Clinical Significance of Mottling Rashes in Diving Decompression Sickness

Ilana D. Breen; Jan Stepanek; Lisa Marks; Katerina Yale; Natasha Mesinkovska; David Swanson

INTRODUCTION: Decompression sickness (DCS) is a medical condition caused by outgassing of dissolved nitrogen following rapid ascent by divers and aviators. Cutaneous DCS, historically termed cutis marmorata (CM), presents as a predominantly truncal reticular violaceous-to-dusky eruption. The prevailing theories for its pathogenesis include: localized cutaneous outgassing, paradoxical embolism across a right-to-left shunt (RLS), and brainstem emboli disrupting autonomic control of cutaneous microcirculation.

METHODS: We conducted a systematic review of reports of cutaneous DCS to investigate relationships among CM, RLS, and neurological sequelae to better elucidate the mechanism of CM. A literature search examining reports of cutaneous DCS yielded 31 eligible studies, comprising a pooled total of 128 patients.

RESULTS: Of the patients with documented workup, 84% showed evidence of RLS with CM. Subsequently 18 patients underwent percutaneous closure of intracardiac RLS with no recurrence of DCS. Of the patients with documented neurological evaluations, 57% experienced both CM and neurological DCS manifestations. The coexistence of RLS and neurological symptoms with CM was noted in numerous cases; exact percentages of overlap cannot be stated due to data unavailability.

DISCUSSION: Our results indicating the striking coexistence of RLS and neurological sequelae in CM patients is supportive of the paradoxical embolism theory of pathogenesis. The frequent coincidence of CM with RLS and neurological symptoms raises concern that CM may signify vulnerability to devastating systemic gas emboli. CM has historically been considered trivial and self-limiting; however, our results support reappraisal of its clinical significance and potential reclassification to the more severe subtype.

KEYWORDS: decompression sickness, diving, livedo racemosa, cutis marmorata, right-to-left shunt, patent foramen ovale.

Breen ID, Stepanek J, Marks L, Yale K, Mesinkovska N, Swanson D. *Clinical significance of mottling rashes in diving decompression sickness*. Aerosp Med Hum Perform. 2024; 95(9):695–702.

Decompression sickness (DCS) is a medical condition observed in divers, caisson workers, and aerospace operations personnel (aviation, spaceflight). It results from outgassing of dissolved nitrogen due to decreased ambient barometric pressure. The U.S. Navy stratifies DCS by severity into Types I and II, a classification scheme first introduced by Golding et al. in 1960.[1,2] Type I DCS is mild and predominantly involves the joints, skin, and lymphatic vessels. Type II manifests with life-threatening cerebral, respiratory, and cardiovascular complications.

The term "decompression sickness" refers to pathological conditions arising both from ascent during diving (hyperbaric) and altitude exposure (hypobaric). The inert gas load, offgassing dynamics, and gas phase evolution are pathophysiologically distinct between the two settings. Unfortunately, their shared DCS nomenclature leads to the problematic conflation of the conditions, which differ in prognostic and treatment considerations. Particularly, cutaneous DCS in diving portends

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This manuscript was received for review in March 2024. It was accepted for publication in April 2024.

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DOI: https://doi.org/10.3357/AMHP.6454.2024

poor outcomes, while cutaneous DCS in altitude-associated DCS typically features a benign course and prognosis. For this review, we specifically limited the investigation to DCS in the context of diving.

"Cutis marmorata" (CM) is the historical term used to describe the reticular violaceous macular eruption seen in cutaneous decompression sickness. Although cutaneous DCS has been dismissed as a localized, self-limited process and relatively benign phenomenon,[3] there is growing evidence that CM occurring in patients with DCS may be a harbinger of coexistent cerebral embolism. At this time, there are no consensus recommendations for CM.

For this systematic review, we examined reports of cutaneous DCS findings in the setting of diving and investigated relationships among neurological sequelae, CM, and right-to-left shunts (RLS). We also discuss the clinical findings, etiology, pathophysiology, and prognostic significance of cutaneous findings in the setting of DCS.

