ELSEVIER

Archives of Medical Research 34 (2003) 26–30

Archives of Medical Research

ORIGINAL ARTICLE

Differential Diagnostic Problems of Decompression Sickness—Examples from Specialist Physicians' Practices in Diving Medicine*

Nadan M. Petri and Dejan Andrić

Undersea and Hyperbaric Medicine Department, Naval Medical Institute, Split, Croatia

Received for publication March 6, 2002; accepted October 18, 2002 (02/043).

It can be expected that the differential diagnosis problem of decompression sickness will increase in the future due to the increasing number of divers. During the last 30 years, 232 divers were treated for decompression sickness (DCS) at the Naval Medical Institute (NMI) in Split, Croatia. In 66 cases (28%), physicians at various diving sites reached diagnosis with difficulty, and 86 divers (37%) came directly to the NMI without seeing a physician first. Physicians at remote diving locations frequently have only basic knowledge of diving medicine and are often inexperienced. The language barrier was a major obstacle in obtaining a medical history and examination of foreign divers. Consultations at the NMI proved a major contribution to correct diagnosis and treatment. We present six illustrative cases from NMI Archives that demonstrate how prejudices, panic, and inexperience could create problems in establishing DCS diagnosis. © 2003 IMSS. Published by Elsevier Science Inc.

Key Words: Diving, Decompression sickness, Differential diagnosis.

Introduction

Decompression sickness (DCS) is a specific health problem of caisson (watertight structure for underwater construction) workers and divers breathing compressed air or artificial gas mixture. The disease is precipitated by gas bubbles that remain in the body after a dive or pressure exposure, causing a variety of signs and symptoms ranging from pruritus to convulsions and death. More facts concerning signs and symptoms may be found in a classic work by Rivera (1). Traditionally, DCS is classified as follows: the less severe type 1 (that includes musculoskeletal, cutaneous, and lymphatic form, and malaise/anorexia/fatigue), and the more severe type 2 (that includes pulmonary and neurologic forms, and hemoconcentration and hypovolemic shock) (2).

Because recreational diving has become more popular and entry health criteria more lax, an increasing number of divers with minor or even major health problems could be expected in the future. Estimated number of divers in the U.S. at the beginning of the present decade could be in excess of 9 million (3). There were approximately 5,000 divers diving annually in Croatia in the early 1990s; at present, approximately 100,000 divers dive in the Croatian part of the Adriatic Sea each year, principally from June to September, the majority being foreign tourists (Lukas N, Croatian Diving Federation, personal communication). Based on the Divers Alert Network (DAN) 2002 Report, the number of injured divers has constantly increased since 1987 when DAN began reporting diving injuries and fatalities; in 2000, DAN received 1,042 reports on injured divers (4). More frequently, divers with various concomitant health problems and suspected DCS request help from diving physicians, placing them in a situation in which differential diagnosis of DCS is more difficult and might not be based solely or mainly on medical history. DCS symptoms vary to such a degree from case to case that it has been said that there is no DCS, but merely people who contract it. Of two divers diving together to the same depth with the same

*The opinions expressed herein are those of the authors and do not necessarily reflect the views of the Naval Medical Institute, the Croatian Navy, or the Ministry of Defense of the Republic of Croatia.

Address reprint requests to: Nadan M. Petri, M.D., Ph.D., Undersea and Hyperbaric Medicine Department, Naval Medical Institute, p/p 196 (HRM), Split, 21000, Croatia. Phone: (+385) (21) 464-543; FAX: (+385) (21) 353-739; E-mail: nadan.petri@morh.hr

PII S0188-4409(02)00458-7

time at sea bottom, identical ascent rate, and after same decompression, one diver could get severe DCS and even die while the other diver could be symptom free. At the NMI we had an experience exactly as described.

There are several excellent reviews on DCS (1,2,5–7) and hundreds of papers investigating this problem; thus, it might appear that everything has been said concerning the issue. However, we do not have sufficient knowledge to date concerning DCS pathophysiology, onset, and treatment. Symptom onset delay after surfacing might vary but in 95% of DCS cases, symptoms would manifest within 12 h (1,2,4–7).

