DELAYED TREATMENT OF DECOMPRESSION SICKNESS TYPE II RESPONSIVE TO HYPERBARIC OXYGEN

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ABSTRACT

A 38-years-old recreational female diver presented at our hospital with fatigue, dizziness, cutis marmorata, swollen and painful ankles, back pain and pulmonary symptoms. The patient was diving in the Maldives, with series of daily dives mostly around 22m, each dive decompression made on 6m with duration of 3 minutes. After more than 30 hours from her last dive, she took an international flight Maldives – North Macedonia that lasted for about 12 hours. Her first checkup was more than 80 hours of the onset of symptoms. Neurological examination, electrocardiogram, chest X-ray and lung ultrasound were normal. Laboratory data only showed evidence of thrombocytopenia and elevated D-dimer. Decompression sickness type II was diagnosed according to history and physical examination. Recompression treatment with hyperbaric oxygen (HBO_2) was immediately started (2.8 ATA on 100% oxygen over a period of 90 minutes) and additional session at the same day with a duration of 70 minutes to 2.4 ATA. Additional courses with HBO_2 were required daily in the next 7 days to 2.4 ATA. The most of the patient's symptoms were resolved after the third therapy with HBO_2, and the cough after the 7th therapy. The patient was released from hospital after resolution of the symptoms. **Conclusion:** Cutis marmorata is often associated with more serious manifestations of decompression sickness. Treatment with HBO_2 is the cornerstone therapy for DCS. Indeed, treatment with hyperbaric oxygen can be successful even in cases with delayed start.

Key Words: c*utis marmorata, decompression sickness, diving, hyperbaric oxygen.*

Introduction

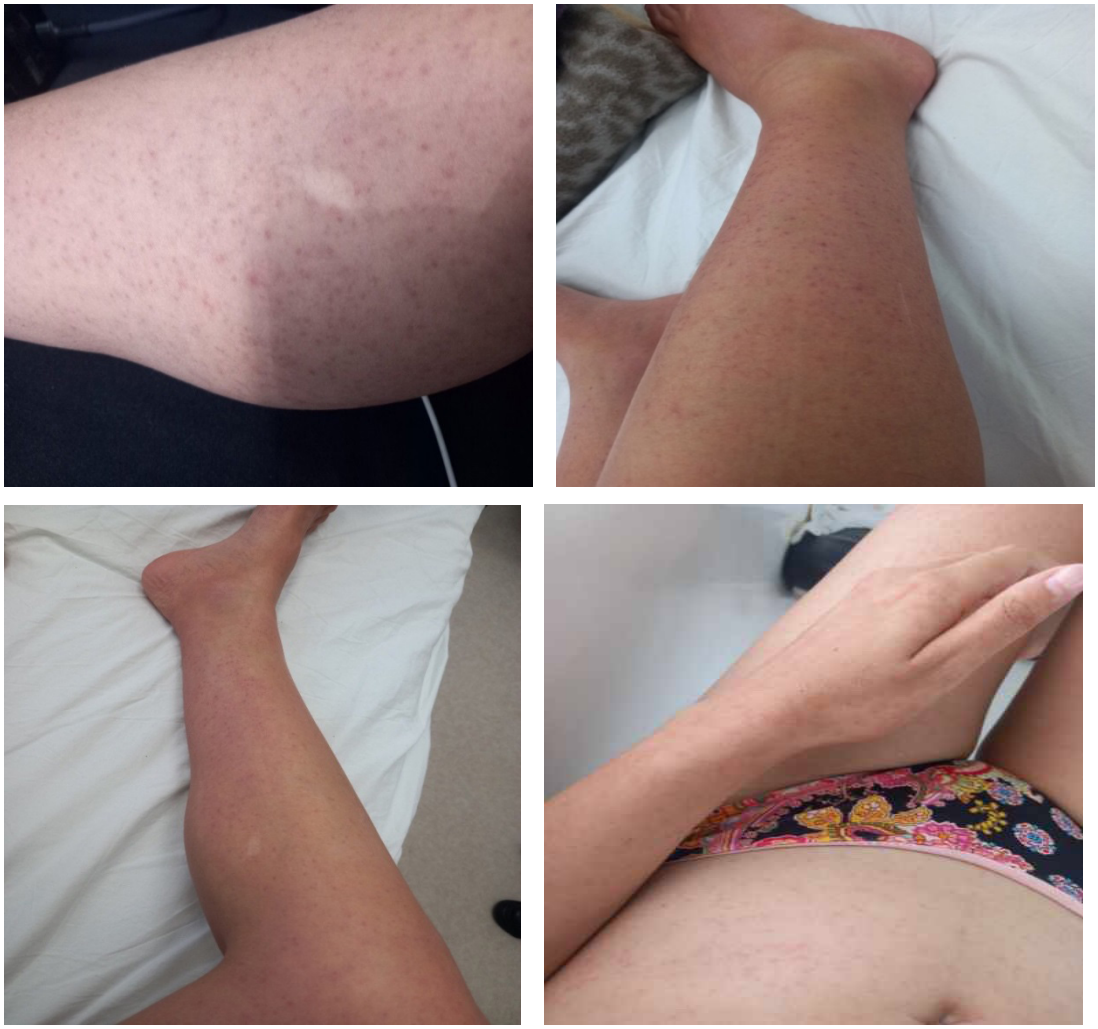
A major risk in scuba diving is the exposure to changes in pressure when diver ascends to the surface too quickly. Diving has become an increasingly popular sport and previously very rare complications, such as Decompression sickness, are becoming more common (1). The development of bubbles in tissues and/ or blood when the total pressure of the dissolved gas exceeds the ambient pressure, results in decompression sickness (also known as "bends"). Mild altitude exposure, like that from a flight in a commercial aircraft, can cause DCS after a dive (2). Decompression illness was first described in mid-1840 during the construction of the Brooklyn Bridge, when construction workers were put in pressurized boxes (caissons) used underwater. The condition was referred to as "the bends" because when workers returned from the compressed atmosphere of the caisson to air pressure, they walked with bent appearance from the joint pain. Later, DCI was divided into two categories: arterial gas embolism (AGE)

