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CASE REPORT

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Occupational decompression sickness: A case report

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Abstract

Decompression sickness describes the clinical pathology that ensues when rapid decompression from a highly pressurized environment causes the formation of venous and extravascular inert gas bubbles. Symptoms vary widely, commonly including arthralgias, myalgias, paresthesias, and numbness. Severe and potentially life-threatening pathology, such as neurologic impairment, cardiopulmonary instability, and gastrointestinal hemorrhage, can occur as well. Most think of diving endeavors as a common exposure predisposing to this condition, commonly referred to as "the bends." Other occupational exposures documented in the medical literature include military training, caisson work, such as in mining and bridge construction, and hyperbaric treatment attendance. This article presents the case of a 32-year-old male presenting with a mottled rash, arthralgias, myalgias, headache, vision changes, and weakness, which is found to have decompression sickness secondary to occupational exposure in a factory-based pressurized chamber. The patient underwent two hyperbaric chamber sessions with complete resolution of his symptoms. During hospitalization, he was found to have a patent foramen ovale. The patient was counseled to avoid further occupational exposure.

1 | INTRODUCTION

Decompression sickness (DCS) describes disease resulting when rapid decompression from a highly pressurized environment causes the formation of inert gas bubbles in venous circulation and in extravascular spaces. Additionally, these bubbles can enter into arterial circulation via a patent foramen ovale (PFO) or pulmonary capillary network resulting in arterial gas embolism.[1–3]

In DCS, dissolved gas, such as nitrogen, will form bubbles when the rate of decompression surpasses the rate at which these inert gasses can wash out of venous circulation.[1] Diagnosis of DCS is made clinically with variable forms. Most commonly, DCS manifests within the musculoskeletal system as arthralgias and myalgias. Neurologic manifestations, such as paresthesias and numbness, are also common, but more serious effects such as ataxia, visual changes, altered mental status, speech difficulty, and paralysis are rare. Audiovestibular disturbance, such as vertigo and imbalance, and cutaneous manifestations like rash and pruritus are fairly common as well. Rarely, patients may develop cardiopulmonary distress and/or gastrointestinal pathology.[1,4] As venous bubbles may cross a PFO into arterial cerebral circulation, it has been argued that this more often causes transient visual and/or cognitive symptoms than true stroke syndromes due to the small size of these venous bubbles.[5]

Decompression sickness was first described in the context of caisson work. Caissons are highly pressurized boxes created to allow for the mining of coal below the water table. An engineer, tasked with

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assessing caisson air quality, noted transient arthralgias and myalgias in resurfacing miners. Thus, the first records of DCS as an occupational hazard were produced.[2] Since then, several additional occupations have been associated with DCS. Diving is most commonly implicated; related occupations are myriad, including military operations, seafood harvesting, construction, forensic, and search and rescue operations. Additionally, caisson work related to mining and bridge construction, hyperbaric medicine, and flight operations are all documented occupational hazards in the literature.[1,2,4]

2 | CASE

A 32-year-old male presented to the emergency department (ED) with a rash. The patient stated he worked in a factory where he packaged materials in pressurized tanks reaching 18–20 pounds per square inch. He noted that a few days prior to presentation he noticed mild joint pain and a rash overlying his left flank and abdomen, with significant improvement upon return to this pressurized chamber. On the day of presentation to the ED, however, he states there was an error causing him to decompress more quickly than usual as he missed a stage of decompression during ascension. The patient further clarified that he typically spends 3–4 h per day four times a week in this pressurized setting, and that the decompression process is not driven by computer software, but rather guided by an analog gauge with noted inconsistencies in decompression process and timing. When brought back to atmospheric pressure, he developed headache, chest tightness, nausea, arthralgias, and vision changes, which he described as "looking through a kaleidoscope." He found himself stumbling due to acute right-sided weakness, which spontaneously resolved prior to presentation. Upon returning home, he noticed an extensive rash overlying his torso, at which point he contacted his supervising health director, who had strong concern for DCS and urged the patient to present to the ED. His physical examination was notable for a mottled, flat, erythematous rash on his torso, and bilateral upper extremities (Figures 1 and 2). No neurologic abnormalities were noted on physical examination.

The patient was given supplemental oxygen via non-rebreather mask and immediately evaluated by a hyperbaric physician, who had been made aware of this patient prior to presentation in the ED. The patient was given intravenous fluids and the following labs were obtained: complete blood count, comprehensive metabolic panel, coagulation studies, high sensitivity troponin panel, creatine kinase, urinalysis, and lactate. Results were remarkable for mild leukocytosis (14.0), elevated lactate (2.2), elevated creatine kinase (254), and elevated creatinine (1.27). Additionally, the admitting provider ordered an echocardiogram in order to evaluate for concurrent PFO given concern for the potential development of arterial gas embolism. The patient was admitted to the hospital and underwent emergent hyperbaric treatment. The next day, the patient noted improvement of his symptoms but complained of residual rash and arthralgias. Unfortunately, echocardiogram revealed a PFO, making recompression and decompression cycles more dangerous. Despite the increased risk of neurologic injury, the patient received one additional, short hyperbaric chamber treatment that resolved his remaining symptoms. The patient was counseled to avoid working in the pressurized tank until further follow up could be achieved, and he was discharged from the hospital.