Injury On-Site Physical Examination and DCS Differential Diagnostic Tests

Physical examination of injured divers includes defining objective DCS symptoms that are sometimes nonspecific. Edema might be a lymphatic system obstruction indication with inert gas bubbles, but also might be nonspecific. DCS signs may include skin rash and itching, a consequence of subdermal circulation obstruction. A diver with DCS type 1 usually has no sign of joint inflammatory reaction but merely a pain which, unlike in an inflammatory process, usually does not change its character and/or intensity during joint movements. If a tensiometer cuff is inflated over the painful joint, pain will usually cease at least for a while or will change its character, probably as a consequence of bubble shrinkage or of its being squeezed into another position. This diagnostic trick is neither mandatorily clear nor sufficiently sensitive in all injured divers. During neurologic examination, paresthetic areas might be found as well as abnormalities of urinary sphincter function (usually with urinary retention), spontaneous erections, vertigo, ataxia, and a myriad of other nervous system defects. One of the most sensitive signs of DCS type 2 is heel-toe test. A subject is asked to walk in a straight line with arms crossed over the chest and eyes closed, touching the toes of one foot with the heel of the other. If divers fail to perform as requested, they are suspected of having a neurologic lesion (cerebral and/or spinal, or inner ear). Once admitted to a hospital, a diver would probably be submitted to creatine-phosphokinase (CPK) and hematocrit testing (8,9). Increased CPK values are a consequence of muscular tissue microembolization. Increased hematocrit values are a consequence of endothelial damage and loss of fluid to interstitium.

If a physician must decide between DCS and carbon monoxide poisoning, carboxy-hemoglobin values should be determined, especially because carboxy-hemoglobin values often do not correlate with intoxication severity (10). If metabolic encephalopathy is suspected, blood glucose, neuroactive substance, or alcohol levels should be determined. Psychologic testing might be helpful in some situations in that changes in psychologic profile might be the only signs of DCS (11,12). In practice, a psychologist might be located miles away, and in addition a very limited number of diving medicine specialists would ask for any tests prior to recompression treatment. This might be a time-consuming effort; thus, the following rule was set forth: when in doubt, recompress. In our practice, we have never used any psychologic test to decide whether a diver should be submitted to recompression treatment.

Painful joints and neurologic manifestations occurring after a dive are usually easily connected with DCS, even in cases when diving was effected without apparent errors in decompression regime; therefore, post-dive onset of joint or muscle pain might be coincidental. Therefore, divers not responding well to initial recompression treatment should be reevaluated carefully. If symptoms that suggest DCS occur 6 h or later after the dive, additional reasons should also be taken into consideration. Understanding symptoms and medical histories in diving medicine is not always an easy task. An incorrect decision might lead to unnecessary engagement of numerous persons participating in first aid efforts, emergency helicopter transport, medical evaluation of divers, and recompression treatment. The objective of this paper was to determine the most common decision-making process errors by analyzing examples from diving medicine specialists' practice.

Materials and Methods

We analyzed data from the Archives of the Department of Undersea and Hyperbaric Medicine, Naval Medical Institute (NMI), Croatian Navy in Split, Croatia in search of the most illustrative cases to demonstrate the difficulty of differential DCS diagnosis.

Results

A total of 232 divers with DCS were treated at the NMI from 1967 to 2001. In only one case was a diver sent for recompression treatment with incorrect diagnosis. In 66 cases (28%), physicians at various diving sites reached diagnosis with difficulty and after prior consultations with the NMI. Eighty-six divers (37%) came directly to the NMI without consulting a physician previously. Symptoms occurred within 12 h of surfacing in 213 (92%) divers.

Case Reports

Case 1. The patient was a 43-year-old male professional diver from Croatia who regularly dives to 40 m, sometimes deeper, using compressed air. He arrived 24 h after a dive within no-decompression limits (27 m/30 min), which according to his report, he effected without difficulties. During the dive, the diver worked hard on underwater construction and reportedly hurt his right elbow. Onset of first painful symptoms occurred approximately 6 h post-dive.