and decompression sickness (DCS) (1). The factors have been discovered that affect the risk of decompression sickness like altitude or time since the previous dive. Dehydration, tiredness, poor physical condition, having a higher body mass index, being older, and having a right to left shunt such a patent foramen ovale are additional risk factors for decompression illness (3). Symptoms of decompression sickness, which can range in severity from mild to severe, can include pain, skin changes, neurologic symptoms and cardiopulmonary symptoms (1,3). DCS is typically divided into two types: type 1, which refers to skin changes and less severe symptoms including joint discomfort, and type 2, which refers to symptoms that are more severe and affect the neurologic, cardiac and pulmonary systems (3). The majority of symptoms surface within 24 hours, unless there is an additional decompression (for example, altitude exposure), with severe symptoms typically appearing one to three hours after decompression (2). An incidence of 1.5 to 10 per 10,000 dives is seen among commercial divers. The duration of the dive and depth will determine this (4). Males are 2.5 times as likely as females to get DCS (4). Diagnosis of DCS is clinical, based on careful evaluation, clinical examination and historical data (1,2). The cornerstone of this condition's therapy is hyperbaric oxygen (HBO_2) (2). The aim of this case report is to present the first case of DCS type II described in our country that was presented in the hospital nearly 4 days after the onset of symptoms and underwent successful recompression treatment of DCS with hyperbaric oxygen in a multiple chamber.

