Spinal cord decompression sickness in an inside attendant after a standard hyperbaric oxygen treatment session

Jacek Kot[1,2], Ewa Lenkiewicz[2], Edward Lizak[2], Piotr Góralczyk[2], Urszula Chreptowicz[2]

[1] *National Centre for Hyperbaric Medicine, Institute of Maritime and Tropical Medicine in Gdynia, Medical University of Gdansk, Poland*
[2] *Department of Hyperbaric Medicine and Sea Rescue, University Centre for Maritime and Tropical Medicine in Gdynia, Poland*

Corresponding author: *Associate Professor Jacek Kot, National Centre for Hyperbaric Medicine , Institute of Maritime and Tropical Medicine, Medical University of Gdansk, Powstania Styczniowego 9B, 81-519 Gdynia, Poland*
jacek.kot@gumed.edu.pl

Key words
Decompression illness; Hyperbaric facilities; Occupational health; Working in compressed air

Abstract

(Kot J, Lenkiewicz E, Lizak E, Góralczyk P, Chreptowicz U. Spinal cord decompression sickness in an inside attendant after a standard hyperbaric oxygen treatment session. Diving and Hyperbaric Medicine. 2021 March 31;51(1):103–106. doi: 10.28920/dhm51.1.103-106. PMID: 33761550.)
Medical personnel in hyperbaric treatment centres are at occupational risk for decompression sickness (DCS) while attending patients inside the multiplace hyperbaric chamber (MHC). A 51-year-old male hyperbaric physician, also an experienced diver, was working as an inside attendant during a standard hyperbaric oxygen therapy (HBOT) session (70 minutes at 253.3 kPa [2.5 atmospheres absolute, 15 metres' seawater equivalent]) in a large walk-in MHC. Within 10 minutes after the end of the session, symptoms of spinal DCS occurred. Recompression started within 90 minutes with an infusion of lignocaine and hydration. All neurological symptoms resolved within 10 minutes breathing 100% oxygen at 283.6 kPa (2.8 atmospheres absolute) and a standard US Navy Treatment Table 6 was completed. He returned to regular hyperbaric work after four weeks of avoiding hyperbaric exposures. Transoesophageal echocardiography with a bubble study was performed 18 months after the event without any sign of a persistent (patent) foramen ovale. Any hyperbaric exposure, even within no-decompression limits, is an essential occupational risk for decompression sickness in internal hyperbaric attendants, especially considering the additional risk factors typical for medical personnel (age, dehydration, tiredness, non-optimal physical capabilities and frequent problems with the lower back).

Introduction

Medical personnel at hyperbaric treatment centres are at occupational risk for decompression sickness (DCS) while attending patients inside the multiplace hyperbaric chamber (MHC). The risk depends on both environmental and physiological factors. The environmental factors define the amount of inert gas dissolved in all tissues, depending on ambient pressure, time of exposure, breathing mixture and the decompression profile after a session. Additionally, physiological factors including age, exercise capability, level of hydration and acclimatisation influence the risk of DCS.[1]

Case description

The patient consented to publication of the following case details.

The patient was a 51-year-old male hyperbaric physician with a medical history of hypertension (well-controlled with drugs) and overweight (body mass index 28 $kg \cdot m^{-2}$), who was an experienced diver (thousands of logged dives including technical ones) with a history of recurrent back pain induced by physical exercise since his youth. He was working as an inside attendant during a standard hyperbaric oxygen treatment (HBOT) session in a large walk-in MHC. The session consisted of a 6-minute linear compression, 70-minutes at the pressure of 253.3 kPa (2.5 atmospheres absolute, 15 metres' seawater equivalent), then a 6-minute linear decompression without any decompression stops according to the Polish regulations.[2] During the whole session, the inside attendant was breathing ambient compressed air. There was no substantial physical activity during the session, and the previous hyperbaric exposure was about three days before. Later on, he claimed that for several days he had been psychologically tired due to work overload, and was stressed, over-caffeinated and dehydrated.

About 10 min after the session, he reported a burning sensation from the back toward the left leg, down to the knee, the loss of feeling in that region, decreased muscle strength, and no Babinski sign or plantar reflex (already lost in his youth). Trans-thoracic echocardiography (TTE) was conducted about 5 minutes later, showing four heart

chambers without detection of any bubbles either in the supine position or after knee bends (done mostly on the right leg). An independent physician, anaesthesiologist and diving medicine/hyperbaric specialist confirmed objective neurological signs. A lignocaine infusion was started (1 $mg \cdot kg^{-1} \cdot hour^{-1}$) with oral rehydration (1.5 L water). The decision was made to commence recompression treatment as soon as possible starting with compression to 283.6 kPa (2.8 atmospheres absolute) with oxygen and then continuing either with US Navy Treatment Table 6 (USN TT6) or converting to Comex CX30 with heliox 50% oxygen/50% helium, as typically used in spinal cord DCS in our hyperbaric centre for diving accidents. Recompression effectively started 90 minutes after the onset of symptoms.

