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An autopsy case of decompression sickness: Hemorrhages in the fat tissue and fat embolism

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Abstract: We present an autopsy case of decompression sickness (DCS). A man in his 50s who was a professional diver died of cardiopulmonary arrest on a ship after solo recreational diving with a hookah dive system. Although he was transported to a medical facility, he was confirmed dead approximately 1 h after cardiopulmonary arrest. At autopsy, the following findings were observed: skin discoloration with subcutaneous hemorrhages; diffuse bleeding in the epicardial fat, greater omentum, and mesentery; fat emboli in the kidneys; and numerous bubbles in the blood vessels. These findings, combined with those of the on-scene investigation, led to the conclusion that the subject died of DCS. The deceased had two distinctive findings related to fat: hemorrhages in the fat tissue and fat embolism. We suggest that these findings are vital reactions indicative of DCS.
Key Words: decompression sickness, decompression illness, fat tissue, fat embolism, diving, autopsy.

In diving, decompression sickness (DCS) occurs when a gas (usually nitrogen) accumulates in body tissues in a positive-pressure environment and, after subsequent reduction of ambient pressure, escapes into body fluids and forms bubbles [1].

DCS has been studied in great detail as diving techniques have progressed and underwater activities have become more common; however, criteria for autopsy diagnosis of DCS have not been well established. Therefore, diagnosis of DCS largely depends upon on-scene investigations [2,3].

Here, we present an autopsy case of DCS with diffuse hemorrhage in fat tissue and fat embolism in the kidneys after recreational diving with a hookah dive system. Previous reports of DCS have noted findings related to fat tissue [4–11].

Our case is discussed together with the results of the previous reports, to analyze the diagnostic value of our findings.

CASE REPORT

Case history

A man in his 50s who was a professional diver died of cardiopulmonary arrest on a ship after solo recreational diving with a hookah dive system. He was familiar with the equipment and regularly used the dive system, and its proper functioning was confirmed by the investigating authority. Because the patient dived alone and did not use a diving computer, a detailed diving log was not available.

Figure 1 shows the diving profile derived from the testimonies of the relevant parties on the ship; according to the testimonies, he dived twice. During the first dive, he descended to a depth of approximately 10 m for 30 min. After that, he rested on the ship for 10 min and then dived to a depth of approximately 20 m for 60 min.

Descent and ascent rates were unknown. After returning to the ship from the second dive, he stated that he

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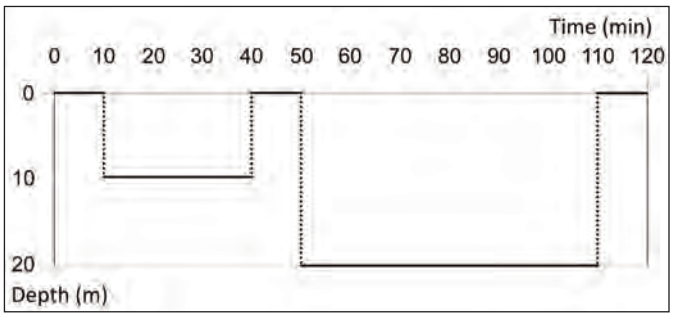

Figure 1. Diving profile derived from the testimonies of the relevant parties on the ship.