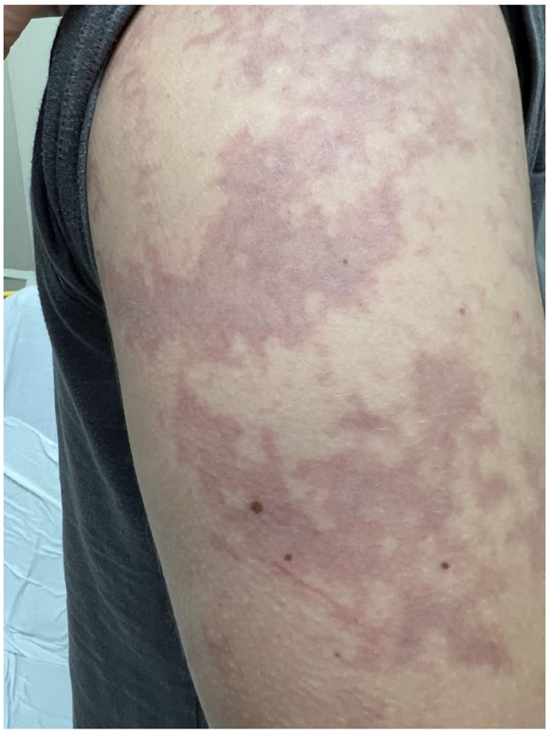

FIGURE 1 Mottled rash on the patient's lateral proximal right upper extremity.

3 | DISCUSSION

This case of a patient suffering from DCS due to exposure to pressurized chambers within a factory is unique among reports of occupational hazard. DCS was first identified and characterized with regard to caisson workers, and more contemporarily studied in commercial, military, and recreational divers.[2,4,6,7] In the commercial diving population specifically, recent attention has been paid across the world to the susceptibility of divers working in fisheries and the financial constraints making them particularly vulnerable.[7] Few reports exist highlighting the risk of DCS in above-ground settings. Specifically, in-chamber hyperbaric treatment attendants are demonstrated to have a small but present risk of DCS in a recent literature review based in France.

In this review, the incidence of DCS among attendants was 0–37 per 100,000 sessions.[8] Similarly, in a military pilot training program using depressurized chambers simulating an altitude of 25,00 feet, trainees were reported to develop DCS upon taking a commercial flight after completion.[9] While one might envision various depressurized and pressurized ground-level occupational environments, there are few reports in the medical literature of cases following other occupational exposures. This is of particular importance to emergency physicians as front-line medical professionals responsible for making this diagnosis

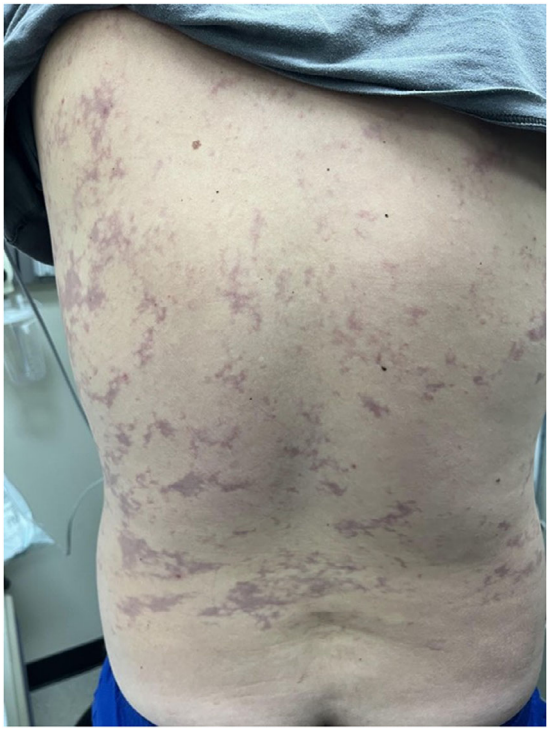

FIGURE 2 Mottled rash on the patient's back.

and arranging for emergent hyperbaric treatment. What is clearly demonstrated by this case is the importance in recognizing the factory environment as a potential risk factor for DCS. Though pressurized packing chambers clearly pose a significant health risk, they are not part of the classic teaching and poorly reflected in the current body of DCS literature.

When patients present with symptoms suggestive of mild DCS, the symptoms most often are vague: myalgias, arthralgias, malaise, and paresthesias.[1,4] Thus, if one considers only the traditional exposures, diagnosis is unlikely. This matters for two primary reasons: first, the patient is unlikely to receive appropriate treatment; second, the lack of diagnostic clarity allows for ongoing ignorant exposure to the occupational hazard. This leaves the patient at increased risk of developing recurrent and potentially severe episodes. Patients do not always offer occupational exposures as causative or even related to their presentation. Thus, the onus is on the physician to consider this rare diagnosis. As the old adage goes, "if a disease is not included in differential diagnosis, it is not likely to be diagnosed."[10] For severe presentations of DCS, rather than presenting a challenge due to vague symptom constellations, the clinical presentation has the capacity to mimic other disease entities with completely different treatment modalities. Neurologic DCS, for example, might closely mimic a stroke. However, traditional ischemic stroke treatments, such as thrombolytics, would only expose this population to risk while withholding the proper treatment.