After 10 min of breathing oxygen at 2.8 ATA, the apparent resolution of neurological symptoms was reported by the patient, and the standard USN TT6 was completed without any extension. After the session, no neurological symptoms persisted, other than the permanently missing plantar reflex. The lignocaine infusion was stopped, and the patient was discharged from the centre. He returned to regular hyperbaric work after four weeks of avoiding hyperbaric exposure. Eighteen months after the incident, transoesophageal echocardiography (TOE) was conducted with bubble contrast injected just before the Valsalva manoeuvre with no sign of persistent (patent) foramen ovale (PFO).

Discussion

Venous gas emboli (VGE) have been observed in inside attendants in a number of cases, depending on exposure pressure and time.[3,4] However, the rate of decompression illness (DCI), defined either as decompression sickness (DCS) or arterial gas embolism (AGE), among IAs differs between centres. A 2018 review of 14 articles on occupational risks for inside attendants participating in 79,776 hyperbaric sessions reported nine DCI cases in two centres; an incidence of 0.01% or one case per 8,864 sessions.[5] In one of two papers where cases were observed, there were four DCS cases reported in 28,747 hyperbaric sessions, but none with a neurological background.[6] The other paper reported in total five cases in 8,072 hyperbaric sessions, including three cases of neurological DCS.[7] Unfortunately, there is no specific information about those DCS with neurological symptoms, other than in two cases it was related to the inner ear.

Severe neurological symptoms of DCS or eventual death of medical attendants after hyperbaric treatment sessions are rare events. Until now, there has been only one fatal case described with a direct relation between death and DCS. This fatality occurred in 1991, when a 52 year-old nurse died within 90 minutes of exiting a MHC and autopsy findings confirmed her cause of death as DCS.[8–12]

One case of severe neurological DCS occurred in 1999 when a 43-year-old hyperbaric nurse became permanently quadriplegic (eventually leading to death from overwhelming infection after several years) due to neurological complications from a premature exit from USN TT6 with omitted decompression and both pulmonary and spinal cord DCS as a result.[11–13] The other publication from 2002 reported that a medical attendant at a hospital hyperbaric centre suffered 'a serious episode of neurological decompression illness', without giving any detailed information.[3]

Another case of cerebral and spinal cord DCS involving an inside attendant, which happened in 2001, was related to rapid chamber decompression due to deterioration of the patient, a diver with ventricular fibrillation, after several hours under pressure with the maximum treatment pressure of 607.8 kPa (6.0 atmospheres absolute). The inside attendant involved, a 44-year-old nurse, breathed oxygen during a very rapid chamber decompression and some minutes later she was recompressed for omitted decompression. After the completion of recompression treatment, she exited from the chamber and eventually returned home, where she was found several hours later in acute distress. Serial hyperbaric oxygen therapy and supportive care were incompletely successful, and she remained cognitively impaired.[12,14]

Yet another case of neurologic DCS was described in 2012.[15] A 50-year-old male complained of weakness and paresthesias in the lower extremities which began after serving as an inside attendant during a standard wound-healing hyperbaric treatment (222.9 kPa, 2.2 atmospheres absolute, 90 minutes at pressure) in a MHC. Within 10 minutes after the conclusion of the session, the patient experienced irritability, confusion and was unable to walk. He was recompressed with a USN TT6 within 60 minutes. His symptoms improved with compression; the patient was then treated with 222.9 kPa (2.2 atmospheres absolute) HBOT sessions until he was asymptomatic. Transthoracic echocardiography with bubble contrast performed 18 months after the event demonstrated a large PFO.

In our case, the spinal cord DCS in an inside attendant occurred after a standard HBOT session with a maximum pressure of 253.3 kPa (2.5 atmospheres absolute) and a bottom time within no-decompression limits. He had no PFO, but some additional risk factors were clearly identified, including age, overweight, dehydration and tiredness.

There are at least several possible pathophysiological mechanisms that may contribute to spinal cord DCS, including gas emboli, venous infarction, autochthonous bubbles or a vacuum phenomenon.[16–17] The specific mechanism cannot be confirmed in our case. The open question is whether lower back problems in the past with some permanent residual signs (loss of planar reflex) can predispose to DCS. A relationship between spinal canal stenosis and the development of spinal cord DCS was described in recreational scuba diving.[18] In the described case, the lower back pain occasionally occurred after heavy

exercise with lifting and gradually subsided within hours or days. CT scans conducted several years before did not show any sign of spinal canal stenosis.

