 min at 40 msw and a 5-min decompression stop at 10 msw. Thirty minutes after surfacing he developed nausea and numbness in his left calf. When he was admitted to the Royal New Zealand Naval Hospital (RNZNH) 13 h later, he also had fleeting pains in both elbows and both knees and constant pain in his lower back and abdomen.

On examination, he had bilateral quadriceps muscle weakness, more pronounced on the left. The left ankle jerk and the anal reflex could not be elicited and those in the upper limbs were pathologically brisk. Sensation was impaired in the left leg. His mentation seemed slow and deliberate; he was unable to perform simple mental arithmetic and recalled only one of three paired objects on short-term memory testing. A diagnosis of neurologic DCI was made, he was started on i.v. fluids (Hartmann's solution, 1 liter stat and 1 liter 4 hourly), and a urinary catheter was inserted. He was recompressed, using RN table 62 (USN 6).

The patient reported relief from joint pain and an improvement in his thinking within 15 min of reaching 18 msw. His muscle weakness resolved within 30 min and this improvement was maintained throughout the treatment. However, the following day he was found to have lax anal tone, an absent left ankle jerk, and anal reflex and a sensory deficit corresponding to the left L5-S1 dermatome. The urinary catheter was left in place and a positive fluid balance maintained. A repeat recompression 16 h after the first hyperbaric treatment (RN table 62 with 2 extensions each at 18 and 9 msw) made no difference to these phenomena. Immediately after this unsuccessful treatment, a lidocaine infusion was begun, with a loading dose of 360 mg over 2 h and then 60 mg/h continuously (lidocaine 500 mg in 500 ml 0.9% NaCl).

Within 24 h of starting the lidocaine, the anal tone was found to be normal and both the anal and left ankle jerk reflexes could be elicited. He was again recompressed to 18 msw breathing oxygen 24 h after the second hyperbaric therapy, but his recovery was already complete before this was begun. The lidocaine infusion was continued for a further 48 h and then stopped. Daily blood levels of lidocaine showed low therapeutic levels of 6.9, 9.1, and 6.4 μmol/liter, using an FPOA assay (12).

The patient remained well, with normal bladder and bowel function and no sensory deficit, and was discharged 2 days later. Although his back pain and left calf sensory loss recurred 36 h after discharge, complete resolution was quickly achieved when lidocaine was infused (doses as above). However, on this occasion, the infusion was concurrent with recompression to 18 msw. Consequently, this response cannot be simply attributed to either modality. Lidocaine levels were again in the low therapeutic range, 6.4 and 7.1 μmol/liter. At final discharge, he had no detectable neurologic deficit and he was still well when reviewed 1 mo. later.

DISCUSSION

In DCI, damage is caused by bubbles, not only directly but also because of their effects on inflammatory proteins, blood, and blood vessels (13). Recompression could therefore effectively remove bubbles, but adverse effects may continue because of the processes already initiated by these bubbles. There are data to show that lidocaine reverses many of these effects (1–11, 14–17) and might ameliorate nervous system damage in DCI.

Systemic administration of lidocaine at therapeutic levels alters cerebral blood flow (1, 15) and reduces intracranial hypertension, including that resulting from arterial bubbles (2, 18). Direct application of lidocaine to the spinal cord increase blood flow (8), and lidocaine in low concentration preserves nerve conduction in isolated nerves and inhibits the leak of cations across a cell membrane (1, 6), an event that may lead to cell death (19).

Intravascular bubbles induce thrombus formation and the adhesion and migration of leukocytes (13); lidocaine inhibits these processes (14, 16, 17), which will improve the microcirculation and reduce the release of both toxic superoxidases and lysosomal enzymes (14, 17).

Several studies, using arterial bubbles to model DCI, have shown a benefit for lidocaine given in doses equivalent to low therapeutic levels in man. Evans et al. (4, 5) showed that both pretreatment with a single dose of lidocaine and a 2-h infusion of lidocaine after 15-min of bubble-induced ischemia in cats resulted in a greater recovery of SEP amplitude and a reduction in infarct size.

McDermott et al. (9) failed to demonstrate a statistically significant difference in recovery of SEP amplitude between cats who received lidocaine plus hyperbaric oxygen (HBO) and cats who received HBO alone. The difference in the extent of recovery between the 2 groups was sufficiently small that expanding the study to obtain statistical significance was unjustifiable. Dutka (3) however, did report a significant benefit in adding lidocaine to HBO in dogs subjected to carotid artery bubbles and then transient hypertension induced by i.v. adrenaline. Animals who received HBO plus lidocaine recovered more somatosensory evoked potentials amplitude than those treated with HBO alone (60 vs. 28%).

These studies suggest that lidocaine is an effective treatment in vivo for bubble-induced nervous system ischemia and may provide additional benefit when added to HBO. Our patient demonstrated an apparent and significant response to i.v. lidocaine after recompression alone had been ineffective in completely resolving his neurologic deficits. Because the benefit of lidocaine in DCI in man cannot be established from case reports, a multicenter prospective controlled and randomized trial of lidocaine as an adjunctive therapy in neurologic DCI needs to be performed.

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