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ACUTE DECOMPRESSION SICKNESS —REPORT OF AN AUTOPSY CASE—

BY

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ABSTRACT

An autopsy case of acute decompression sickness is reported. A 45-year-old fisherman was affected after diving 10 times 35 meters below the sea and died 30 hours after the disease developed. By autopsy, wide infarcts were found in the spinal cord, lung, heart and colon.

INTRODUCTION

Decompression sickness is principally caused by bubble formation in the body tissue and fluid from inert gas taken up during exposure and inadequately eliminated during decompression. The appearance and severity of the symptoms depend on the depth and duration of submersion, the rapidity of ascent and predisposing conditions. Most of the patients are successfully treated by adequate recompression and slow decompression, but some patients take a fatal course. Reports of autopsy cases in the literature are few (Okudaira and Nashimoto [1], Okuno and Hayashida [2], Kitano, Hayashi and Kawashima [3], Kitano and Hayashi [4]), and this paper is an addition of one more case.

CASE REPORT

A 45-year-old fisherman dived about 35 meters below the sea to catch lobsters in the morning of one December day around the Hachijo Island. He dived 10 times, when he complained of intrathoracic tightness and then lost his consciousness. When he respired after two hours of O_2 inhalation, he noticed paralysis of the lower extremities and localized pain of the right shoulder joint. He was transported by airplane in the afternoon and admitted to the Tokyo Medical and Dental University Hospital at 6:00 p.m. on the same day. He was accomodated into the high pressure chamber of 5 atmospheric pressure. Examinations revealed subendocardial ischemia, sensory paralysis spinal cord lower than Th-10, motor paralysis of the lower extremities, disappearance of the abdominal and Achilles' tendon reflexes and pain in the right shoulder. Neither pathologic reflex nor cloudiness of the consciousness were observed. On the next day at 7:00 a.m. he fell into a shock state and the electrocardiogram showed an anteroseptal infarction of the heart. At 9.00 a.m. the atmospheric pressure was decompressed to normal, but he was in a comatose state. At 2.50 p.m. he expired.

METHODS

The autopsy was performed routinely and the systemic organs were examined histologically. The preparations were

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stained by hematoxylin and eosin, and specific stains were done if necessary. They contained periodic acid-Schiff, Azan-Mallory, Masson trichrome, alcian blue, silver impregnation, Heidenhein iron hematoxylin, phosphotungustic hematoxylin and Klüver-Barrera myelin stain (luxol fast blue).

Autopsy Findings

Macroscopically the bubble formation was found in the superficial veins of the brain and spinal cord. The subarachnoidal veins of the spinal cord were congested. Demyelination and histolytic change of the spinal cord were found principally in the tractus corticospinalis lateralis, tractus spinocerebellaris, anterior funiculi of the cervical and thoracic spinal cords (Figs. 1, 2). These changes were not seen in the lumbar spinal cord. No softening lesions were found in the brain. The perivascular edema, hemorrhage, siderophages or lymphocytic accumulation of the vascular system were present in the brain and spinal cord.

In the lungs, multiple hen's egg-sized hemorrhagic infarcts were found bilaterally (left 450 gm, right 540 gm). Multiple thrombi were formed in the pulmonary arteries. Moderate edema and congestion were observed in the remainder of the lungs. There were hemosiderin-laden macrophages in the intraalveolar spaces. Rupture of the alveoli or irregular emphysema was observed in some parts (Fig. 4).

The heart was slightly hypertrophic and weighed 370 grams. Histologically moderate interstitial edema and slight atrophy of the myocardial cells were found. The degenerated myocardial cells showed a coagulated or granular appearance of the cytoplasm, disappearance of nuclei and cross striations, and they were mixed with the apparently intact cells. Inflammatory cell infiltration, hemorrhage and evident myocardial necrosis were not found. There were seen slightly organized thrombi in the branches of the left anterior descending arteries. Arteriosclerotic change of the coronary arteries was slight in degree.

Multiple, rice grain to broad bean-sized shallow ulcers were found in the colon (Fig. 5). Histologically there was ulceration of the mucosa, associated with neutrophile infiltration in the submucosal layer (Fig. 6).