The pain gradually increased. The diver mentioned that he had similar difficulties previously and that pain would always disappear after a few days. He complained of constant pain in the right elbow. There were no visible external signs of injury and no signs of pathologic process were found on X-rays. During cuff inflation test, pain intensity and character did not change. U.S. Navy (USN) Treatment Table 6 (TT6) was chosen for initial recompression treatment (14). After a few minutes on oxygen at 2.8 bars, the diver complained of worsening pain. Paradoxically, this actually occurs in some divers with DCS while breathing oxygen during recompression treatment and is described as pain-under-pressure. This pain is attributed to shrinkage of bubbles and to periost returning to normal position. All problems vanished after 40 min of oxygen breathing. The patient did not experience pain after initial treatment termination and did not complain of additional problems. Diagnosis: type 1 DCS.

Case 2. A 22-year-old female recreational diver from Austria was a recent graduate from a dive course. On admission at the NMI, the patient complained of difficulties in walking, slight-to-moderate vertigo, and paresthesia of inner part of upper legs. Symptoms began approximately 12 h after what the diver described as a somewhat faster ascent from a 10-min, no-decompression dive to 36 m. She was first seen by the attending physician on a nearby island who, during phone consultation, stated that the case was suspicious of hypoglycemia in that the patient had diabetes.

The patient also disclosed she had consumed alcohol during the previous evening. At admission, we found blood glucose within normal limits but both creatine phosphokinase (CPK) and hematocrit values increased. TT6 was immediately administered together with ample rehydration by intravenous (i.v.) normal saline. The diver's condition improved dramatically after first oxygen cycle at 2.8 bars. After initial treatment, the patient complained of sensation loss in lower abdomen. During the subsequent 5 days, our standard (60 min at 2.2 bars) hyperbaric oxygenation (HBO_2) was administered once daily. On the sixth day, the patient was transferred to the recompression chamber in Graz, Austria for further treatment. Diagnosis was type 2 DCS.

Case 3. A 34-year-old male recreational diver from the Czech Republic dove to 44 m, total bottom time 24 min, 72 h previous to admission at the NMI. During ascent, he followed standard USN air decompression tables but admitted that ascent to first in-water stop was appreciably more rapid. From ascent to surface until admission, the patient had constant problems with urination and vertigo. He also had slight difficulty in walking, which worsened hourly. The patient stated that he did not believe he had DCS, that all problems were most probably a consequence of a party he attended the night before the dive, and that he may have contracted food poisoning. TT6 was commenced immediately. Because neurologic status did not improve during the first 20 min of oxygen breathing time, the hyperbaric chamber physician decided to add two extensions to standard TT6: the first at 2.8 bars for 20 min, and the second at 1.9 bars lasting 60 min. After initial treatment, the diver experienced only slight instability in walking. This problem was completely solved during the following week when our standard HBO_2 was administered once per day. Patient diagnosis was type 2 DCS.

Case 4. This case involved a 32-year-old Italian male recreational scuba diver diving to 38 m and taking pictures; total bottom time was 40 min. The patient stated he was well informed of all diving medicine problems and therefore drank ample amounts of juice prior to and after the dive to prevent possible dehydration, a known risk factor for DCS (15). The patient and his diving buddy ascended observing USN standard air decompression tables. Approximately 8 h after the dive, he experienced itching on his back and extreme fatigue, although there was no objective reason for his feeling that tired; the other diver had no problems. The doctor on the island did not suspect DCS, although the diver did. At admission, increased CPK and hematocrit values were found; initial treatment was provided using TT6. During initial minutes of breathing oxygen at 2.8 bars, the condition of the diver improved considerably. After completion of TT6, the patient reported feeling completely well. His diagnosis was type 1 DCS.