METHODS

We searched the Ovid MEDLINE (1946 to present), Ovid EMBASE (1974–2023), Web of Science (1975–2023), and Scopus (Inception to present) databases. A combination of keywords and Medical Subject Headings (MeSH) terms were used for the search strategy. Keywords included: "livedo racemosa," "livedo reticularis," "cutis marmorata," "spider vein," "bends," "Caisson disease," and "decompression sickness." The Boolean operators "AND" and "OR" were used to combine terms, keywords, and concepts. MeSH headings included: "livedo reticularis," "skin diseases, vascular," "decompression sickness," and "barotrauma." Filters were placed to search for English language articles involving human subjects. Articles were selected if they represented case reports, case series, or retrospective case control studies of CM in the setting of diving-associated DCS. Additionally, we searched citations from eligible articles for other relevant articles matching our inclusion criteria. The search strategy was compliant with Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) and yielded 31 total eligible studies, including 25 case reports, 5 case series, and 1 retrospective case control study (**Fig. 1**).

RESULTS

The 31 included studies contained a pooled total of 128 patients with diving-related CM (**Table I**). Of these, 63 patients demonstrated neurological deficits, while 47 had no neurological sequelae, and 18 were unknown (e.g., data unavailable or unreported). Of the patients with documented neurological evaluations (N = 110), 57% (63/110) experienced both skin and neurological DCS manifestations.

In total, 76 patients had evidence of RLS, while 14 had no shunt, and 38 were unknown (e.g., data unavailable or unreported). Of the patients with documented workup for RLS (N = 90), 84% (76/90) showed evidence of RLS in addition to DCS skin manifestations. Of the 76 patients with documented shunts, 19 were classified as patent foramen ovale (PFO), 2 as atrial septal defect, 3 as noncardiac shunt (2 pulmonic RLS, 1 liver-associated right-to-left arteriovenous malformation), and 52 as intracardiac RLS not otherwise specified. The intracardiac shunts were diagnosed with transthoracic echocardiography in 69 cases and with transesophageal echocardiography in 2 cases; the noncardiac shunts were diagnosed with CT imaging. At least one patient initially tested negative for intracardiac RLS using transthoracic echocardiography imaging with antecubital injection of agitated saline, but, notably, the shunt was unmasked with improved technique and intrafemoral injection. Subsequently 18 patients underwent percutaneous closure of intracardiac RLS with no recurrence of cutaneous DCS recurring in at least 17 of these patients. At least 2 patients reported complete cessation of diving activities following diagnosis of intracardiac RLS; both patients deferred option for percutaneous closure.

The reported instances of RLS and neurological symptoms were frequently coincidental (see Table I); the exact percentages of overlap cannot be stated due to lack of data availability. Full recovery ensued in most cases, achieved either spontaneously (6%), with supplemental oxygen (9%), or with hyperbaric oxygen therapy (84%). There were 4 fatalities reported in the 128 pooled cases examined; all these patients presented with skin, cardiopulmonary, and neurological signs and symptoms. Data on the presence of RLS was not reported for all four patients who suffered fatalities. All four patients died despite standard-of-care hyperbaric oxygen therapy or before hyperbaric oxygen therapy could be initiated.

DISCUSSION

"Cutis marmorata" is the term used to describe the reticular violaceous macular eruption seen in Type I DCS. This is a red-violaceous, nonblanchable reticular network of macules that presents predominantly in a truncal distribution, with acral sparing. The term "cutis marmorata" also refers to the mottling physiological rash seen physiologically, often times in newborns with immature vascular development. Despite the widely accepted nomenclature of "cutis marmorata" to describe the cutaneous DCS rashes, the clinical appearance is more consistent with livedo racemosa (LRC) (**Fig. 2**), which is commonly seen in medium-vessel vasculitis and vasculopathies such as polyarteritis nodosa, or calciphylaxis. A recent article by Hartig reappraised the nomenclature of cutaneous DCS and introduced "livedo racemosa" as the dermatologically correct term.[11] We will herein refer to the rash of cutaneous DCS as CM/LRC for clarity, given the recent change in proposed nomenclature.