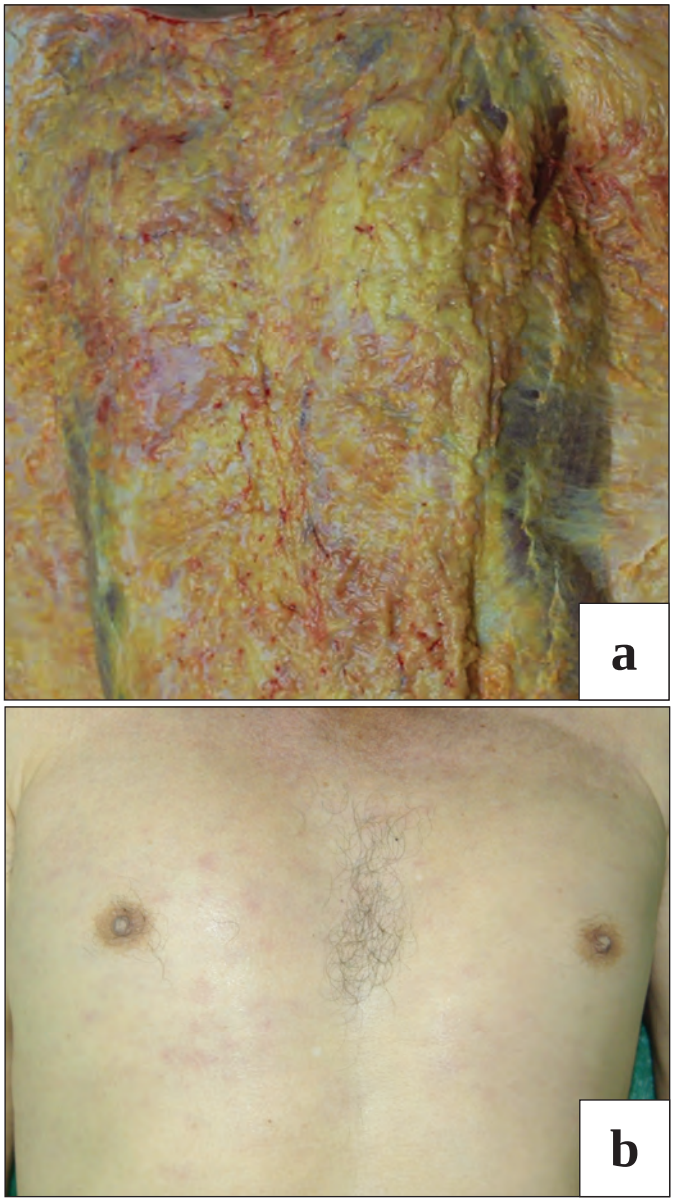

Figure 2. (a) Skin discolorations on the anterior side of the chest. (b) Slight hemorrhages widely observed in the subcutaneous fat tissue. The discoloration was thought to be caused by these hemorrhages.

might have ascended to the surface too rapidly. Soon after, he became unconscious and suffered cardiopulmonary arrest. Although he was transported to a medical facility and received cardiopulmonary resuscitation, he was confirmed dead approximately 1 h after cardiopulmonary arrest without compression chamber therapy. An autopsy was performed 22 h after his death.

Hookah dive system

The hookah dive system, which is also known as the surface-supplied dive system, supplies air to the diver using an umbilical cord from the surface. Similar to the self-contained underwater breathing apparatus (SCUBA) dive system, the air pressure is adjusted to the atmospheric pressure.

Autopsy findings

The length and weight of the body were 166 cm and 67 kg, respectively. Facial congestion was observed. A few petechiae were observed in both palpebral conjunctivae, whereas no petechiae were observed in the bulbar conjunctiva or mucosa of the oral cavity. The subject had a number of red skin discolorations with subcutaneous hemorrhages (Figure 2).

Subcutaneous emphysema and pneumothorax were not observed. Diffuse hemorrhages were observed in the greater omentum and mesentery (Figure 3a and 3b).

The heart weighed 350 g, and diffuse hemorrhages were observed in fat tissue under the epicardium (Figure 3c). Bubbles were released when the superior vena cava was dissected (Figure 4), and many bubbles were seen in the coronary veins.