Case 5. A 37-year-old German female recreational diver ascended in panic due to equipment problems after approximately 2 min at 37 m. Immediately upon ascent, the patient felt a strong abdominal pain. The attending physician on the island suspected DCS; thus, the patient was transported by helicopter directly to the NMI. During transport, the diver breathed 100% oxygen via close-fitting face mask, indeed an important measure in case of DCS (13). Although no signs of DCS were found at admission, her panicked diver companions insisted on immediate recompression treatment. Instead, the patient was referred to the X-ray department because of painful and tense abdomen. Abdominal X-rays showed ample amount of free air; therefore, the patient was transferred to an abdominal surgeon due to suspected stomach rupture. At surgery, a rupture of the lesser curvature that measured approximately 4 cm was found and sutured. This rare case was more extensively covered in a recent report (16). Patient diagnosis was barotraumatic stomach rupture.

Case 6. A 53-year-old Hungarian male scientific diver dove using air-filled scuba gear to 60 m for approximately 10 min as a member of an international diving expedition to a deep-sea shipwreck. Approximately 12–18 h after the dive, the diver began to feel strong pain in both knees and was unable to urinate or to walk unassisted. Prior to NMI admission, he was seen by three international physicians; only the fourth physician gave an exact diagnosis and in-

sisted on consultations with our emergency intervention team. The patient was transported immediately via helicopter with no treatment during transport. TT6 was initiated immediately, but no improvement occurred although TT6 was extended twice, first at 2.8 bars for 20 min and second at 1.9 bars during 60 min. On the following day, TT5 was administered twice. During the subsequent 10 days, our standard HBO_2 was administered once daily. Patient's walking ability recovered completely between the fifth and sixth days of treatment and urinary sphincter function returned on the day prior to discharge. Six years after the incident, the diver has no major difficulties but only minor upper-leg sensitivity loss. Patient diagnosis was type 2 DCS.

Discussion

DCS incidence in the diving community is generally low, ranging from 0.013 to 1.25% (17–19); nonetheless, one should bear in mind that divers for various reasons frequently tend to tolerate (i.e., tough-out) minor symptoms. In sport divers, type 2 is far more frequent than type 1 DCS, comprising up to 80% of reported cases. In commercial and military diving, type 1 DCS occurs in 86% symptomatic decompressions (5). DCS incidence depends on numerous risk factors (15), but most important are basic determinants of decompression stress, including depth, bottom time, and ascent rate (1–7). However, DCS importance lies not only in its incidence but also in the fact that it might be a cause of permanent invalidism and in rare cases even of fatal outcome. Thus, even minor symptoms require urgent recompression treatment. DCS is not a rare event in Croatia at present because an increasing number of divers visit the country, dive deeper, and extend bottom times using artificial gas mixtures.

Dive computers permit decompressions shorter than required according to USN Air Decompression Tables. Currently, conservative standard USN Air Decompression Tables, generally considered safe, are used infrequently in Croatia. According to the 2002 DAN Report, proportion of injured divers using dive computers (73% in 2000) continues to increase in number over previous years, although this may reflect continued growth in dive computer popularity rather than increased risk concerning dive tables (4). However, it was estimated that use of 40 feet of sea water (fsw)/1.2 bars for 200-min schedule in decompression computer is likely to result in DCS incidence 2.5- to 70-fold greater than that observed in USN diving using table-based procedures (18).

Some cases of DCS represent an intriguing differential diagnostic problem even for experienced diving medicine specialists, a puzzle in many situations at the diving accident site. At times, it is difficult to relate existing symptoms with diving because DCS symptom onset might occur with significant delay (2,4–7). DCS symptoms at times mimic (Table 1) other conditions that could scarcely be attributed to diving in that at least at first glance no errors in decompression profile were committed or were any clear DCS risk factors present during a particular dive. To describe the latter situation, the terms undeserved, paradox, or unexpected are used. Principally, in some 60% of cases DCS manifests as the bends, a condition similar to nonspecific rheumatoid problems (5). A diver, whether consciously or unconsciously, misleads the attending physician to arrive at an incorrect conclusion because the majority of divers experience a type of guilt concerning DCS and know that they will have to face frustrating reactions from fellow divers. Questions such as "Were you diving?" will be more frequent in the future at primary health care institutions and at emergency hospital admission departments; nevertheless, although the answer may be affirmative, DCS diagnosis would not yet be established. Physicians at remote maritime locations frequently have only basic knowledge concerning diving medicine. Incorrect presentation of symptoms by injured divers might contribute to major errors during consultations with a recompression center. It appears that DCS, although extremely rarely, might also occur in breath-hold diving, but this is not important for practical considerations (20).

