

The Distribution of Gas in a Patient with Cardiac Arrest due to Decompression Sickness Who Received Chest Compression

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Abstract: A 52-year-old Japanese diving instructor lost consciousness after 46 minutes of diving at a maximum depth 21 m with decompression performed via a regular decompressive table. He received basic life support, including activation of an automated external defibrillator once. On arrival, he remained in cardiac arrest. The rhythm was asystole. Advanced cardiac life support failed to obtain spontaneous circulation. Whole-body computed tomography (CT) immediately after ceasing resuscitation revealed multiple gas molecules in his body, mainly on the ventral side of the thoracic and abdominal wall in addition to in the right ventricle, the portal vein in the liver, the inferior vena cava, the descending aorta and the spinal canal. However, head CT showed no gas in the brain. This is the second report from Japan describing gas on CT images in a patient with cardiac arrest induced by decompression illness. Off-gassing from the tissue by decompression along with vacuum phenomenon by chest compression are the most likely explanations for the ventral truncal distribution of gases in this patient.

Key words: cardiac arrest; decompression illness; chest compression.

INTRODUCTION

Decompression illness (DCI) is caused by bubbles that appear in either the bloodstream or in other tissues in the body by ascending too fast from either underwater to the surface or from the surface to a high altitude, such as in high-altitude fighter pilots[1]. Gas collection on postmortem computed tomography (CT) were not visible or highly underestimated in the autopsy because the CT had high sensitivity to detect gas[2]. In Japan, there has been only one report describing the appearance of gas on CT images in a patient with cardiac arrest induced by DCI[3].

We herein report the distribution of gas in a patient with cardiac arrest due to DCI who received chest compression.

CASE REPORT

A 52-year-old Japanese diving instructor lost consciousness after 46 minutes of diving at a maximum depth 21 m with decompression performed via a regular decompressive table. He received basic life support, including activation of an automated external defibrillator once. When the staff of a physician-staffed helicopter encountered him, he was in a state of cardiac arrest. The initial rhythm was pulseless electrical

activity. He received tracheal intubation, with a venous route secured and 1 mg adrenaline infused every 4 minutes for 4 times in total, and he was transported to our hospital. Portable ultrasound revealed no bubbles in the inferior vena cava.

On arrival, he remained in cardiac arrest. The rhythm was asystole. After a fifth infusion of 1 mg adrenaline, asystole changed to ventricular fibrillation, so electrical shock was performed. This failed to obtain spontaneous circulation, and resuscitation was ceased. An arterial gas analysis revealed a pH of 6.500, PCO₂ of 44 mmHg, PO₂ of 134 mmHg, HCO₃⁻ of 9.8 mmol/l, base excess of -40.6 mml/L and a lactate level of 27 mmol/l. The main results of a biochemical analysis of the blood were as follows: white blood cell count of 13,500/μl, hemoglobin level of 15.3 g/dl, platelet count of 23.9 × 10⁴/μl, total protein level of 74.6 g/dl, aspartate aminotransferase level of 224 IU/L, alanine aminotransferase level of 268 IU/L, glucose level of 409 mg/dl, blood urea nitrogen level of 17.1 mg/dl, creatinine level of 1.76 mg/dl, creatinine phosphokinase level of 218 IU/l, C-reactive protein level of 0.37 mg/dl, activated partial thromboplastin time of 57.9 (27.5) seconds, international normalized ratio of prothrombin time of 1.35, fibrinogen level of 304 mg/dl and

fibrinogen degradation product level of 67.4 µg/ml. Whole-body CT immediately after ceasing resuscitation revealed multiple gas molecules in his body, the ventral side of the thoracic and abdominal wall, the right

ventricle, the portal vein in the liver, the inferior vena cava, the descending aorta and the spinal canal (Figure 1-5). However, head CT showed no gas in the brain.