Approximately 60% stenosis was observed in the coronary arteries. The foramen ovale was closed. Valvular function was normal, and no ischemic change was observed in the myocardium. The left and right lungs weighed 410 g and 490 g, respectively. A number of rice-grain-sized or smaller petechiae were observed in the pleura. No hemorrhagic or opacified areas were observed in the pulmonary parenchyma. The liver weighed 1930 g and was swollen and mildly fatty. Foamy blood flowed out from the dissected blood vessels when the liver was harvested. The gall bladder was also under tension, and gas was discharged when the organ was incised. Four greenish-brown calculi were found in the gall bladder. Numerous bubbles were observed in the veins of the small intestine. The brain weighed 1200 g, and there were no signs of cerebral swelling. Numerous bubbles were found in blood vessels on the surface of the brain. No cerebral hemorrhages or infarctions were detected.

Microscopically, no injuries that indicated pulmonary barotrauma were observed, and the alveoli in the lung sections appeared to be inflated. In the liver, unstained round cavities in the interstitium, and hepatocytes exhibiting hydropic degeneration, were evident at high magnification. Congestion was observed in the small intestine, and round cavities of various sizes that were considered to be microbubbles were seen in the veins of the small intestine. There was no aggregation of inflammatory cells in the small intestine. Fat emboli were not detected in the oil red O-stained brain and lung sections.

In oil red O-stained kidney sections, a small number of fat emboli were observed in glomeruli (Figure 5), arterioles, and tubules.

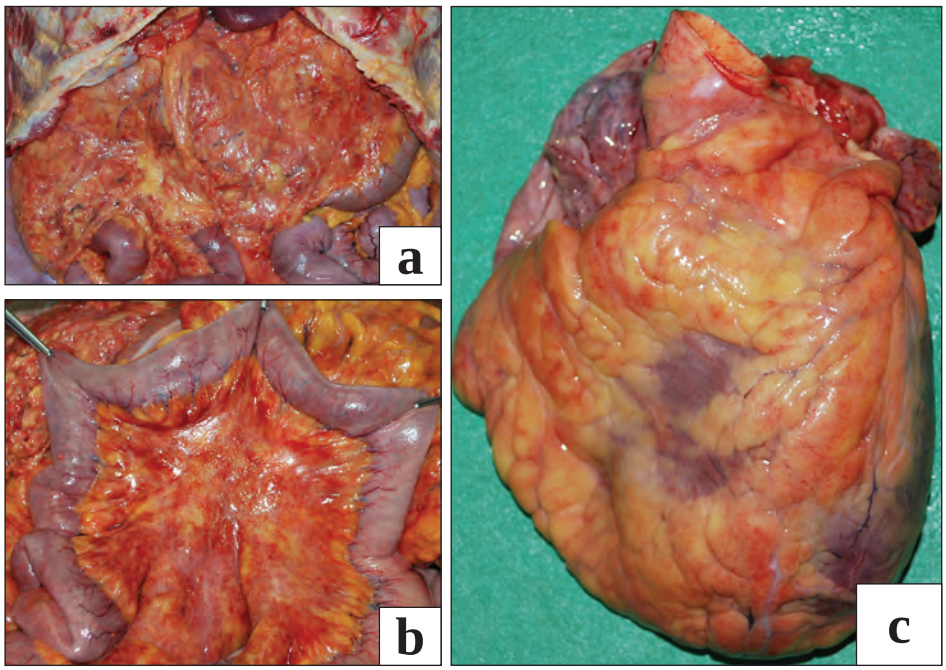

Figure 3. (a–c) Diffuse hemorrhages in fat tissue. Hemorrhages in the (a) greater omentum, (b) mesentery, and (c) epicardial fat tissue.