Case Presentation

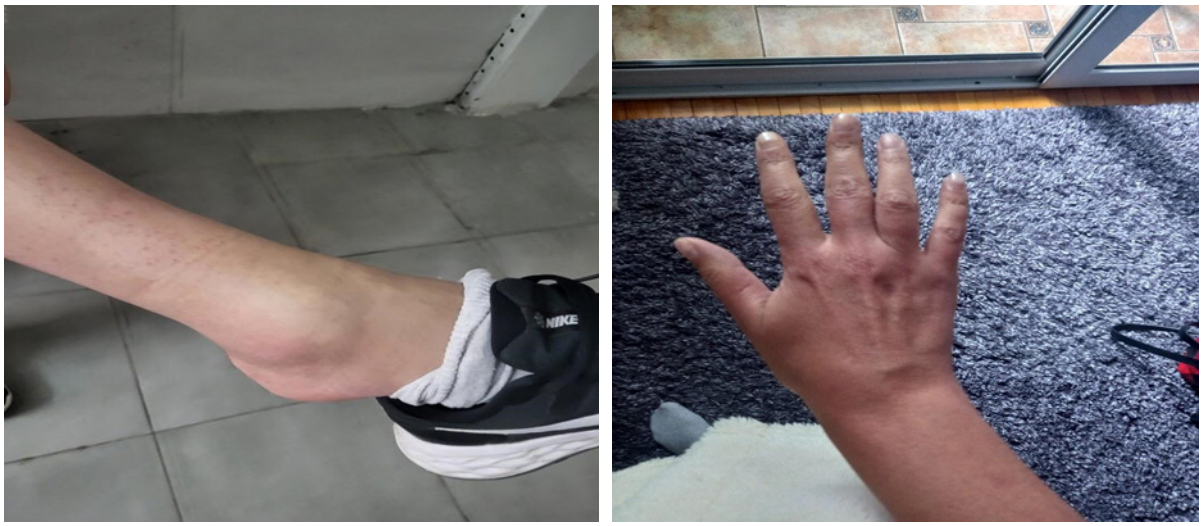
We describe a case of 38–years-old female recreational diver with no prior medical history, no family history either. She is a smoker, weighing 57kg, BMI 19.3kg/m^2 with no previous history of diving accidents. She had a sum of 35 dives during the 10-years period. While on vacation in the Maldives, she went diving every day for 8 days. This consisted of a daily dive schedule of two repetitive dives with duration of 40-45 minutes each with a 45minutes pause between them. The dives typically occurred at a depth of 22 meters (each dive with 3 minutes safety stop on 6 meters). She used a dive computer, and appropriate decompression schedules were followed while diving with tank, using enriched air nitrox (EAN). The last day of diving she made two repetitive dives with duration of 40-45 minutes each with a 45-minute pause between them. Approximately 20 minutes after surfacing, she noticed a mottled rash covering her face and body, which she thought were due to sunburn. She used some topic solution with no effect. The rash was followed by fatigue, discomfort and malaise, that prevented her from diving again the next two days before taking a long international flight to come back home, but she continued with her daily activities like boat floating. After more than 30 hours from her last dive, she took a flight from Maldives to North Macedonia that lasted for about 12 hours, and which include one transfer. When she landed (the third day of onset of symptoms), she noticed swelling of the ankles, especially the right one, which she thought was due to long fight. The next day (4^{th} day of onset of symptoms) when she woke up, she had swollen and painful elbows and hands, chest pain and cough. More than 80 hours after the start of her symptoms, she went for her first checkup at the University Clinic of Plastic and Reconstructive Surgery, where she complained of fatigue, dizziness, malaise, swollen and painful ankles and elbows, back pain, dyspnea, an intense irritating cough and chest pain. During the initial medical examination, she was conscious, oriented, afebrile, normotensive, with purpuric-violaceus macular rash on her face, trunk and limbs (Figure 1 - a, b, c, d - cutis marmorata).

Figure 1.- a, b, c, d. *Cutis marmorata in 38-years-old woman after diving.*



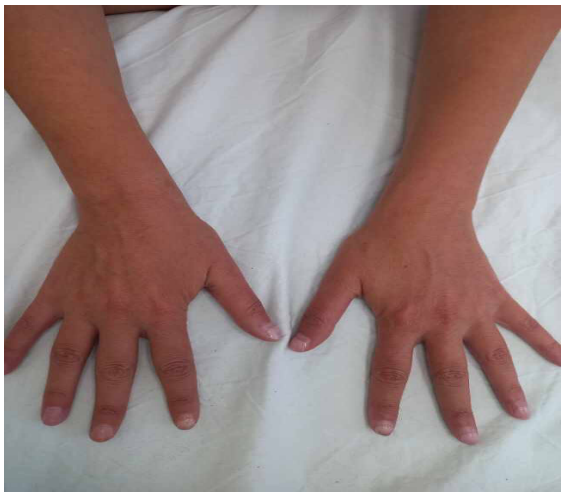
On auscultation of the lung breath sounds were vesicular. Abdomen was soft and non-tender. The neurological examination showed normal cranial nerve function. Motor and sensory functions were normal. Romberg sign was negative. Her elbows and ankles were swollen, painful, with restriction in movement (Figure 2 - a, b).

