Mottled, Blanching Skin Changes After Aggressive Diving

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ABSTRACT

The initial livedo skin changes of cutis marmorata, also known as cutaneous decompression sickness (DCS), are transient in nature. Accordingly, early images of violaceous skin changes with variegated, marbled, or mottled appearance are rare, whereas later images of deep, erythematous, or violaceous skin changes are readily available. This case presents the opportunity to view the early skin changes characteristic of cutaneous DCS, which would likely manifest at Level I care in the setting of a diving injury during Special Operations missions in austere environments. The unique diving context also allows an overview of DCS in addition to a review of skin eruptions associated with various marine life. As diving is frequently used by Naval Special Warfare, topics presented in this case have significant relevance to Special Operations.

KEYWORDS: *skin; dermatology; cutis marmorata; cutaneous decompression sickness*

Introduction

Cutis marmorata is characterized by a dark red or purple rash with a netlike or reticular appearance. The affected skin blanches with pressure and can be pruritic. The eruption can spread peripherally and eventually becomes deeply erythematous or violaceous. Cutis marmorata is one form of DCS and can stand alone, but it is frequently associated with more severe signs of DCS. It is treated with hyperbaric recompression and supplemental oxygen.[1]

Here we present a case of cutis marmorata in conjunction with neurological symptoms in a 57-year-old man after aggressive diving.

Case Presentation

A 57-year-old man presented with a mottled, violaceous rash over the anterior chest and abdomen 20 minutes after surfacing from a third scuba dive (Figures 1 and 2).

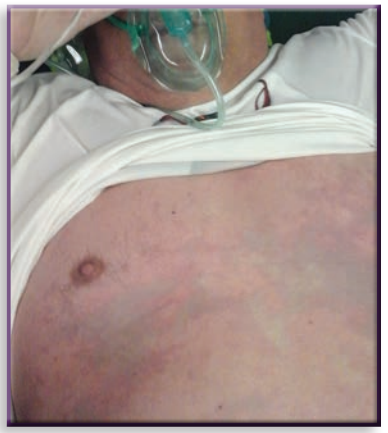

FIGURE 1 *Cutis marmorata. Skin changes on arrival at the emergency room of Chuuk State Hospital.*

Source: Julius Caesar G. Arsenal, MD (chief surgeon, Chuuk State Hospital).

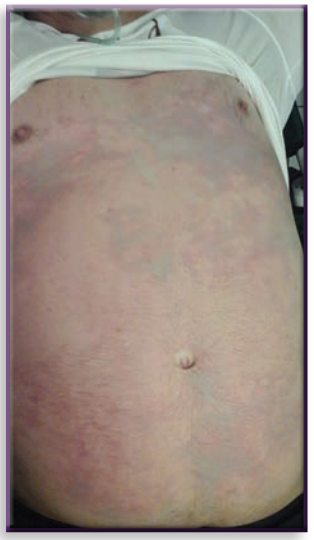

FIGURE 2 *Cutis marmorata. Transient, reticulate, and violaceous eruption.*

Source: Julius Caesar G. Arsenal, MD (chief surgeon, Chuuk State Hospital).

He was diving in the Marianas with air. The following is his dive profile:

- First dive: maximum depth of 35 m for 50 minutes with a 2.5-hour surface interval
- Second dive: maximum depth of 28 m for 24 minutes with a 50-minute surface interval
- Third dive: maximum depth of 18 m for 30 minutes

Based on US Navy Diving Tables, he owed over 300 minutes of decompression time.

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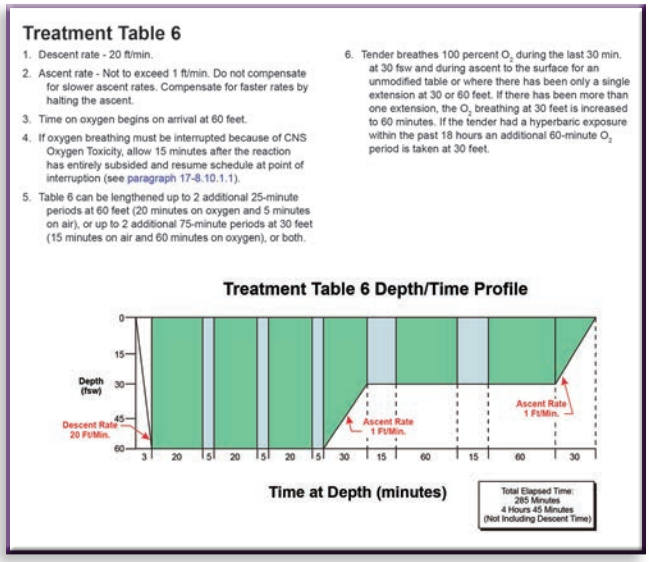
The rash blanched with pressure. Additional symptoms included shortness of breath and generalized malaise. A physical examination revealed left lower leg paresis.

