Letter to the Editor

Do skin rash and *cutis marmorata* stem from lamellar bodies within the skin?

Cutis marmorata (CM) manifests as bluish-red spots on the skin following decompression. These are often itchy or painful to touch, and appear half to one hour after surfacing. The pathogenesis of skin lesions in decompression illness (DCI) remains unresolved. The common belief has been that bubbles that shunted to the arterial circulation reached the skin and clogged blood vessels. An alternative explanation from studies in which air was injected into the internal carotid artery of swine is that arterial bubbles at the brain stem disturb the control of skin blood flow, causing CM.[1] Other brain syndromes have also been seen to cause CM. It was suggested that bubbles affecting the brain stem result in the release of neuropeptides in the skin which control vasodilatation and vasoconstriction. However, this does not explain the inflammation in the skin lesions, with red blood cells, haemorrhage and neutrophil infiltrates. The percentage of right-to-left circulatory shunts in divers who suffered CM was 77% compared with 28% in divers with no record of CM, a finding which supports either of these explanations.[2]

Another study in swine concluded that there was "*strong evidence to support autochthonous bubbles as the etiology of skin lesions*".[3] Lesions appeared without right-to-left shunting. Skin thickness from the squamous keratin to the dermis increased by 10% in the affected areas. The lesions showed congestion, haemorrhage and neutrophil infiltrates. Superficial counter-diffusion as a cause of CM, the increased risk of CM in a dry as opposed to a wet dive and the prevalence of CM in proximity to subcutaneous fat (which acts as a nitrogen reservoir), all support an autochthonous origin.

Decompression bubbles can develop and expand only from pre-existing gas micronuclei. It is known that nanobubbles form spontaneously when a smooth hydrophobic surface is submerged in water containing dissolved gas. We have shown that these nanobubbles are the gas micronuclei underlying decompression bubbles and DCI.[4] After decompression, bubbles evolved at definite hydrophobic sites composed of the lung surfactant dipalmitoylphosphatidylcholine. Nanobubbles are formed on the surface of these lamellar layers of phospholipids, and on decompression expand into venous and arterial bubbles.

Lamellar bodies of phospholipids produced in the granular layer of the skin are used for the formation of a hydrophobic barrier at the cornified layer.[5] We suggest that the hydrophobic layers in the skin may be the site at which bubbles develop from nanobubbles and cause CM, just as occurs at the active hydrophobic spots on the luminal aspect of a blood vessel. This is the reason no bubbles were observed in the skin microcirculation. Unlike bubbles on the inner wall of venous blood vessels, which are supplied with high quantities of nitrogen from the incoming venous blood, the expansion of skin bubbles will be limited due to a low supply of nitrogen (possibly from the nearby subcutaneous fat). Therefore, skin bubbles should be small and have a short life span, which may be why they have hitherto remained undetected. The sensitivity of some divers to CM and its localization to specific skin areas may be related to individual variability in the lamellar bodies and phospholipid skin barriers.

Support for the present hypothesis may be found in the observation in some cases (though not all) of the movement of gas under the skin by means of echography (Balestra C, personal communication, 2018). CM is more frequent in female divers, and more so in subtropical than in cold European waters (van Ooij P-JAM, personal communication, 2018). This may be explained by women having more subcutaneous fat than men, coupled with the higher skin perfusion (and nitrogen loading) in warm water. This suggestion of possible autochthonous bubble formation in the skin does not exclude other causes, but may open a window for further investigation.

References

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