The pathophysiology of DCS is driven by the governing principles of gas mechanics, including Henry's law. During ascent, the decreasing barometric pressure leads to decreased

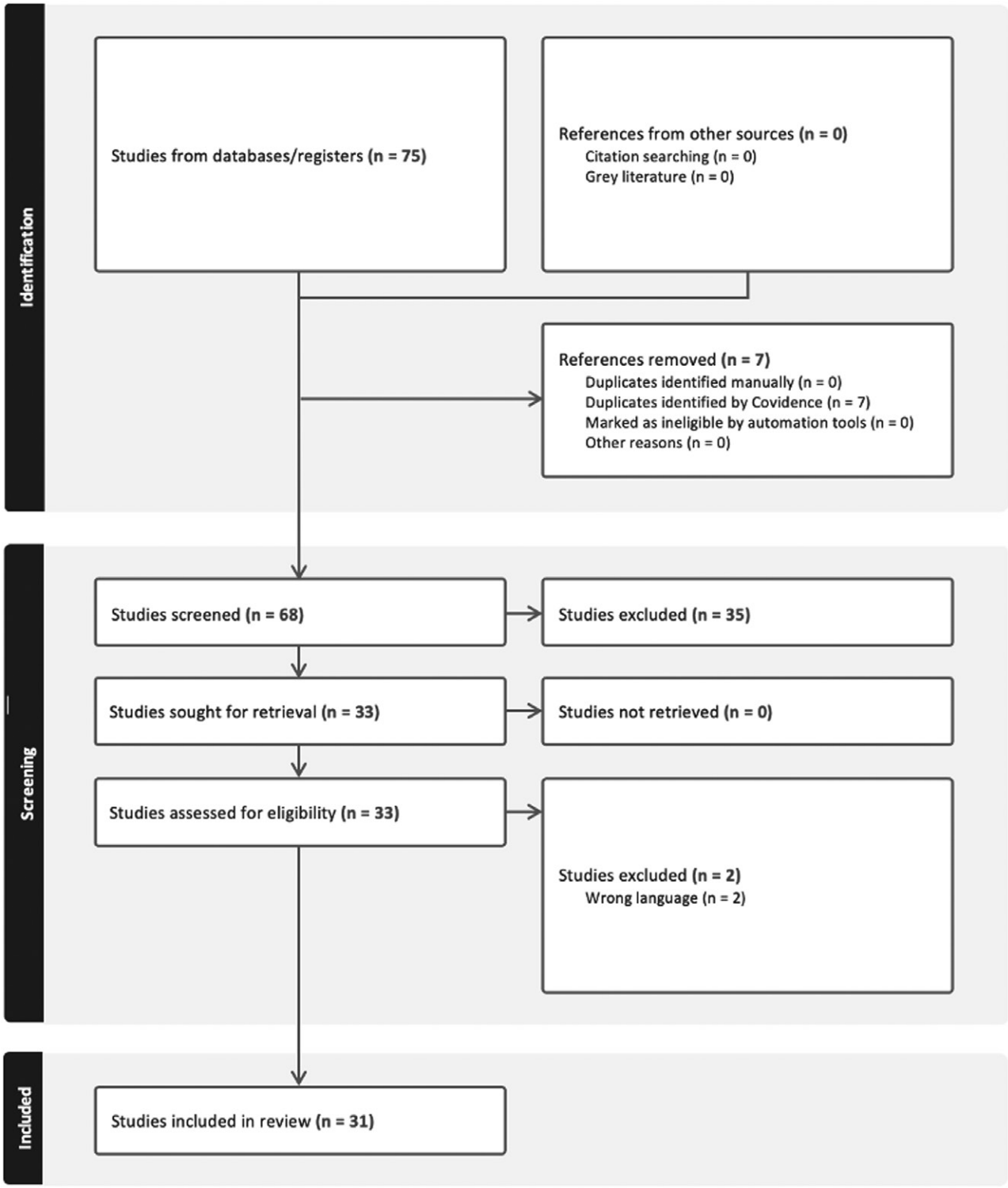

Fig. 1. PRISMA search strategy flow diagram.

dissolved inert gases and consequent formation of nitrogen bubbles, known as outgassing. When the production of dissolved gases exceeds the removal rate during exhalation, supersaturation occurs, and gases may bubble out. Outgassing may also occur directly at the tissue interface due to the high inert gas partial pressure differential between blood and tissue.[3]

DCS has been regarded as an occlusive-ischemic event, with venous gas emboli (VGE) obstructing vessels, leading to endothelial disruption, increase in nitric oxide in lesion tissue, coagulation cascade activation, and vascular spasm.[35–37] The persistence of DCS symptoms following hyperbaric treatment implicates a VGE-induced inflammatory cascade as the driver

Table I. Interrelationships Among Right-to-Left Shunt, Neurological Symptoms, and Cutaneous DCS.

