Case report

Cutaneous decompression sickness

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Abstract

(Tasios K, Sidiras GG, Kalentzos V, Pyrpasopoulou A. Cutaneous decompression sickness. *Diving and Hyperbaric Medicine*. 2014 March;44(1):45-47.)

A probable case of decompression illness with associated *cutis marmorata* is presented, which regressed over a few hours with oxygen breathing and after intravenous methylprednisolone and fluid resuscitation without recompression. He was eventually transferred for hyperbaric treatment some 10 hours post dive. Cutaneous decompression illness is not associated with high mortality per se, but prompt and accurate recognition is warranted, as it may represent a prodromal feature of potentially life-threatening complications. However, in this case, as differential diagnosis, an allergic reaction remains possible.

Key words

Decompression sickness, decompression illness, allergy, first aid, oxygen, treatment

Introduction

Decompression illness (DCI) is a major complication of diving. It is caused by intravascular and/or extravascular bubbles that are formed as a result of a reduction in environmental pressure (decompression). Severity of the syndrome may vary, with manifestations ranging from arthralgias and skin rashes to paralysis and death. Due to its rarity, experience of the emergency room physician in the recognition and treatment of this syndrome is limited. Cutaneous manifestations, when present, are usually a transient feature of the disease and thus rarely captured. We report a diver with prominent skin manifestations typical of decompression sickness (DCS).

Case report

A 52-year-old male patient was brought to the emergency department with circulatory collapse and confusion following the ascent after diving. The patient had remained at 18 metres' sea water (msw) depth for a total of 1.5 hours, with intermittent ascents to the surface about every 20–30 min. His medical history was unremarkable apart from mild chronic obstructive pulmonary disease, which he had developed possibly secondary to diving.

The patient was found by the notified medical team on his sailing boat; he was confused, with severe hypotension (systolic blood pressure 60 mmHg). Inhaled oxygen and intravenous fluid administration were initiated. The patient's state of consciousness improved rapidly, without obvious neurological deficit. On presentation in the emergency department, the patient was fully alert and orientated but remained hypotensive (BP 105/80 mmHg) and was anuric. Other vital signs were normal (temperature 36.0^{O}C, oxygen saturation 97% on 100% inhaled O_2). The patient's skin was remarkable for *cutis marmorata* ('marble skin') of the torso, as well as the thighs and knees (Figures 1 and 2) and a fine, confluent macular rash of the upper extremities. The patient did not report any fish bite or any perceived sting.

Bolus methylprednisolone (125 mg) and 2 L normal saline (over 4 hours) were administered intravenously. The patient gradually improved haemodynamically, urination was restored, and the skin rash regressed over 3 hours. Mild leukocytosis (total white blood cell count 15,500 μl^{-1}, 86.6% neutrophils) was noted, but haematological and biochemical profiles were otherwise normal. After communication with a specialized hyperbaric unit, the patient was transferred for further evaluation and management.

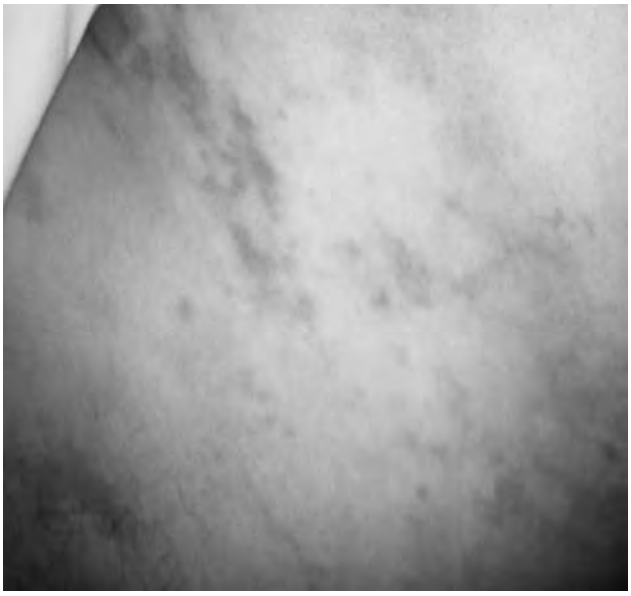
The patient arrived at the Diving and Hyperbaric Medicine Unit (DHMU) approximately 10 hours post dive. He had a mild recurrence of the rash on his trunk, could not fully control his bladder, and his white blood cells remained elevated (18,280 μl^{-1}, 94.3% neutrophils). He received five hyperbaric oxygen treatments (HBOT) over four days (1st: 200 kPa/60 min – 180 kPa/60 min – 150 kPa/60 min, 2nd: 200 kPa/20 min – 180 kPa/50 min – 150 kPa/10 min, 3rd: 180 kPa/70 min, 4th and 5th: 200 kPa/60 min – 180 kPa/15 min – 150 kPa/10 min) and had complete regression of his symptoms and haematological values.