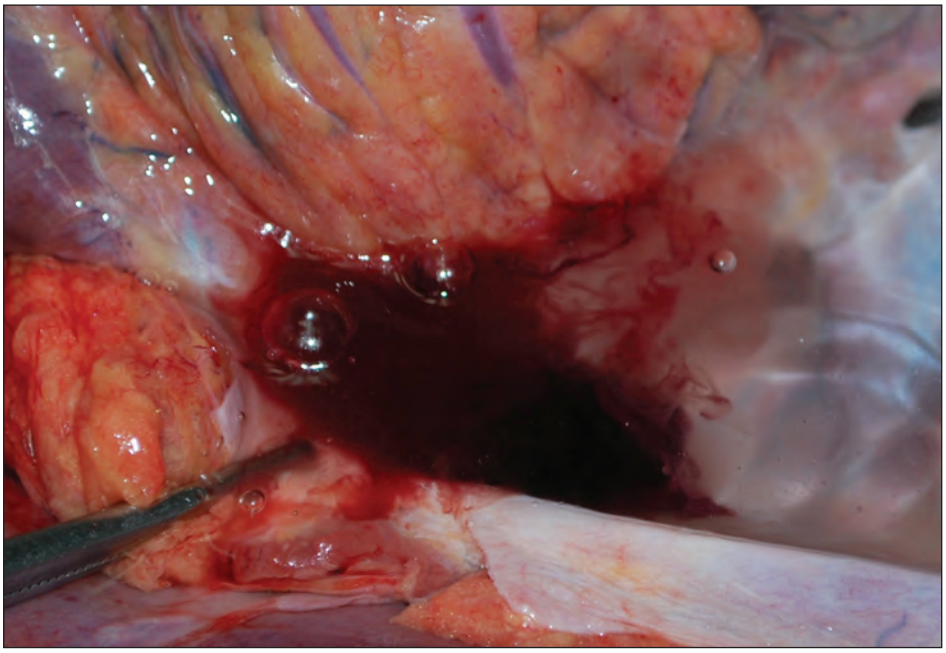

Figure 4. The inferior vena cava was dissected in the incised cardiac sac, which was filled with water; many bubbles were released along with blood.

Toxicological examinations including alcohol testing yielded no positive findings.

The cause of death was determined to be DCS on the basis of autopsy findings and evaluation of the scene.

DISCUSSION

DCS can be associated with a variety of symptoms; most are mild, such as joint pain and paresthesias, and resolve without sequel. However, DCS infrequently develops into a fatal condition called "chokes" (cardiorespiratory decompression sickness). Theoretically, bubbles produced by DCS enter the arterial circulation [1]. Nakayama *et al.* [12] reported that the rate of DCS occurrence was 1.9% among 3078 leisure divers, indicating that DCS is not rare among leisure divers in Japan.

In our case, we observed four characteristic findings: red skin discoloration, hemorrhage of fat tissues, fat emboli in the kidneys, and bubbles in the blood vessels. Red skin discoloration is one of the well-known signs of DCS [1], and persists after death. In our case, skin discoloration was caused by (and classified as) hemorrhage in the subcutaneous fat tissue. Previously, Möttönen *et al.* [4] reported macroscopic hemorrhage in epicardial fat in an autopsy case of DCS. In another report of DCS published by Kitano *et al.* [5], microscopic hemorrhages in the subcutaneous fat tissues were observed in autopsy cases and in experimental animals. They assumed that this occurred because fat tissues, which are thought to be the main storage compartment for nitrogen gas, were disrupted by bubble formation. In our case, the most distinctive findings were the hemorrhages observed in a range of fat tissues including subcutaneous fat, epicardial fat, and the greater omentum and mesentery. To our knowledge, no autopsy reports of DCS have described diffuse hemorrhages in the greater omentum and mesentery. Considering this information along with that of previous reports, we suggest that the aforementioned reactions are vital reactions, indicative of DCS.

In previous reports of autopsy cases of DCS death, fat emboli were occasionally observed in the lungs, brain, and kidneys [5–8]. Animal models of DCS have also demonstrated fat emboli in these organs [5, 9–11]. These findings are thought to be related to the high solubility of nitrogen gas in fat tissue, which is susceptible to bubble formation [5]. Consequently, fat emboli should be considered vital reactions indicative of DCS. We conclude that the fat emboli observed in the kidneys in our case were of the same kind, and that they constitute evidence for a diagnosis of DCS.