STUDY	STUDY TYPE (PATIENT NUMBER)	CLINICAL PRESENTATION	RIGHT-TO-LEFT SHUNT	NEUROLOGICAL EVENTS	RECOVERY
Avivi et al.[4]	Case report (1)	Skin rash, portal venous gas emboli	Unknown	Yes	Significant after hyperbaric oxygen therapy
Azzopardi et al.[5]	Case report (1)	Skin rash only	Yes	No	Full after hyperbaric oxygen
Dziewiatowsi et al.[6]	Case report (1)	Skin rash, neurological, vertigo	Yes	Yes	Full after hyperbaric oxygen
Magri et al.[7]	Case series with matched controls (7)	Skin rash, neurological involvement (2/7), multisystem involvement (3/7)	Unknown	Yes 2/7	Full after hyperbaric oxygen
Arjomand et al.[8]	Case report (1)	Loss of consciousness, cardiovascular shock, skin rash, severe hypoxia	Unknown	Yes	Residual DCS-related neurological deficits after hyperbaric oxygen
Selesny & Singh[9]	Case report (1)	Skin rash, lightheadedness	Unknown	No	Full with hyperbaric oxygen
Yu et al.[10]	Case report (1)	Skin rash, cardiac, joint, abdominal, respiratory, cardiovascular	Unknown	No	Full after hyperbaric oxygen
Hartig et al.[11]	Case series (18)	Skin rash	Yes 18/18	Unknown	Unknown
Schwob et al.[12]	Case report (1)	Skin rash	Yes	Yes	Unknown
Garcia & Mitchell[13]	Case series (4)	Skin rash only	Yes 4/4	Yes 4/4	Full after hyperbaric oxygen
Yount[14]	Case report (1)	Skin rash, neurological, cardiovascular, renal	Unknown	Yes	Full after hyperbaric oxygen and ICU supportive care
Lau et al.[15]	Case report (1)	Skin rash only	Unknown	No	Full
Kerut et al.[16]	Case report (1)	Skin rash only	Yes	No	Full after hyperbaric oxygen
Strauss[17]	Case report (1)	Skin rash only	Unknown	Yes	Full after high flow 100% oxygen
Rozenblat & Ziv[18]	Case report (1)	Skin rash only	Unknown	No	Full (spontaneously, no intervention)
Mendez et al.[19]	Case report (1)	Skin rash, neurological, cardiopulmonary	Unknown	Yes	Fatality
Sun & Gao[20]	Case report (1)	Skin rash, portal venous	Unknown	No	Full after hyperbaric oxygen
Perez-Lopez et al.[21]	Case report (1)	Skin rash only	Yes	No	Full (spontaneously, no intervention)
Jitsuiki et al.[22]	Case report (1)	Skin rash, neurological, cardiopulmonary	Unknown	Yes	Fatality
Kemper et al.[23]	Case report (1)	Skin rash only	Yes	No	Full after 100% oxygen
Tasios et al.[24]	Case report (1)	Skin rash only	Unknown	No	Full after 100% oxygen
Modell[25]	Case report (1)	Skin rash, neurological, lymphatic	Yes	Yes	Full after hyperbaric oxygen
Wen et al.[26]	Case report (1)	Skin rash, cardiopulmonary, neurological	Unknown	Yes	Fatality
Oode et al.[27]	Case report (1)	Skin rash, cardiopulmonary, neurological	Unknown	Yes	Fatality
Akca et al.[28]	Case report (1)	Skin rash, neurological	Unknown	Yes	Full after hyperbaric oxygen
Sundal et al.[29]	Case series (10)	Skin rash, joints, neurological	Unknown	Yes 7/10	Unknown
Mutluoglu et al.[30]	Case report (1)	Skin rash, joints, neurological	Unknown	Yes	Full after hyperbaric oxygen
Bledsoe et al.[31]	Case report (1)	Skin rash, musculoskeletal	Unknown	No	Full after hyperbaric oxygen
Kalentzos[32]	Case report (1)	Skin rash, neurological	Unknown	Yes	Full after hyperbaric oxygen
Wilmshurst et al.[33]	Retrospective case-control (61)	Skin rash, neurological (35/61), cardiovascular (3/61), musculoskeletal (12/61)	Yes 47/61	Yes 35/61	Full
Li[34]	Case series (3)	Skin rash, joints, neurological	Unknown	Yes 1/3	Full after hyperbaric oxygen

of tissue damage, in addition to deposition of the gas bubbles themselves.[38] It is important to note that most cases of DCS arise even in spite of strict adherence to ascent procedure guidelines (dive/decompression tables). In addition, all factors held equal, some divers may develop DCS while most others do not. These characteristics suggest that pathophysiology of DCS is more complex than what is currently understood.