He was immediately placed on surface oxygen on the diving boat and then transported to the state hospital via ambulance. He was treated with surface oxygen at 11 L/min overnight via nonrebreather mask. His symptoms improved on surface oxygen, which he continued to breathe until transport was secured.

He was appropriately transported to Guam via turboprop air ambulance with the cabin pressurized to 1 standard atm of pressure and at 2133.6 m of altitude. He arrived at the Naval Base Guam Recompression Chamber about 36 hours after the onset of symptoms. By this time, shortness of breath had resolved, and the left lower leg demonstrated movement against some resistance on examination. The skin changes had remained confined to the chest and abdomen but had become erythematous.

The treating team decided to pursue hyperbaric recompression according to a Treatment Table 6 protocol with maximum extensions at 18 m. Figure 3 from the *US Navy Diving Manual* depicts the treatment protocol.[2]

FIGURE 3 *Treatment Table 6.*[2]



He was brought to 18 m of depth at a rate of 6.096 m/min. He breathed 100% oxygen for three 20-minute periods at 18 m according to standard protocol. His treatment was extended with two additional 20-minute periods at 18 m as his left lower leg weakness resolved toward the end of the third oxygen period. All oxygen periods at 18 m were followed by 5-minute air breaks to prevent oxygen toxicity. He continued to breathe 100% oxygen during a 30-minute ascent at 0.3048 m/min to 9 m, at which time a 15-minute air break was administered. The table concludes with two additional 60-minute, 100% oxygen periods at 9 m with an interval 15-minute air break between periods. The patient was then brought back to surface, ascending at a rate of 0.3048 m/min, on 100% oxygen.[2]

Discussion

Cutis marmorata in the adult population is rarely associated with medical conditions other than DCS. Violaceous, reticular skin eruptions may precede retiform purpura in medical conditions to include calciphylaxis, antiphospholipid antibody syndrome, and warfarin-induced skin necrosis.[3] According to the *Divers Alert Network Annual Diving Report 2017 Edition*, there were 76 cases of cutis marmorata in 2015. However, the true incidence is unknown because there are no official reporting requirements and because cases may not be reported to the Divers Alert Network Medical Services Call Center (DANMSCC), which provides medical information in addition to coordinating treatment and evacuation for injured divers. Well known to the US Navy and civilian divers, the Divers Alert Network collects data regarding diving injuries, not including fatalities, from DANMSCC and the Annual Survey of Hyperbaric Chambers. The initial information is entered into a database. Follow-up is conducted to confirm actual diagnosis of DCS. Results are analyzed and published in the Annual Diving Report.[4]

DCS is a pathologic response to bubble formation. The pathophysiology is governed by Henry's law: the amount of gas that will dissolve in a liquid at a given temperature is directly proportional to the partial pressure of that gas. Inert gas, mainly nitrogen, is forced into the tissues of the body by increased ambient pressure at depth during dives. During controlled ascent, inert gas gradually comes out of solution as ambient pressure decreases. When ambient pressure reduction occurs too quickly, intravascular and extravascular bubbles may form.[1] The latter is easily visualized when opening a beer can. Removing the lid causes a quick reduction in pressure and bubbles form as a result. In this case, the diver omitted more than 300 minutes of decompression time, which is time expected to be spent at depth allowing nitrogen gas to slowly come out of his tissues. This severely increased his risk of DCS.

Bubbles cause injury through two main mechanisms: mechanical and nonmechanical. Mechanically, bubbles cause distortion or compression of tissue, leading to pain and edema, or it can cause vascular obstruction, leading to stroke-like signs. Nonmechanically, bubbles may act as a foreign body, provoking the inflammatory response.[1]

A literature search reveals multiple cutis marmorata images of deeply erythematous and violaceous skin eruptions that are similar to the skin changes of this patient at 36 hours after onset. The mechanisms of injury support the transient nature of cutaneous DCS lesions. Vascular obstruction initially leads to ischemia and, therefore, early reticular, violaceous changes. Eventually, the inflammatory response causes deep erythema. However, there are limited data characterizing DCS skin lesions in relation to time. One study published in July 2017 depicts a temporal development of skin lesions in swine with induced DCS. The lesions were characterized from stage I to stage VI. The reticular lesions depicted in Figures 1 and 2 in this case are comparable to lesions of stage I and stage II in the study, respectively, which appeared early after surfacing and were short lived.[5]

DCS is categorized into type I and type II. Type I DCS is further classified as musculoskeletal, cutaneous, or lymphatic. Type II DCS is further classified as pulmonary or neurological. Neurological DCS is further subclassified into peripheral, spinal cord or central nervous system, cerebral, vestibular, and ocular. Characteristics of the subtypes of type I DCS and type II DCS are delineated in Table 1 and Table 2, respectively.[1]