Bubbles in the blood vessels suggest DCS; however, care must be taken when diagnosing DCS by

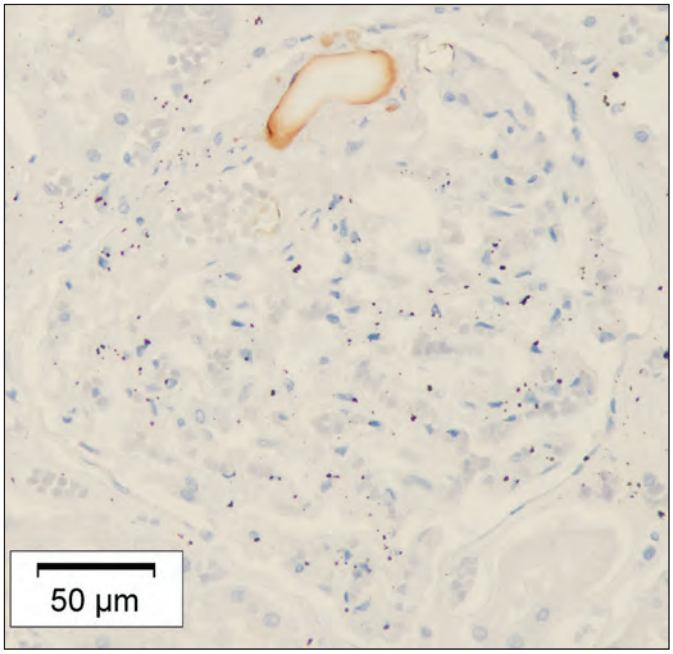

Figure 5. Oil red O-stained specimens. Fat emboli were observed in the glomeruli (×400).

this finding, because such bubbles may also be artifacts of the postmortem phenomenon called off-gassing [13, 14]. Off-gassing (or "postmortem decompression disease") occurs in deceased subjects who are raised from beneath the water to the surface. When the external pressure decreases, dissolved gas can come out of solution and produce bubbles. It has been demonstrated in both autopsy cases and animal experiments [15, 16] that intravascular bubbles are produced after death when a living body dies under pressure. Although Bajanowski *et al.* [17] suggested that gas analysis can be helpful in diagnosing fatal gas embolism, the method cannot distinguish antemortem gas formation from postmortem off-gassing in diving deaths because the gas is nitrogen in both situations. Thus, in diving deaths, the usefulness of this method is limited.

Conversely, another disorder that rapidly develops soon after diving is arterial gas embolism (AGE). In recent clinical practice, DCS has often been treated as decompression illness together with AGE because of the difficulty in distinguishing between AGE and DCS, and the similar treatment approaches to the two conditions [1]. At present, these two diseases are treated separately at autopsy, and bubbles in blood vessels and evidence of pulmonary barotrauma such as alveoli rupture, subcutaneous emphysema, and pneumothorax are regarded as autopsy findings of AGE [2]. That is, the autopsy findings of AGE and those of DCS partially overlap, and there may be cases where AGE and DCS cannot be clearly distinguished. In our case, the situation suggested AGE while the autopsy findings suggested DCS. However, we concluded that the cause of death was DCS because findings consistent with DCS were observed, while indicators of barotrauma, which strongly suggest AGE, were not found during autopsy.

In summary, we report an autopsy case of DCS. We observed the following findings at autopsy: skin discoloration with subcutaneous hemorrhages; diffuse bleeding in the epicardial fat, greater omentum, and mesentery; fat emboli in the kidneys; and numerous bubbles in the blood vessels. The deceased showed two distinctive findings related to fat: hemorrhages in fat tissues and fat embolism. From previous autopsy reports of DCS along with our autopsy results, it appears that these two findings are useful for autopsy diagnosis of DCS.

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