Recompression treatment and adjunctive therapy (mainly lignocaine, nonsteroidal anti-inflammatory drugs [NSAID]) in spinal cord DCS is still debatable. A Cochrane review concludes that both the use of heliox and the addition of NSAID may reduce the number of recompressions required but neither improve the odds of recovery.[19] The European Committee for Hyperbaric Medicine (ECHM) recommends HBOT/recompression treatment tables (USN TT6 or helium/oxygen (heliox) Comex Cx30 or equivalent) for the initial treatment of DCI (strong recommendation, low level of evidence), but at the same time suggests the use of lignocaine and heliox recompression tables for severe neurological DCI (weak recommendation, low level of evidence), as well as oral tenoxicam (or similar NSAID) for appropriately selected DCI cases (weak recommendation, moderate level of evidence).[20] The Undersea and Hyperbaric Medicine Society (UHMS) advocates using US Navy oxygen treatment tables (or the similar RN and Comex tables, with initial recompression to 283.6 kPa (2.82 atmospheres absolute, 18 metres' seawater, 60 feet seawater equivalent) claiming that treatments at pressures exceeding 283.6 kPa or using helium as a diluent gas has not been demonstrated to be superior, and that their 'speculative' use should be reserved for facilities with experience and suitable hardware.[21] On the other hand, the UHMS does not give clear recommendations for adjunctive pharmacological therapy for DCI but presents guidelines for clinical efficacy of using different drugs, including lignocaine and NSAID. In summary of those guidelines, usefulness/efficacy of both lignocaine and NSAID in neurological DCI is less well established by evidence/opinion (Class IIb) based either on the consensus opinion of experts only (for lignocaine) or data derived from a single randomised trial or nonrandomised studies (for NSAID). In our clinical practice, a decision on using heliox recompression tables, lignocaine and NSAID is left to the treating physician, but most patients with neurological DCI receive all of them. In this case, the decision was agreed between treating physician and the patient (also the hyperbaric specialist) to try an oxygen table first (USN TT6) before considering switching to heliox Cx30 table (available at any moment in the same chamber). Fast resolution of all symptoms within the first 10 minutes under pressure confirmed the choice and prompted cessation of pharmacological therapy after the session.

In our hyperbaric centre, the decompression schedule of medical staff after standard HBOT sessions is planned according to the Polish regulations for commercial diving operations.[2] For standard HBOT sessions at 253.3 kPa (2.5 atmospheres absolute), it is allowed to have a bottom time of 80 minutes for no-decompression exposures. According to standard operating procedures, the decompression utilises only compressed air breathing to ensure the freedom of attendants to take direct care of patients in case of need. However, the personnel are advised to breathe 100% oxygen for either 10 minutes before commencing decompression or during decompression and decompression stops, or both, according to the recommendations of the ECHM.[22] Unfortunately, during this particular session, oxygen was not used for breathing.

Conclusion

Any hyperbaric exposure, even within no-decompression limits, is an occupational risk for decompression sickness in hyperbaric attendants, especially considering the additional risk factors typical for medical personnel (age, dehydration, tiredness, non-optimal physical capabilities and frequent problems with the lower back).

References

1 Pollock NW. Factors in decompression stress. In: Pollock NW, Sellers SH, Godfrey JM, editors. Rebreathers and scientific diving. Proceedings of NPS/NOAA/DAN/AAUS Workshop; June 16-19, 2015. Catalina Island (CA): Wrigley Marine Science Center; 2016. p. 145–56. [cited 2020 August 1]. Available from: https://www.omao.noaa.gov/sites/default/files/documents/Rebreathers%20and%20Scientific%20Diving%20Proceedings%202016.pdf. .

2 Kot J, Sićko Z. New Polish occupational health and safety regulations for underwater works. Int Marit Health. 2007;58(1–4):149–56. PMID: 18350984.

3 Risberg J, Englund M, Aanderud L, Eftedal O, Flook V, Thorsen E. Venous gas embolism in chamber attendants after hyperbaric exposure. Undersea Hyperb Med. 2004;31:417–29. PMID: 15686273.

4 Cooper PD, Van den Broek C, Smart DR, Nishi RY, Eastman D. Hyperbaric chamber attendant safety I: Doppler analysis of decompression stress in multiplace chamber attendants. Diving Hyperb Med. 2009;39:63–70. PMID: 22753198.