Most cases of DCS will present within 12 hours of surfacing from a dive, with more than 90% presenting within 24 hours. More than 80% of neurological DCS will present within 1 hour and thus will be likely to be encountered early in the echelons of care. DCS is treated with hyperbaric recompression and supplemental oxygen.[1] Protocols for treatment are specified in the *United States Navy Dive Manual, Revision 7*.[2] Surface oxygen suffices for initial treatment if a hyperbaric chamber is not available.[1]

TABLE 1 *Type I DCS*

Condition	Characteristics
Musculoskeletal	Dull, aching joint pain unaffected by movement or rest
Cutaneous	**Cutis marmorata –** violaceous skin changes with marbled or mottled appearance, blanches with pressure
Lymphatic	Painful localized area of swelling involving lymph nodes and contiguous areas

TABLE 2 *Type II DCS*

Condition	Characteristics
Pulmonary	Pain on inspiration or expiration, cough, substernal discomfort
Neurologic	**Peripheral –** weakness or paresthesia in single nerve distribution
	Spinal cord (central nervous system) – multicord level weakness or multifocal, nondermatomal paresthesia
	Cerebral – mental status changes, facial weakness, diplopia, hearing loss, hemiparesis, monoparesis, loss of coordination
	Vestibular – vertigo, tinnitus, hearing loss, nystagmus
	Ocular – visual field defects, diplopia, scotoma, ocular muscle impairment, convergence insufficiency

Skin rashes occur frequently after diving. However, it is important to distinguish cutis marmorata as it is often associated with or may prelude additional severe signs and symptoms of DCS.[6] The differential diagnosis includes itching and mild urticaria, lymphatic DCS, fire coral dermatitis, and Portuguese man-of-war envenomation.

Itching and mild urticaria may present after diving. It is typically more common with hyperbaric chamber dives. Urticarial lesions are erythematous and blanche with pressure, but the main distinction from cutis marmorata is papular quality and elevation. Urticaria is not considered DCS and does not need to be treated with recompression.[1]

Lymphatic DCS is rare. It typically presents with localized swelling and involves contingent lymph nodes. It is associated with severe pain. Areas affected can include the face, breast, abdomen, and extremities. The treatment is recompression.[1]

Fire coral dermatitis is caused by contact with fire coral, which are reef-colonizing organisms found in subtropical and tropical waters. Their appearance varies with environment, but they generally have branching structure and range from yellow-green to brown in color. Dermatitis caused by fire coral is seen mainly in scuba divers.[7] Fire coral have nematocysts in their tentacles that release toxins, which provoke cutaneous changes that may appear ulcerated, eczema-like, vesicular, or urticarial. Diagnosis is aided by history and often concurrent systemic symptoms to include malaise, fever, nausea, and abdominal pain. Initial treatment consists of nematocyst removal from the skin and then cleansing and debridement with soap and seawater.[8] A delayed type IV hypersensitivity reaction may occur days to weeks after exposure and present with erythematous macules, plaques, or papules in the distribution of fire coral contact. Treatment is supportive and includes topical steroids and antihistamines.[9]

Portuguese men-of-war inhabit waters of the Atlantic and the Indo-Pacific. They have blue, gas-filled bladders and long tentacles.[10] Envenomation by a Portuguese man-of-war can result in an immediate type I reaction, caused by toxic action or allergic action, or result in a delayed type IV reaction, caused by allergic action. Toxic action-induced immediate reaction can result in a linear, erythematous, papular eruption, which can progress to development of vesicles and necrosis. In severe cases of envenomation by a Portuguese man-of-war, toxic action-induced immediate reaction can include systemic manifestations to include cardiopulmonary shock, which would warrant resuscitative measures. Allergic action-induced immediate reaction results in anaphylaxis or angioedema, both of which are treated with intramuscular epinephrine and intravenous steroids. Delayed allergic reactions include persistent vesicular, urticarial, or eczema-like lesions 48 hours after envenomation, development of the latter lesions at sites distant from initial envenomation, and cycles of recurrent cutaneous reactions after resolution. Delayed allergic reactions are treated supportively with antihistamines and corticosteroids.[11]

In summary, cutis marmorata is one form of type I DCS and is often associated with or precedes more severe signs of DCS. It is actual pathology that Special Operators are at risk of while training for and conducting missions in the undersea environment, even when complying with safe diving standards. As a harbinger of potential DCS progression, recognition and prompt treatment of cutis marmorata by Special Operators are of clinical significance.

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Author Contributions
AL is the first author. MJ and SR made substantial contributions to structuring the article, revising the article, and approving the final version.

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