Fig-1: Truncal computed tomography (CT) 1. CT revealed multiple instances of intravascular gas at the ventral side of the chest wall (arrow)



Fig-2: Truncal computed tomography (CT) 2. CT revealed multiple gas molecules in the right ventricle (arrow)

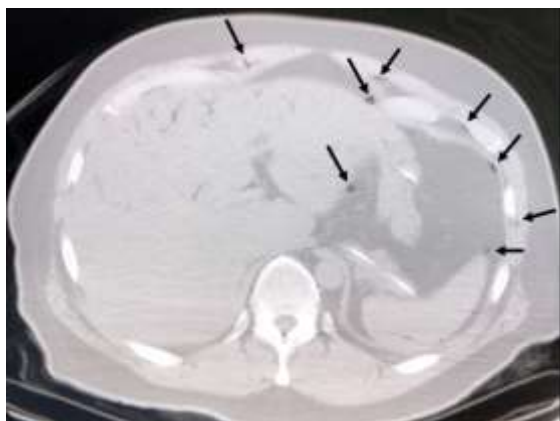


Fig-3: Truncal computed tomography (CT) 3. CT revealed multiple gas molecules in the portal vein at the liver and intravascular gas at the ventral side of the abdominal wall (arrow) and in the intra-abdominal fat



Fig-4: Truncal computed tomography (CT) 4. CT revealed multiple gas molecules in the portal vein at the liver and the expansion of gases at the ventral side of the intestine and pneumorrhachis (arrow)



Fig-5: Truncal computed tomography (CT) 5. CT revealed multiple gas molecules at the ventral side of the intestine and in the inferior vena cava and descending aorta (arrow)

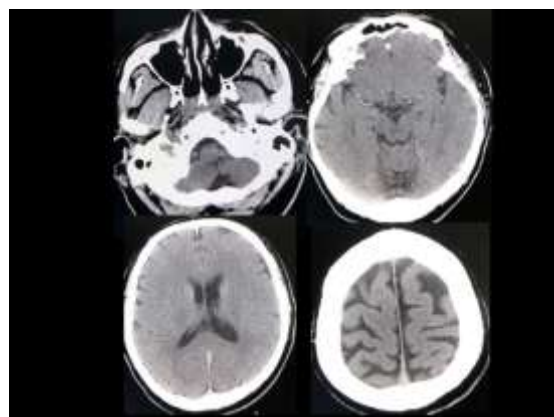


Fig-6: Head computed tomography (CT). CT revealed the loss of the corticomedullary junction but no gas in the patient's head

DISCUSSION

This is the second report from Japan describing gas on CT images in a patient with cardiac arrest induced by DCI. DCI is caused by bubble formation (off-gassing) from dissolved inert gas. Postmortem putrefaction or off-gassing phenomenon has been confirmed by previous reports of autopsy studies in patients who did not decompress [4]. In their study to differentiate between postmortem putrefaction/off-

gassing and DCI, Laurent *et al.* examined the postmortem putrefaction/off-gassing that occurred three hours postmortem in animal models using CT[5]. They confirmed that the gases that were observed could only correspond to DCI within three hours from cardiac arrest. The present patient had been in a compressive situation and showed gas at one hour from collapse. Accordingly, we diagnosed the gases in the present case as having been induced by DCI.

Gas in the disc space or joints is occasionally detected by CT in normal subjects and is termed the vacuum phenomenon (VP). The mechanism responsible for the VP is as follows: if an enclosed tissue space is allowed to expand as a rebound phenomenon after an external impact, the volume within the enclosed space will increase. In the setting of expanding volume, the pressure within the space will decrease. The solubility of the gas in the enclosed space will decrease as the pressure of the space decreases. Decreased solubility allows a gas to leave a solution [6]. The VP is typically observed at locations that have experienced traumatic impact [7]. Chest compression is a kind of traumatized impact; indeed, it can cause rib fracture, sternum fracture, pneumothorax and/or VP [8]. If the diver received chest compression immediately after diving, this impact might have caused bubble formation (off-gassing) from the dissolved inert gas [9]. Laurent *et al.* also showed that subcutaneous emphysema limited to the thoracic area presented a positive predictive value of 100% for chest compression [2,5]. Resuscitation maneuvers, such as chest compression or high-pressure ventilation, can cause barotrauma, resulting in the rupture of the alveolocapillar barrier and massive arterial embolism. However, such gases tended to locate in abundant quantities, especially in the cerebral circulation [2]. The present patient showed no gas in the brain, with the gas being primarily distributed at the ventral side of the trunk where chest compression had been applied. Accordingly, off-gassing from the tissue by decompression along with VP by chest compression are the most likely explanations for the ventral truncal distribution of gases in this patient.

CONCLUSION

This is the second report from Japan describing gas on CT images in a patient with cardiac arrest induced by decompression illness. Off-gassing from the tissue by decompression along with VP by chest compression are the most likely explanations for the ventral truncal distribution of gases in this patient.

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