Discussion

The estimated number of injured divers who need recompression treatment in European hyperbaric facilities varies between 10 and 100 per year per facility depending on the number of divers in the population, number of dives performed annually, and the number of hyperbaric centres in the country.[1] Because of its rarity, the experience of the emergency doctor in the recognition and treatment of this

Figure 1
Cutis marmorata of the right groin and upper thigh

Figure 2
Cutis marmorata of the right thigh



syndrome is often limited. Decompression illness is caused by intravascular and/or extravascular bubbles that are formed as a result of reduction in environmental pressure (decompression) in situ and the introduction of gas bubbles into the arterial system via intra- or extra-cardiac shunts.[2] It usually presents in the context of underwater diving but may be experienced in other depressurisation events, such as in caisson workers, flying in unpressurised aircraft, and extra-vehicular activity from spacecraft.[3] Severity of the syndrome may vary, with manifestations ranging from arthralgias and rashes to paralysis and death, and appears to be inversely related to the time interval to the manifestation of symptoms.[4] Onset of symptomatology in the vast majority of cases occurs within 6 h after surfacing.[5]

First-aid treatment of decompression illness includes breathing 100% O_2, intravenous fluid administration, and transfer for hyperbaric treatment.[6] Adjunctive treatments (non-steroidal anti-inflammatory agents and the use of steroids) have been tested but their benefit remains to be proven.[7] In a recent study to determine the potential risk factors associated with the development of severe diving-related spinal cord decompression illness, the time to recompression and the choice of initial hyperbaric procedure did not appear to significantly influence recovery; however, clinical symptoms of spinal cord decompression syndrome and their initial course before admission to the hyperbaric centre were identified as major prognostic factors in recovery.[8]

Cutaneous manifestations of decompression sickness are usually a transient feature of the disease and thus rarely captured. They do not appear to be directly related to the severity of the syndrome; however, prompt and accurate recognition is important, as they may represent a prodromal feature of potentially life-threatening complications.[9,10] Most divers who suffer cutaneous decompression illness also have a right-to-left shunt. The shunt is usually across a patent foramen ovale, but some have pulmonary shunts.[11] Skin manifestations typically include erythema accompanied by pruritus; the rash spreads irregularly and deepens in color, developing a mottled appearance, with areas of pallor surrounded by cyanotic patches (*cutis marmorata*).[12] Analogous lesions in pigs revealed abnormalities in 20 of 22 animals, mainly vascular congestion, focal areas of vasculitis, perivascular neutrophil infiltrates, oedema and occasional haemorrhage.[13]

In this case, the possibility of an allergic reaction cannot be excluded. However, the severity of the symptomatology (including neurological signs) compared to the small dose of corticosteroids administered, the gradual restoration of haemodynamic stability and the nature of the rash, which rather resembled areas of impaired perfusion, rendered the initial diagnosis of DCI more likely. The management in our case followed the DHMU guidelines.[14] DHMU is considered the national centre for diving accidents in Greece. Each treatment is individualised, commonly developed on site by the hyperbaric physician on duty. The choice of duration, pressure, breathing mixtures, intravenous medications, and fluid replacement depends on various factors, including the clinical manifestations.[14] HBOT was ceased one day after our patient showed no further improvement.

References

1 Kot J, Sićko Z, Michałkiewicz M, Lizak E, Góralczyk P. Recompression treatment for decompression illness: 5-year report (2003–2007) from National Centre for Hyperbaric Medicine in Poland. *Int Marit Health*. 2008;59:69-80.

2 Vann RD, Butler FK, Mitchell SJ, Moon RE. Decompression illness. *Lancet*. 2011;377:153-64. doi: 10.1016/S0140-6736(10)61085-9.
3 Butler WP. Epidemic decompression sickness: case report, literature review, and clinical commentary. *Aviat Space Environ Med*. 2002;73:798-804.
4 Türkmen N, Akan O, Cetin S, Eren B, Gürses MS, Gündoğmuş UN. Scuba diver deaths due to air embolism: two case reports. *Soud Lek*. 2013;58:26-8.
5 Xu W, Liu W, Huang G, Zou Z, Cai Z, Xu W. Decompression illness: clinical aspects of 5278 consecutive cases treated in a single hyperbaric unit. *PLoS One*. 2012;7:e50079. doi: 10.1371/journal.pone.0050079. Epub 2012 Nov 21.
6 Antonelli C, Franchi F, Della Marta ME, Carinci A, Sbrana G, Tanasi P, et al. Guiding principles in choosing a therapeutic table for DCI hyperbaric therapy. *Minerva Anestesiol*. 2009;75:151-61.
7 Bennett MH, Lehm JP, Mitchell SJ, Wasiak J. Recompression and adjunctive therapy for decompression illness. *Cochrane Database of Systematic Reviews*. 2012. May 16;5:CD005277. doi: 10.1002/14651858.CD005277.pub3.
8 Blatteau JE, Gempp E, Simon O, Coulange M, Delafosse B, Souday V, et al. Clinical symptoms of spinal cord DCS and their initial course before admission to the hyperbaric center should be considered as major prognostic factors in recovery. *Neurocrit Care*. 2011;15:120-7.
9 Ozyigit T, Egi SM, Denoble P, Balestra C, Aydin S, Vann R, et al. Decompression illness medically reported by hyperbaric treatment facilities: cluster analysis of 1929 cases. *Aviat Space Environ Med*. 2010;81:3-7.
10 Bledsoe BE, Loptien M, Berkeley RP. A desert rash. *West J Emerg Med*. 2011;12:563-4. doi: 10.5811/westjem.2010.9.2075
11 Wilmshurst PT, Pearson MJ, Walsh KP, Morrison WL, Bryson P. Relationship between right-to-left shunts and cutaneous decompression illness. *Clin Sci* (Lond). 2001;100:539-42.
12 Kalentzos VN. Images in clinical medicine. *Cutis marmorata* in decompression sickness. *N Engl J Med*. 2010;362:e67.
13 Buttolph TB, Dick EJ Jr, Toner CB, Broome JR, Williams R, Kang YH, Wilt NL Cutaneous lesions in swine after decompression: histopathology and ultrastructure. *Undersea Hyperb Med*. 1998;25:115-21.
14 Sidiras G. Management of diving accidents using hyperbaric oxygenation. *Polish Journal of Emergency Medicine*. 2008;1:19-25.

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