In cases 1 and 2, DCS occurred after no-decompression dives (27 m/30 min and 36 m/10 min, respectively), which

Table 1. Conditions that might mimic decompression sickness (13)[a]

- Arterial cerebral gas embolism due to pulmonary barotrauma
- Contaminated breathing gas (such as CO and other gases)
- Near-drowning and hypoxic brain injury
- Seafood toxin poisoning
- Migraine
- Guillain-Barré syndrome
- Porphyria
- Multiple sclerosis
- Transverse myelitis
- Different-origin spinal cord compression
- Inner ear barotrauma
- Unrelated seizure (hypoglycemia, epilepsy)
- Cerebral stroke
- Subarachnoid hemorrhage
- Acute myocardial infarction
- Pulmonary edema induced by cold water immersion
- Different origin functional abnormalities
- Pneumothorax
- Stomach or intestinal rupture due to rapid ascent
- Unrelated abdominal cramps
- Side effects or drug poisoning

[a]Adapted from Moon RE. Treatment of decompression sickness and arterial gas embolism. In: Bove AA, editor. Diving medicine. 3rd ed. Philadelphia, PA: W.B. Saunders;1997. pp. 184–204.

might seem unusual; however, in both cases risk factors (hard work, rapid ascent) provoked the disease. In five of six cases presented, divers were not Croatian citizens and spoke only their native language and very little English; thus, the language barrier appeared to be a major problem while taking medical histories and during examinations. In case 1, the diver emphasized injury to the elbow. If his statement was taken as rendered, DCS would have remained undiscovered with all consequences that occur in untreated cases (13,21). In case 2, symptoms could have been attributed to diabetes and important spinal cord injury could have been omitted. In case 3, alcoholic intoxication and perhaps food poisoning could have been understood as the cause of the problem. In case 4, nonspecific itching could have been diagnosed rather than DCS. Waiting for other clearer symptoms to develop might have lead to type 2 DCS development. An estimated 20–30% of untreated type 1 DCS cases might progress to type 2 DCS (22). In case 5, if DCS was diagnosed and recompression treatment initiated, acute abdomen might have developed during chamber treatment, leading to a much more complicated situation (23). In case 6, if the NMI was not consulted the diver could have been understood as a fatigued, elderly man who simply needed rest.

Onset of DCS signs and symptoms in our group (92% within 12 h) was basically similar to those in previous reports (1–7), but distribution of signs and symptoms in our group (Table 2) varied slightly from a distribution reported previously. Rivera described localized pain in 91.8%, numbness or paresthesia in 21.2%, muscular weakness in 20.6%, skin rash in 14.9%, dizziness or vertigo in 8.5%, paralysis in 6.1%, nausea in 7.9%, and urinary disturbances in 2.5%, respectively (1).

In that only correct diagnosis and immediate recompression treatment ensure complete cure for the majority of injured divers, it is useful to be familiar with most common differential diagnostic problems in DCS and with measures to be taken at site of injury and during transportation to recompression chamber. In our practice, in-servicing consultations provided to physicians at remote locations proved to be valuable aids in understanding certain cases of DSC.

Table 2. Distribution of signs and symptoms in divers ($n = 232$) treated due to decompression sickness at the Naval Medical Institute, Split, Croatia, 1967–2001

Sign or symptom	Number of cases (%)
Localized pain	204 (88%)
Numbness and/or paresthesia	61 (26%)
Muscular weakness	53 (23%)
Skin rash	18 (8%)
Vertigo	18 (8%)
Paralysis	16 (7%)
Nausea	14 (6%)
Urinary disturbances	9 (4%)
Other	<1%