5 Pougnet R, Pougnet L, Lucas D, Henckes A, Loddé B, Dewitte JD. Health effects of hyperbaric exposure on chamber attendants: A literature review. Int Marit Health. 2018;69:58–62. doi: 10.5603/IMH.2018.0009. PMID: 29611615.

6 Bell J, Thombs PA, Davison WJ, Weaver LK. Decompression tables for inside chamber attendants working at altitude. Undersea Hyperb Med. 2014;41:505–13. PMID: 25562942.

7 Pougnet R, Henckes A, Pougnet L, Cochard G, Dantec F, Dewitte JD, et al. Occupational accidents among attendants inside hyperbaric chambers in France. Med Lav. 2015;106:17–22. PMID: 25607284.

8 Hyperbaric chamber nurse dies of decompression sickness; unit gets OK. Hosp Secur Saf Manage. 1992;13:3. PMID: 10122695.

9 Court Listener. Harley L Vause v. Bay Medical Center, Douglas L. Florida, USA: District Court of Appeal of Florida, First District; 1996. [cited 2020 Aug 1]. Available from: https://www.courtlistener.com/opinion/1797165/vause-v-bay-medical-center/.

10 Desautels DA. UHMS experience and mishap report (unpublished report). Undersea and Hyperbaric Medical Society Annual Scientific Meeting; Cancun, Mexico. UHMS; 1997.

11 Sheffield PJ, Pirone CJ. Decompression sickness in inside attendants. In: Workman WT, editor. Hyperbaric facility

safety: A practical guide. Flagstaff (AZ): Best Publishing Company; 1999. p. 643–63.
12 Clarke R. Health care worker decompression sickness: Incidence, risk and mitigation. Undersea Hyperb Med. 2017;44:509–19. doi: 10.22462/11.12.2017.2. PMID: 29281188.
13 OSHA Accident: 170902555 – Employee paralyzed from nitrogen saturation. Occupational Safety and Health Administration; 1999 Apr 25. Report No.: 0950645. [cited 2020 August 1]. Available from: https://www.osha.gov/pls/imis/accidentsearch.accident_detail?id=170902555.
14 Gonzales D. Hyperbaric safety pre-course. Undersea and Hyperbaric Medical Society 2015 Annual Scientific Meeting; Montreal, Canada. UHMS; 2015.
15 Johnson-Arbor K. Type II decompression sickness in a hyperbaric inside attendant. Undersea Hyperb Med. 2012;39:915–9. PMID: 23045920.
16 Yanagawa Y, Omori K, Ishikawa K, Jitsuiki K, Yoshizawa T, Takeuchi I, et al. Hypothesis: The influence of cavitation or vacuum phenomenon for decompression sickness. Diving Hyperb Med. 2016;46:190. PMID: 27723023.
17 Francis TJR, Mitchell SJ. Pathophysiology of decompression sickness. In: Brubakk AO, Neuman TS, editors. Bennett and Elliott's physiology and medicine of diving. 5th ed. Edinburgh: Saunders; 2003. p. 530–56.
18 Gempp E, Louge P, Lafolie T, Demaistre S, Hugon M, Blatteau JE. Relation between cervical and thoracic spinal canal stenosis and the development of spinal cord decompression sickness in recreational scuba divers. Spinal Cord. 2014;52:236–40. doi: 10.1038/sc.2013.121. PMID: 24126850.
19 Bennett MH, Lehm JP, Mitchell SJ, Wasiak J. Recompression and adjunctive therapy for decompression illness. The Cochrane Database Syst Rev. 2012;2012(5):CD005277. doi: 10.1002/14651858.CD005277.pub3. PMID: 22592704. PMCID: PMC6516885.
20 Mathieu D, Marroni A, Kot J. Tenth European Consensus Conference on Hyperbaric Medicine: Recommendations for accepted and non-accepted clinical indications and practice of hyperbaric oxygen treatment. Diving Hyperb Med. 2017;47:24–32. doi: 10.28920/dhm47.1.24-32. PMID: 28357821. PMCID: PMC6147240.
21 Moon RE, Mitchell SJ. Decompression sickness. In: Moon RE, editor. UHMS Hyperbaric oxygen therapy indications. 14th ed. North Palm Beach (FL): Best Publishing Company; 2019. p. 153–62.
22 ECHM. The 6th European Consensus Conference on prevention of dysbaric injuries in diving and hyperbaric work Geneva, Switzerland: European Committee for Hyperbaric Medicine; 2003. [cited 2020 April 1]. Available from: http://www.echm.org/documents/ECHM%206th%20Consensus%20Conference%20Geneve%202003.pdf.

Conflicts of interest and funding: nil

Submitted: 08 July 2020
Accepted after revision: 22 September 2020