The pathophysiology of skin manifestations specifically in DCS is controversial. Histopathology of CM/LRC biopsies has shown vascular congestion, perivascular neutrophil infiltrates, edema, and occasionally hemorrhage.[39] There are three theories of cause, which include: autochthonous outgassing, paradoxical arterial emboli deposition in skin, and paradoxical emboli to the central nervous system (CNS) with disrupted autonomous control of cutaneous microcirculation.[13] One study sought to elucidate CM/LRC pathogenesis by observing and systematically describing the progression of DCS skin manifestations in a swine model through hyperbaric diving simulations.[40] Outgassing was exclusively noted in the venous circulation with strong correlations between bubble load, skin lesion area, temporal lesion course, and presence of lesions. Neurological evaluation yielded no signs of CNS dysfunction, appearing to support the first theory rather than the brainstem hypothesis[40]; notably, however, neurological evaluation of the

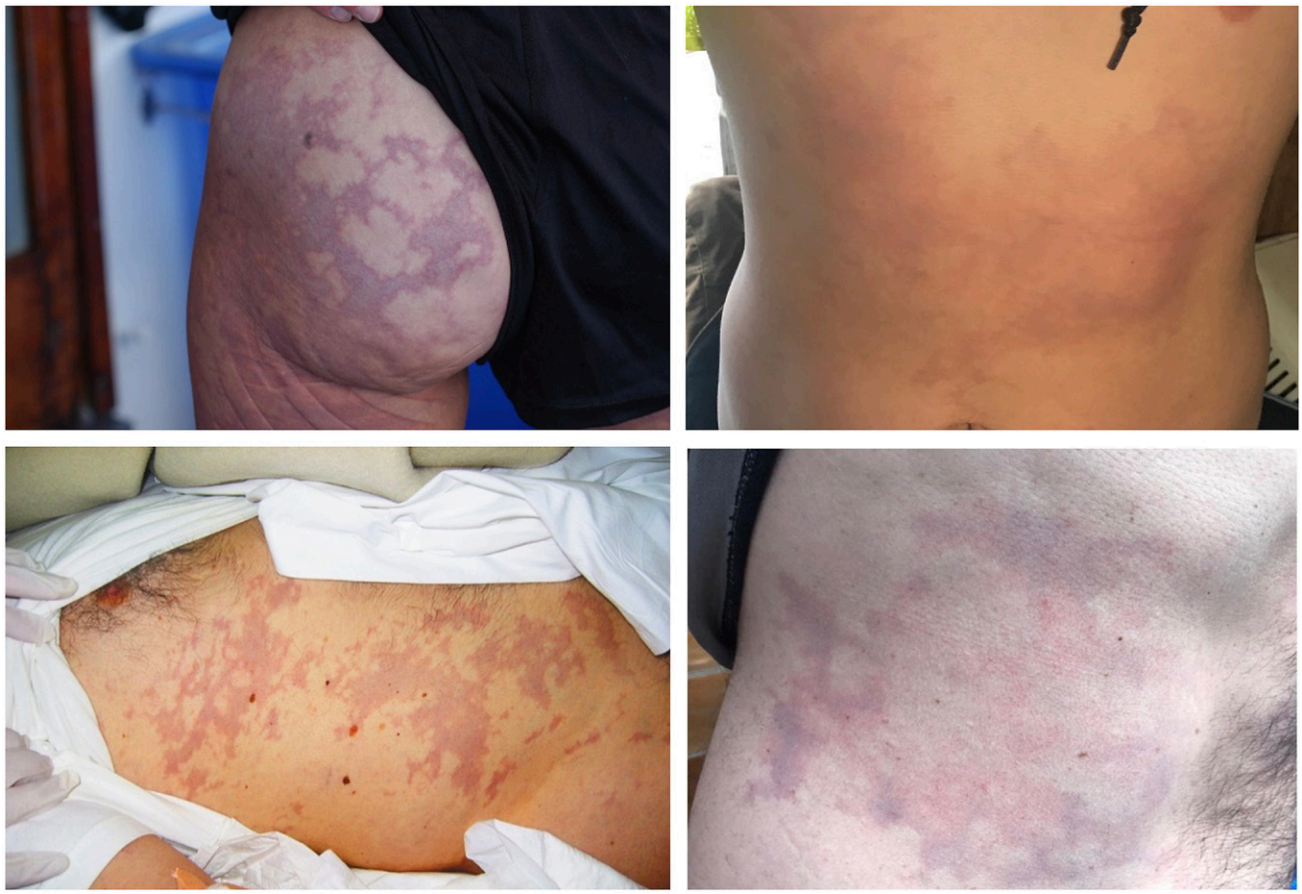

Fig. 2. Cutis marmorata in the setting of diving decompression sickness on the buttock and posterolateral thigh (top left), anterior torso (top right), left lateral torso (bottom left), and anterolateral torso (bottom right).

swine model is handicapped by lack of symptom self-reporting beyond severe deficits. Other studies have also shown evidence in support of the first theory.[13,41] Other studies further link paradoxical arterial emboli directly to the subcutaneous capillary plexus, supporting the second theory.[13,33,42] In a different swine study, an air embolus injected into the internal carotid artery induced a CM/LRC rash, seeming to support the third theory.[23] The pattern of concurrent CNS and skin DCS manifestations noted in the literature over time has sustained speculation about the brainstem emboli hypothesis.[3,39]

There is growing evidence that CM/LRC may represent a signal of contemporaneous cerebral embolism rather than an isolated cutaneous manifestation. At this time, there are no consensus recommendations for CM/LRC management, such as hyperbaric oxygen therapy; CM/LRC has been historically dismissed as a localized, self-limited process and not a medical emergency.[3] Our results showed an 84% coincidence with CM/LRC and RLS and a 57% coincidence with CM/LRC and neurological symptoms. It may be time to rethink this clinical sign, given an emerging paradigm of paradoxical emboli and cerebral injury in patients with DCS.