References

1. Rivera JC. Decompression sickness amongst divers: an analysis of 935 cases. Mil Med 1963;129:314–334.
2. Elliott DH, Kindwall EP. Manifestations of the decompression disorders. In: Bennet PB, Elliott DH, editors. The physiology and medicine of diving. London: Bailliere-Tindall;1982. pp. 461–472.
3. Newton HB. Neurologic complications of scuba diving. Am Fam Physician 2001;63:2211–2218.
4. Anon. Report on diving accidents and facilities. Durham, NC, USA: Divers Alert Network;2002.
5. Dutka AJ, Francis TJ. Pathophysiology of decompression sickness. In: Bove AA, editor. Diving medicine. 3rd ed. Philadelphia, PA, USA: W.B. Saunders;1997. pp. 159–175.
6. Edmonds C, Lowry C, Pennefather J. Decompression sickness: clinical manifestations. In: Diving and subaquatic medicine. 3rd ed. Oxford, UK: Butterworth-Heinemann;1992. pp. 159–178.
7. Elliott RE, Moon RE. Manifestations of the decompression disorders. In: Bennett PB, Elliott DH, editors. The physiology and medicine of diving. 4th ed. London: WB Saunders;1993. pp. 481–505.
8. Smith RM, Neuman TS. Elevation of serum creatine kinase in divers with arterial gas embolization. N Engl J Med 1994;330:19–24.
9. Bruner F, Frick P, Buhlmann A. Post-decompression shock due to extravasation of plasma. Lancet 1964;1:1071–1073.
10. Jain KK. Carbon monoxide and other tissue poisons. In: Textbook of hyperbaric medicine. Seattle, WA, USA: Hogrefe and Huber, Publishers:1996. pp. 146–177.
11. Curley MD, Schwartz MJC, Zwingelberg KM. Neuropsychological assessment of cerebral decompression sickness and gas embolism. Undersea Biomed Res 1988;15:223–236.
12. Curely MD, Amerson TL. The using of psychometric testing in decompression illness. In: Moon RE, Sheffield PJ, editors. Treatment of decompression illness. Kensington, MD, USA: Undersea and Hyperbaric Medical Society;1996. pp. 152–162.
13. Moon RE. Treatment of decompression sickness and arterial gas embolism. In: Bove AA, editor. Diving medicine. 3rd ed. Philadelphia, PA, USA: WB Saunders;1997. pp. 184–204.
14. Anon. Diving medicine. In: United States Navy diving manual. Vol. 5. Washington D.C., USA: Naval Sea Systems Command;1999.
15. Gorman DF, Pearce A, Webb RK. Dysbaric illness treated at the Royal Adelaide Hospital 1987: a factorial analysis. South Pac Underwater Med Soc J 1988;18:95–101.
16. Petri NM, Vranjkovic-Petri L, Aras N, Druzijanic N. Gastric rupture due to rapid ascent. Croat Med J 2002;1:42–44.
17. Arness MK. Scuba decompression illness and diving fatalities in an overseas military community. Aviat Space Environ Med 1997;4:325–333.
18. Ball R, Parker EC. A trial to determine the risk of decompression sickness after a 40 feet of seawater for 200 minute no-stop air dive. Aviat Space Environ Med 2000;71:102–108.
19. Berghage TE, Durman D. US Navy air decompression table and risk analysis. Naval Medical Research Institute, Tech. Rep. 80–1, 1980.
20. Hong SK. Breath-hold diving. In: Bove AA, editor. Diving medicine. 3rd ed. Philadelphia, PA, USA: WB Saunders;1997. pp. 65–74.
21. Dromsky DM, Toner CB, Survanshi S, Fahlman A, Parker E, Weathersby P. Natural history of severe decompression sickness after rapid ascent from air saturation in a porcine model. J Appl Physiol 2000;2:791–798.
22. Davis JC. Treatment of decompression sickness and arterial gas embolism. In: Bove AA, Davis JC, editors. Diving medicine. Philadelphia, PA, USA: WB Saunders;1990. pp. 249–260.
23. Molenat FA, Boussuges AH. Rupture of the stomach complicating diving accidents. Undersea Hyperbaric Med 1995;22:87–96.