



H62 Hemorrhagic Death With Particular Postmortem Computed Tomography Findings Associated With Decompression Illness: A Case Report

Yui Igari, MD*, Forensic Medicine, Tohoku University, 2-1 Seiryomachi, Aoba-ku, Sendai, Miyagi 980-8575, JAPAN; Tomoya Ikeda, MD, Forensic Medicine, Tohoku University, 2-1 Seiryomachi, Aoba-ku, Sendai 980-8575, JAPAN; Tadashi Hosoya, MD, Forensic Medicine, Tohoku University, 2-1 Seiryomachi, Aoba-ku, Sendai 980-8575, JAPAN; Akihito Usui, MS, Tohoku University, 2-1 Seiryomachi, Aoba-ku, Sendai 980-8575, JAPAN; Yusuke Kawasumi, MD, PhD, Tohoku University, 2-1 Seiryomachi, Aoba-ku, Sendai 980-8575, JAPAN; and Masato Funayama, JAPAN

The goal of this presentation is to describe an unusual case of fatal intraperitoneal hemorrhage with Decompression Illness (DCI).

This presentation will impact the forensic science community by illustrating particular postmortem computed tomography findings associated with DCI.

DCI is characterized by a wide range of symptoms caused by decompression of the body and includes two syndromes: Arterial Gas Embolism (AGE) and Decompression Sickness (DCS). In AGE, alveolar gas (secondary to pulmonary barotrauma) or venous gas emboli (via a cardiac shunt or the pulmonary vessels) enters the arterial circulation. DCS is caused by *in situ* bubble formation from inert gas dissolved within body tissues. Clinical manifestations range from trivial to fatal.¹

This report describes a case involving a diver in his mid-fifties who was working at a depth of 5m below the sea surface. About ten minutes after he started working, there was an explosive sound, and he returned to the sea surface rapidly. He was conscious at that time, but said that he could not breathe and collapsed shortly thereafter. He was transferred to a hospital and underwent cardiopulmonary resuscitation, but he could not be resuscitated. A doctor in the emergency room identified air bubbles within the blood during blood sample collection.

Postmortem Computed Tomography (CT) revealed massive hemorrhagic ascites and a large amount of gas within the heart chambers and blood vessels of the brain, liver, and groin region. In particular, the heart appeared to be expanded with gas. A radiologist stated that all of the gas was not likely to have been produced by putrefaction.

An autopsy was performed approximately 18 hours after the patient's death. No antemortem injuries were apparent on the surface of his body. The autopsy revealed traumatic rupture of the mesentery and approximately 2,600ml of blood with soft blood clots in the abdominal cavity. Only 5ml of blood was present in the heart, and the liver and kidneys were ischemic. No postmortem putrefaction changes were visible. Visualization of gas bubbles within the circulating blood was impossible because too little blood remained within the blood vessels. No bubbles were present in the hemorrhagic ascites. The decedent weighed 77kg and such massive hemorrhage was undoubtedly fatal. Therefore, the autopsy report concluded that the cause of death was attributable to intraperitoneal hemorrhage caused by traumatic rupture of the mesentery.

The characteristic finding in this case was a large amount of gas in the cardiovascular system, which was detected on postmortem CT. According to the literature, AGE can occur after ascent from a depth as shallow as 1.0m to 1.5m, and the minimum diving depth after which venous gas emboli can form is 3.6m, whereas DCS is uncommon at depths of <10m.^{1,2} In the present case, some of the gas may have formed gradually after death and it was impossible to determine the extent to which the gas contributed to the patient's death. Therefore, it was concluded that the gas had formed secondary to DCI, but the influence of the gas on the patient's death was not clear.

Reports of DCI with such particular CT findings as described in this case are rare.

References:

1. Vann RD, Butler FK, Mitchell SJ, Moon RE. Decompression illness. *Lancet* 2011; 377: 153-64.
2. Eckenhoff RG, Olstad CS, Carrod G. Human dose-response relationship for decompression and endogenous bubble formation. *J Appl Physiol* 1990; 69: 914-8.

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