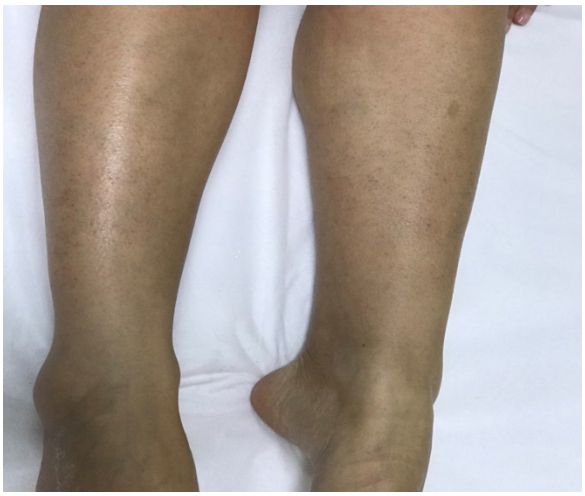
Figure 2 - a, b. *Swollen ankle and elbow due to decompression sickness***.**



Vital signs were within normal limits: blood pressure 120/80mmHg, heart rate 56/minutes, saturation O_2-97%. Electrocardiogram showed sinus rhythm, with normal p wave, QRS complex and T wave. An arterial gas blood demonstrated pH 7.41, pCO_2 41.3mmHg, pO_2 96.4mmHg, carboxyhemoglobin 2.1%, methemoglobin 1.1%, lactate 0.9mmol/L, bicarbonate 25.7mmol/L, base deficit 1.7mmol/l; On admission laboratory data showed evidence of thrombocytopenia 89 10^9/L (referent values 150-450) and elevated D-dimer 884.16ng/ml (referent values <500). The rest of the findings were within normal range: RBC 4.58 10^{12}/L, HGB 145g/l, WBC 5.3 10^9/l, Ne 57.5%, CRP 0mg/l, myoglobin 18.2ng/ml, Na 135mmol/l, K 4.17mmol/l. No abnormalities were found on chest X-ray and lung ultrasound. Based on the clinical presentation, dive history and medical examination a diagnosis of type II Decompression sickness was taken into consideration. Recompression treatment in multiple chamber was immediately started of pressuring to 2.8 ATA initially according to the US Navy treatment table on 100% oxygen over a period of 90 minutes and second session with hyperbaric oxygen at the same day with a duration of 70 minutes to 2.4 ATA. Additional courses were required daily in the next 7 days with 2.4 ATA over a period of 70 minutes. Crystalloids, non-steroid anti-inflammatory drugs and treatment with low molecular weight heparin (Enoxaparin sodium) were carried out in the first three days. The patient underwent a total of 9 hyperbaric chamber treatments over 8 days. The patient's symptoms like chest pain, back pain, dyspnea, rash and swelling of the ankles and elbows resolved after the third therapy with hyperbaric oxygen (Figure 3 - a, b).

Figure 3 - a, b. *After treatment with hyperbaric oxygen (after third therapy).*





The cough resolved after the 7th therapy. The patient was released from hospital after resolution of the symptoms. Control laboratory findings made on the sixth day of treatment showed normal platelet count and D-dimer 212ng/ml. An outpatient echocardiography about evaluation for PFO was suggested, but the patient refused to conduct the diagnostic procedure. We advised no diving in the next two months to the patient. A month later, she reported being healthy and experiencing no new symptoms during a phone call.

