Cutis marmorata as a manifestation of decompression illness

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Abstract
We present images of skin lesions due to decompression illness (known as cutis marmorata). These alterations are usually transient, but they could be a warning sign of a more severe manifestation of decompression illness.

Introduction
Skin lesions are visual clinical manifestations of Decompression Illness (DCI). DCI has a wide range of manifestations and a type 1 DCI is usually characterized by musculo-skeletal pain and mild cutaneous symptoms. Common type 1 skin manifestations include itching and mild rashes (as distinct from a clear, mottled or marbled, and sometimes raised discoloration of the skin known as cutis marmorata).

Case Report
During recreational diving activities in Maldives cruise, a 51-year-old man presented with a cutaneous rash that developed about 1 hour after diving over 30 meters using air, with no omission of decompression safety stops. He denied any cardiovascular pathology (particularly, he did not report the presence of patent foramen ovale) and he had dived previously with no abnormalities. On physical examination, normal vital signs were noted along with a reticulated red-purple discoloration on the trunk and proximal superior extremities. Other physical examination findings were within normal limits.

The logistical situation, during the cruise, limited the treatment to normo-baric oxygen; the subject was treated with 100% oxygen administration and the rash resolved within about 1 hour with no other sequelae.

Discussion
DCI is a result of nitrogen accumulation within the body’s tissues, causing reduced blood flow and impaired oxygen delivery. Cutis marmorata is a distinct cutaneous manifestation indicative of systemic DCI pathology. The rash begins with cyanotic mottling and may quickly spread peripherally, becoming erythematous.

Histological analysis of cutis marmorata has shown areas of inflammation, localized vasoconstriction alternated with vasodilation, but no evidence of gas bubbles in the cutaneous microcirculation. Etiology of this form of DCI is somewhat controversial. In addiction to a “local” skin effect, several investigators recently postulated that a disruption of the brainstem vasomotor regulatory neurons occurs due to embolization of venous gas emboli into vertebral arteries. This disruption provokes inappropriate vasodilation and vasoconstriction in areas of the skin controlled by the affected neurons, as an autonomic dysregulation.

Therefore, the cutis marmorata could be considered as a heralding sign of more severe symptoms of central nervous system DCI.

Moreover, the association between patent foramen ovale and certain forms of DCI has been well established, and an association between cutis marmorata and the presence of patent foramen ovale has also been described.

Cutis marmorata after diving is usually regarded as a mild form of DCI (type I), and often not, or insufficiently treated. Some divers regard the occurrence of skin rash after dives almost as “normal” and often considered it as a local phenomenon. With prompt first aid using high-flow pure oxygen breathing, symptoms may rapidly disappear completely. Moreover, in case hyperbaric treatment is available, it should be administered as for any other central nervous system DCI.

A likely cause of DCI in our patient could be repetitive and multiple day diving, leading to an accumulation of residual nitrogen within tissues. Moreover, we suggested to the patient to perform a contrast echocardiography for detection of a patent foramen ovale.

Conclusions
On the basis of etiopathogenetic hypothesis suggested by several authors, cutis marmorata should no longer be considered a mild, innocuous form, but rather a serious neurological form, thus early diagnosis is essential. It could be a warning sign of a more severe manifestation of DCI and could represent a prodromal feature of potentially life-threatening complication, so that prompt and accurate recognition and careful follow-up is required.

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