DCS and Association with RLS and Neurological Events

The coexistence of skin DCS and RLS is well documented in the literature. One study revealed a 77% prevalence of RLS in divers with cutaneous DCS compared to 27% in control divers without DCS.[33] Of all those who had shunts, those divers with cutaneous DCS had larger shunts compared to those without cutaneous DCS. Another study implicated PFO in nearly 100% of CM/LRC cases.[3] Yet another study demonstrated a 100% RLS prevalence in a study of 18 divers with cutaneous DCS; 83% were intracardiac and 17% were pulmonary/liver-associated.[11] Our review similarly found that 84% of patients with DCS-associated skin manifestations possessed RLS where data was available.

As mentioned previously, 18 patients with documented cardiac RLS underwent percutaneous closure, with no further DCS events in at least 17 of these patients. However, notably, DCS is a probabilistic event and thus this statistic must be interpreted in the context of number of dives previously completed and the nature of the dives (i.e., whether the dives complied with recompression guidelines during ascent). Of the 17 patients who underwent percutaneous closure of RLS cardiac shunt, 1 had logged 3996 lifetime dives at the time of her DCS insult and had experienced 3 prior episodes of DCS (one with lymphatic symptoms, two others with cutaneous symptoms). For the other 17 of the 18 patients who underwent percutaneous closure, the number of prior lifetime dives and/or episodes of DCS were not reported. For all 18 patients, there were no noted deviations from standard diving ascent guidelines during

the dives that resulted in DCS. While it is tempting to attribute resolution of DCS events to the percutaneous RLS closure given the timing, there is not enough information reported on the dives nor sufficient power to make any definite claims.

The prevalence of PFO in the adult population is between 25–30%.[43,44] However, this figure likely underestimates a higher prevalence of RLS by excluding probe-patent foramen ovale, a finding only observable on autopsy.[43] Furthermore, echocardiography and transcranial Doppler studies used to search for RLS carry high false negative results, since high intrathoracic pressures in hyperbaric conditions cannot be replicated with simple induced Valsalva during testing, further underestimating the prevalence.[11] Therefore, our results may similarly underestimate the presence of RLS in the patients we reviewed with cutaneous DCS.