Differential diagnosis:

- Myocardial infarction (normal ECG), no history of angina, no risk factors;
- Pneumothorax, Bronchopneumonia-afebrile, no abnormalities on x-ray of the lungs;
- Thrombotic thrombocytopenic purpura (no fever, no hemolytic anemia, no kidney failure or neurologic deficit);
- Immersion pulmonary edema;
- Pulmonary thromboembolism**.**

Discussion

Increases in ambient pressure during a dive's descent phase lead to greater amounts of nitrogen dissolving in body tissues up until the point at which the tissues are saturated. When ascending from a dive, inert nitrogen gas bubbles enter the circulation and tissues, which results in decompression sickness (DCS) (5). This is explained by Boyle's law, which states that at a constant temperature, the absolute volume of a fixed mass of gas is inversely proportional to absolute pressure. Furthermore according to Henry's law the amount of gas that will dissolve in a liquid is proportional to the partial pressure of the gas over the liquid at a constant temperature (3,6,7). If a person dives and then flies, DCS is more likely to happen (3,7). Decreased pressure during flying and possibility of dehydration are DCS risk factors (8). Based on the organs damaged and the presenting symptoms, DCS was divided into the two categories - DCS type I and DCS type II.

The clinical manifestations of Type I DCS may include one or more of the following symptoms: arthralgia, myalgia, dermal manifestations and fatigue, while Type II DCS is associated with neurological symptoms or cough, chest pain, dyspnea and other cardiopulmonary symptoms known as "chokes" (5,9-12). Delayed symptoms are caused by intravascular and extravascular bubbles mechanically obstructing the vasculature, which results in stroke-like signs and symptoms and activates clotting and inflammatory cascades, as well as platelet activation and deposition or consumption (3,10–12). Mitchell S et al. say that changes in the coagulation system after decompression include a modest decrease in circulating platelets due to activation and higher consumption, as well as an increase in circulating fibrin monomer (12). Diagnosis of DCI is made almost entirely on history and clinical examination (1,2,11,12). Indeed, in the case of divers, diagnostic tests frequently do not influence treatment choices and should not postpone definitive treatment (12). Inman AL et al. suggest that the absence of symptoms following recompression with HBO2 therapy supports the diagnosis of DCS (13). Our patient herein was 38-years-old woman, recreational diver, smoker, that was diagnosed DCS Type 2 according to the clinical manifestations and history data. She developed DCS while being on a diving vacation in the Maldives. Her first symptoms were cutaneous manifestation known as cutis marmorata and fatigue. She attributed the symptoms to sunburn and tiredness. Kerut C et al. say that cutis marmorata is often associated with a more serious background like neurological symptoms or atrial level shunt, mostly PFO (14). Our patient did not have neurological manifestations, neurologic physical examination showed no abnormalities, Romberg test was negative. Echocardiography was suggested, but the patient refused to conduct it. Her situation worsened after exposure because of the changes in ambient pressure in dives and flight transfers. According to DAN recommendations for flying after diving it was advised that a single no-decompression dive be followed by a minimum 12-hours surface break, a surface interval of at least 18 hours while diving repeatedly over several days and significantly more than 18 hours after diving with mandatory decompression, or while using heliox and trimix (15). The pre-flight surface period in our case was correct according to DAN, but she had already developed symptoms which worsened due to the flight**.** Lee S W et al reported that shortness of breath, dyspnea, and cough are typical symptoms of exceptionally high loads of gas microemboli in the pulmonary arteries (so-called "chokes"). Substernal chest pain, which is sometimes described as burning, is another common symptom (16). This is a rare, but serious manifestation of DCS. Our patient had intense chest pain and dry irritating cough, but she had a good saturation with oxygen on admission and remained hemodynamically stable, without any signs of hypoxemia or hypotension. Thrombocytopenia and elevated D-dimer were noted. Treatment with hyperbaric oxygen according to US Navy treatment Table 6 is recommended for both decompression sickness and arterial gas embolism. It reduces bubbles' size while boosting the gradients in inert gas partial pressure between tissue and alveolar gas, which leads to redistribution and resolution of bubbles trapped in the microcirculation (2, 11, 12). The likelihood of a good recovery is considerably increased by early recompression treatment considered Tawar et al. (9). On the other hand, Hadanny A et al., Inman AL et al. reported in their research that the findings show that hyperbaric treatment, even though delayed, still has significant clinical value and can result in full recovery in 76% of divers, similar to earlier treatment. Indeed, in comparison to the early treatment group, there was no discernible difference (13, 17). Although our patient was delayed with her first medical examination due to late recognition of DCS and delayed with her first treatment, she responded positively to the therapy with hyperbaric oxygen, NSAIL, crystalloids and enoxaparin sodium. She had complete resolution of symptoms with recompression therapy 12 days after the symptom onset.