Lastly, the situation of concurrent DCS with PFO deserves attention due to the excess risk of developing arterial gas embolism. Patients with a PFO, present in 27% of the population, have demonstrated increased risk for developing DCS and associated pathophysiology.[11–13] In one prospective study, the frequency of PFO among divers who developed DCS was found to be 97.2% as compared to 35.5% in controls.[12] Another prospective cohort study demonstrated a significant increase in risk for DCS among those with high-risk PFO, with an odds ratio of 9.34.[13] With respect to the distinctive rash our patient demonstrated, often called "livedo racemosa," one retrospective study found that of patients with diagnosed livedo racemosa due to DCS all had a right-to-left shunt between venous and arterial systems, with 77.8% of these being PFOs.[14] Clearly, this risk is non-negligible and suggests two important principles for emergency physicians as relates to the above case. For patients with a known PFO, symptoms that may be consistent with DCS should prompt a more detailed and targeted exposure history to adequately rule out the diagnosis. Additionally, patients found to have PFO should receive counseling to avoid such exposures both professionally and recreationally.

Overall, this case presents a young, healthy male who developed severe DCS requiring and responsive to emergent hyperbaric chamber decompression therapy as a result of occupational exposure to a highly pressurized materials packing chamber in a US-based factory. Although underrepresented in the current literature, emergency physicians must be aware of this unique exposure as it may allow one to make this uncommon diagnosis in adequate time to arrange the highly specialized and unique treatment.

CONFLICT OF INTEREST STATEMENT

The authors declare that there is no conflict of interest.

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REFERENCES

1. Vann RD, Butler FK, Mitchell SJ, Moon RE. Decompression illness. *Lancet*. 2011;377(9760):153-164. doi:10.1016/S0140-6736(10)61085-9
2. Ninokawa S, Nordham K. Discovery of caisson disease: a dive into the history of decompression sickness. *Proc (Bayl Univ Med Cent)*. 2022;35(1):129-132. doi:10.1080/08998280.2021.1967021
3. Mitchell SJ, Bennett MH, Moon RE. Decompression sickness and arterial gas embolism. *N Engl J Med*. 2022;386(13):1254-1264. doi:10.1056/NEJMra2116554
4. Lee Y II, Ye BJ. Underwater and hyperbaric medicine as a branch of occupational and environmental medicine. *Ann Occup Environ Med*. 2013;25(1):39. doi:10.1186/2052-4374-25-39
5. Mitchell SJ. DCS or DCI? The difference and why it matters. *Diving Hyperb Med*. 2019;49(3):152-153. doi:10.28920/dhm49.3.152-153
6. el-Ghawabi SH, Mansour MB, Youssef FL, el-Ghawabi MH, el-Latif MM. Decompression sickness in caisson workers. *Br J Ind Med*. 1971;28(4):323-329. doi:10.1136/oem.28.4.323
7. Cha SG, Byun YS, Jeon MJ, Sakong J. Diving patterns and decompression sickness among South Korean fishery divers. *J Occup Health*. 2019;61(1):143-153. doi:10.1002/1348-9585.12035
8. Pougnet R, Pougnet L, Lucas D, Henckes A, Loddé B, Dewitte J-D. Health effects of hyperbaric exposure on chamber attendants: a literature review. *Int Marit Health*. 2018;69(1):58-62. doi:10.5603/IMH.2018.0009
9. Allan GM, Kenny D. High-altitude decompression illness: case report and discussion. *C Can Med Assoc J = J l'Association medicale Can*. 2003;169(8):803-807.

10. Jain B. The key role of differential diagnosis in diagnosis. *Diagnosis*. 2017;4(4):239-240. doi:10.1515/dx-2017-0005
11. Hagen PT, Scholz DG, Edwards WD. Incidence and size of patent foramen ovale during the first 10 decades of life: an autopsy study of 965 normal hearts. *Mayo Clin Proc*. 1984;59(1):17-20. doi:10.1016/S0025-6196(12)60336-X
12. Honěk J, Šrámek M, Šefc L, et al. High-grade patent foramen ovale is a risk factor of unprovoked decompression sickness in recreational divers. *J Cardiol*. 2019;74(6):519-523. doi:10.1016/j.jjcc.2019.04.014
13. Lee H-J, Lim DS, Lee J, et al. Decompression illness in divers with or without patent foramen ovale: a cohort study. *Ann Intern Med*. 2023;176(7):934-939. doi:10.7326/M23-0260
14. Hartig F, Reider N, Sojer M, et al. Livedo racemosa—the pathophysiology of decompression-associated cutis marmorata and right/left shunt. *Front Physiol*. 2020;11:994. doi:10.3389/fphys.2020.00994

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