The pressure differential between right and left circulations at the atrial level typically prevents blood flow through a PFO; however, diving-related straining and maneuvers, such as heavy lifting of diving equipment and encouraged Valsalva to equalize Eustachian pressures during ascent, may contribute to transient RLS.[39,45,46] Moreover, high levels of venous outgassing into the pulmonary microcirculation may cause increased right ventricular filling pressure, further facilitating RLS.[47,48] The exceptionally high coincidence of skin DCS and RLS found in this review favors the hypothesis of paradoxical embolus. Two previously expounded theories include paradoxical embolus to brain and skin, respectively. However, patients suffering embolic stroke to the brainstem rarely demonstrate skin findings akin to diving DCS, which argues against the brainstem hypothesis. In addition, newer studies have questioned previously reported experimental models suggestive of the brainstem hypothesis (e.g., injection of internal carotid artery with subsequent CM/LRC rash development in swine) about whether the rash was secondary to sympathetic surge from the stress of large cerebral air introduction, rather than the embolus itself.[49]

The discussion of RLS prevalence in cutaneous DCS deserves mention because of its implicated role in neurological DCS. A large systematic review assessed the risk of neurological DCS in individuals with PFO and discovered that the combined odds ratio of nervous system sequelae in divers with RLS was 4.23. A meta-analysis specifically of those with large shunts revealed an even higher odds ratio of 6.49.[50] However, the interpretation of these findings is confounded by contradicting studies.[51] Nonetheless, the high odds ratios warrant additional investigation into the significance of RLS as it pertains to neurological DCS.

Association of Cutaneous DCS and Neurological Involvement: We found that 57% of subjects with cutaneous DCS had signs or symptoms of neurological involvement, where data was available. This follows previous literature reports of an association between cutaneous DCS and neurological symptoms. One study even recommended that all cutaneous DCS cases be managed as neurological DCS.[29] A study in porcine model simulated diving conditions and induced neurological DCS in 73% of animals. Affected pigs had earlier onset of cutaneous involvement and subsequent autopsy showed a majority of cases had spinal cord petechial hemorrhages, again demonstrating co-occurrence of skin and neurological DCS findings.[52] Another study indicated that the presence of LRC in Sneddon's syndrome, a genetic disorder associated with ischemic strokes and vascular brain abnormalities, may reinforce the third theory of DCS skin rash association with CNS dysfunction.[23] While the brainstem embolus hypothesis nicely unifies these two organ system involvements, it is also possible that skin and neurological symptoms manifest from separate paradoxical emboli through RLS.

Reappraisal of the Term Cutaneous DCS

The terms "cutis marmorata," "livedo reticularis," and "livedo racemosa" have been used interchangeably in the literature. The visible cutaneous patterns of livedo reticularis and CM is due to benign vasospastic response to cold temperatures and is characterized as a nonpainful, flat, uniform, regular network of closed rings located diffusely. In contrast, LRC is due to obstruction of dermal medium vessel arterioles and appears grossly as a raised, painful, violaceous, and irregular net-like pattern located primarily on the limbs, trunk, and buttocks.[11] Truncal predilection is attributable to the highly lipid-soluble nature of nitrogen, which preferentially is offloaded in adipose-rich areas.[33]

In accordance with the findings and analysis of Hartig et al.,[11] we would like to challenge the paradigm of "cutis marmorata" as the accepted term for all mottling diving-associated skin rashes, a distinction that confers mechanistic implications. While many previous reports seek to unify all mottling skin findings to one pathophysiology, we agree with Hartig et al. that there are different types of skin rashes with unique pathogeneses that present following diving. Livedo reticularis is a mottling reticular rash that can be observed following diving exercise due to lowering of body surface temperature, but is unrelated and distinguishable from DCS rashes secondary to embolic damage. Dive-related LRC, in contrast, is an inflammatory vasculopathic reticuloform rash and likely results from paradoxical VGE through RLS with obstructive embolic deposition in skin. In this sense, CM may be a misnomer for some diving-related rashes reported in literature.