Conclusion

DCS needs to be suspected anytime a scuba diver experiences any signs of DCS even when followed by appropriate decompression schedules, especially if they have air travel planned. Cutis marmorata is often associated with more serious manifestations of decompression sickness. Treatment with hyperbaric oxygen is the cornerstone therapy for DCS. Indeed, treatment with hyperbaric oxygen can be successful even in cases with delayed start of treatment.

References

1. Zhang XC, Golden A, Bullard DS. Neurologic Deep Dive: A Simulation Case of Diagnosing and Treating Decompression Sickness for Emergency Medicine Residents. MedEdPORTAL J Teach Learn Resour. 2016 Sep 28; 12:10473.
2. Moon RE, Mitchell SJ. Hyperbaric oxygen for decompression sickness. Undersea Hyperb Med J Undersea Hyperb Med Soc Inc. 2021;48(2):195–203.
3. de la Cruz RA, Clemente Fuentes RW, Wonnum SJ, et al. Aerospace Decompression Illness. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 [cited 2023 May 30]. Available from: http://www.ncbi.nlm.nih.gov/books/NBK448160/ .
4. Cooper JS, Hanson KC. Decompression Sickness. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 [cited 2023 May 30]. Available from: http://www.ncbi.nlm.nih.gov/books/NBK537264/ .
5. Saadi A, Ferenczi EA, Reda H. Spinal Decompression Sickness in an Experienced Scuba Diver: A Case Report and Review of Literature. The Neurohospitalist. 2019 Oct;9(4):235–8.
6. Shosholcheva M. Physics for anesthesiologist. 2014. 59–67 p.
7. Yao CK, Huang KL, Peng CK. Near-Fatal Air Travel after Diving: A Case of Impending Tension Pneumothorax on an International Flight. . Vol. 33(6).
8. St Leger Dowse M, Howell S, Smerdon GR. Flying after diving: a questionnaire-based evaluation of pre-flight diving behaviour in a recreational diving cohort. Diving Hyperb Med. 2021 Dec 20;51(4):361–7.
9. Tawar A, Gokulakrishnan P. Decompression Illness. J Mar Med Soc. 2019 Dec;21(2):112.
10. Okeya K, Suzuki S, Koyama Y, Inoue Y. Urticaria-like decompression illness in a caisson worker treated successfully in a monoplace chamber. Acute Med Surg. 2022;9(1) :e808.
11. Vann RD, Butler FK, Mitchell SJ, et al. Decompression illness. The Lancet. 2011 Jan 8;377(9760):153–64.
12. Mitchell SJ, Bennett MH, Moon RE. Decompression Sickness and Arterial Gas Embolism. N Engl J Med. 2022 Mar 31;386(13):1254–64.
13. Inman AL, Sorrell LP, Lagina AT. Decompression sickness responsive to delayed treatment with hyperbaric oxygen: a case report of two divers. Undersea Hyperb Med. 2020 Jan 1;47(4):551–4.
14. Kerut, C. K., Serio, J. R., & Kerut, E. K. (2019). Cutis marmorata in decompression sickness is associated with a patent foramen ovale. Echocardiography. doi:10.1111/echo.14360 10.1111/echo.14360 .
15. Divers Alert Network (DAN). Decompression sickness. 2019. Available at: https://dan.org/health-medicine/health-resource/health-safety-guidelines/guidelines-for flying-after-diving/ .

16. Lee, S. W., Shim, S. S., Choi, J., et al. (2013). Pulmonary Involvement in Decompression Sickness of a Self Contained Underwater Breath Apparatus Diver. The Korean Journal of Sports Medicine, 31(1), 30. doi:10.5763/kjsm.2013.31.1.30 10.5763/kjsm.2013.31.1.30 .
17. Hadanny A, Fishlev G, Bechor Y, et al. (2015) Delayed Recompression for Decompression Sickness: Retrospective Analysis. PLoS ONE 10(4): e0124919. doi:10.1371/journal.pone.0124919 .