Significance of Cutaneous DCS

A number of models have emerged to prevent, predict, and prognosticate DCS manifestations. The earliest is the Haldane decompression algorithm, which introduced diving ascent guidelines that markedly reduced incidence and severity of DCS.[53] Later probabilistic models improved understanding and prediction of DCS; but they did not predict severity of DCS or indicate probability of specific body system involvement.[54,55] The severity of DCS has historically been classified into Type I (minor) and Type II (major), a system pioneered by the U.S. Navy.[1] One study sought to discriminate DCS severity further by introducing the "perceived severity index (PSI)," a hierarchical list of six categories of symptomatology that are generally thought to correlate with morbidity/mortality. The list in decreasing order is: 1) serious neurological

(e.g., ataxia, areflexia, vision changes, unconsciousness); 2) cardiopulmonary (e.g., cough, hemoptysis, dyspnea); 3) mild neurological (e.g., numbness, tingling); 4) pain (e.g., aches, cramps, joint pain); 5) lymphatic/skin (e.g., edema, itching, marbling); and 6) constitutional/nonspecific (e.g., fatigue chills, restlessness).[55] The presence of skin near the bottom of the PSI underscores its historic widespread acceptance as a mild form of DCS disease.

In some cases of diving skin rashes, such as in CM/LRC or cold-induced urticaria, benign courses may be expected, However, LRC appears to be more than simply localized cutaneous DCS. LRC due to embolic inflammatory changes has been known to precede disastrous outcomes, up to and including death.[27] For this reason, it has been recently suggested hyperbaric skin DCS should be managed as severe Type II DCS and a potential medical emergency.[3,11] There is conflicting data regarding the importance and influence of early recompression in Type II DCS on outcome.[56–58] A study assessing early recompression therapy in Type I DCS showed improved symptomatic relief but no change in outcome.[59] Therefore, more investigation is warranted to determine which DCS patient subsets (considering patient demographics, diving context, etc.) would most benefit from treatment with hyperbaric oxygen therapy. Interestingly, a longitudinal study of North Sea divers revealed decreased health-related quality of life among individuals who had suffered from neurological DCS. Strikingly, they also discovered reduced health-related quality of life in divers who had suffered skin DCS. They speculated that severe skin symptoms may have detracted from concomitant but undetected neurological symptoms at the time.[60] This finding certainly supports the earlier associations discussed between skin and neurological DCS manifestations.

CM/LRC has historically been considered trivial, with expectation of spontaneous resolution. The existing classification schemes, including the Type I/Type II system developed by Golding and the more recent PSI, do not capture the nuance and significance of cutaneous DCS. Both systems, while useful stratification tools, are reductive and imply that cutaneous manifestations are localized and self-limited. However, our findings of an 84% coincidence with CM/LRC and RLS and a 57% coincidence with CM/LRC and neurological symptoms raise concern that cutaneous DCS is a manifestation of a severe underlying systemic vaso-occlusive process. Other studies noting the tight association of cutaneous and neurological symptoms further reinforce that CM/LRC may be a harbinger of clinically important cerebral embolism.

Therefore, it may be time to rethink this clinical sign given the emerging paradigm of paradoxical emboli and cerebral injury in patients with cutaneous DCS. This understanding may inform DCS screening practices and treatment algorithms in the clinical setting. Namely, the presence of CM/LRC should arouse suspicion for a severe underlying vaso-occlusive process with potential for devastating sequelae, including neurological injury and other end-organ damage. As such, it may be time for refinement of the DCS treatment algorithm given the notable absence of consensus guidelines for CM/LRC management. Currently, the majority of guidelines categorize skin symptoms as "mild DCS" and do not strictly necessitate aggressive interventions, such as immediate evacuation and recompression initiation.[1,61,62] The findings from this systematic review of CM/LRC in literature call for a reappraisal of management practices, specifically consideration of aggressive and immediate medical attention for those displaying signs of cutaneous DCS. In addition, we agree with Hartig et al.[11] that cutaneous DCS arising from embolic damage has been historically mischaracterized as CM and that cutaneous DCS should be classified as LRC.

ACKNOWLEDGMENTS

The authors thank Ron Strauss, Vasileios Kalentzos, Edmund Kerut, and Richard Moon for generously sharing their clinical images of diving-associated cutis marmorata featured in Fig. 2.

Financial Disclosure Statement: The authors have no competing interests to declare.

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