Histologically bubble formation was found in the bone marrow of the vertebrae. In the thoracic vertebra, there were seen focal acellular areas, which were composed merely of the fat tissue (Fig. 7). There was vacuolar swelling and focal necrosis of the inner layer of the glomerular zone and in the outer layer of the fascicular zone of the adrenal glands (left 6 gm, right 6 gm). Erythro- and plasmastasis were present in the systemic veins, particularly in the central nervous system and lungs.

In the other organs, there were seen a fatty degeneration of the liver (1990 gm) and congestion of the kidneys (left 180 gm, right 170 gm) and spleen (150 gm).

Discussion

The chief manifestations of the decompression sickness consist of localized pain (bends), acute neurological, pulmonary (chokes) or circulatory disturbances, abdominal pain, skin reaction such as itching, formication, rashes mottling (marbling), aseptic bone necrosis and lasting neurological defects. The disease is usually non-fatal, but sometimes followed by unconsciousness, profound coma and death.

The major autopsy findings in the present case were injury of the spinal cord, early infarct of the heart and hemorrhagic infarct of the lungs, multiple ulcers of the

colon and focal necrosis of the adrenal cortex. Air bubbles were seen in the surperficial veins of the brain and spinal cord. Histologically air bubbles were present in the lung or bone marrow.

The decompression sickness is classified into the cardiopulmonary, spinal and cerebral types and the present case is classified into the spinal type. The lesions in the spinal cord were distributed in the tractus corticospinalis lateralis, tractus spinocerebellaris, anterior funiculi of the cervical and thoracic spinal cords. Kitano, Hayashi and Kawashima [3], and Kitano and Hayashi [4] examined in detail the lesions of the spinal cords of four autopsy cases. The changes in the spinal cord were usually located in the white matter, and the grey matter was affected only by the extension of the lesions emanating from the white matter. The thoracic segments were the sites of predilection.

In the cardiopulmonary type, the alveolar septum and pleura were ruptured over a wide area permitting a large quantity of air to enter the pleural cavity, mediastinum or the pulmonary circulation. The present case did not show any prominent destruction of the lung, but the electrocardiogram revealed the presence of the acute myocardial infarction of the heart. Histological findings of the heart seemed to be compatible with the acute ischemic change of a duration of 20 to 30 hours.

The multiple shallow ulcers of the colon might be related to the cause of the abdominal pain seen in the decompression sickness. The focal necrosis of the adrenal cortex might be ascribed to the circulatory disturbance which had occurred in the shock state.

In the bone marrow, a focal area with a loss of hematopoietic cells was present and it had a similarity to the aseptic bone marrow. However, aseptic bone marrow usually occurred a few months to a few years after the development of decompression sickness (McCallum and Walder [5]). Kawashima [6] reported an incidence of 59.5 percent of aseptic bone necrosis in 450 divers.

The exact mechanism of the pathogensis of decompression sickness remains obscure. It is generally agreed that the basic underlying pathologic process is the local formation of bubbles in the body tissue or fluid, both intravascular and extravascular. Under the condition of increased atmospheric pressure, nitrogen is more soluble in the organs or tissues rich in lipid content, such as the spinal cord, bone marrow and adipose tissue. The bubbles arising in the interstitium provoke tissue injury and could enter the circulation.

In the autopsy cases of decompression sickness, thrombi are found rather frequently. Although the thrombi were not seen in the present case, they are considered as one of the causal factors of the injury of the spinal cord. Usually the meningeal vein and epidural venous plexus are engorged. As to the pathogenesis of the spinal injury, firstly the tissue is destroyed by the autochtonous bubble formation and secondly by a severe circulatory disturbance, especially in the venous return associated with thrombi formation.

Recently, the significance of fat embolism is considered in the development and progression of decompression sickness (Kitano and Hayashi [9], Antopoi et al. [7]). Kitano, Hayashi and Kawashima [4] reported an autopsy case with widespread fat embolism. Cockett, Nakamura and Frank [8] found bone marrow emboli and numerous fat droplets in the pulmonary arteries, arterioles and capillaries in most cases of the experimental animals. Kitano, Kawashima and Hayashi [9] also reported the experimental studies on the hyper-

coagulability of the blood in decompression sickness.

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EXPLANATION OF FIGURES

Fig. 1. Demyelination in both the lateral and anterior funiculi at the 10th level of the thoracic spinal cord. Klüver-Barrera myelin stain, ×8.5.

Fig. 2. Demyelination and histolytic change at the 1st level of the thoracic spinal cord. Klüver-Barrera myelin stain, ×120.

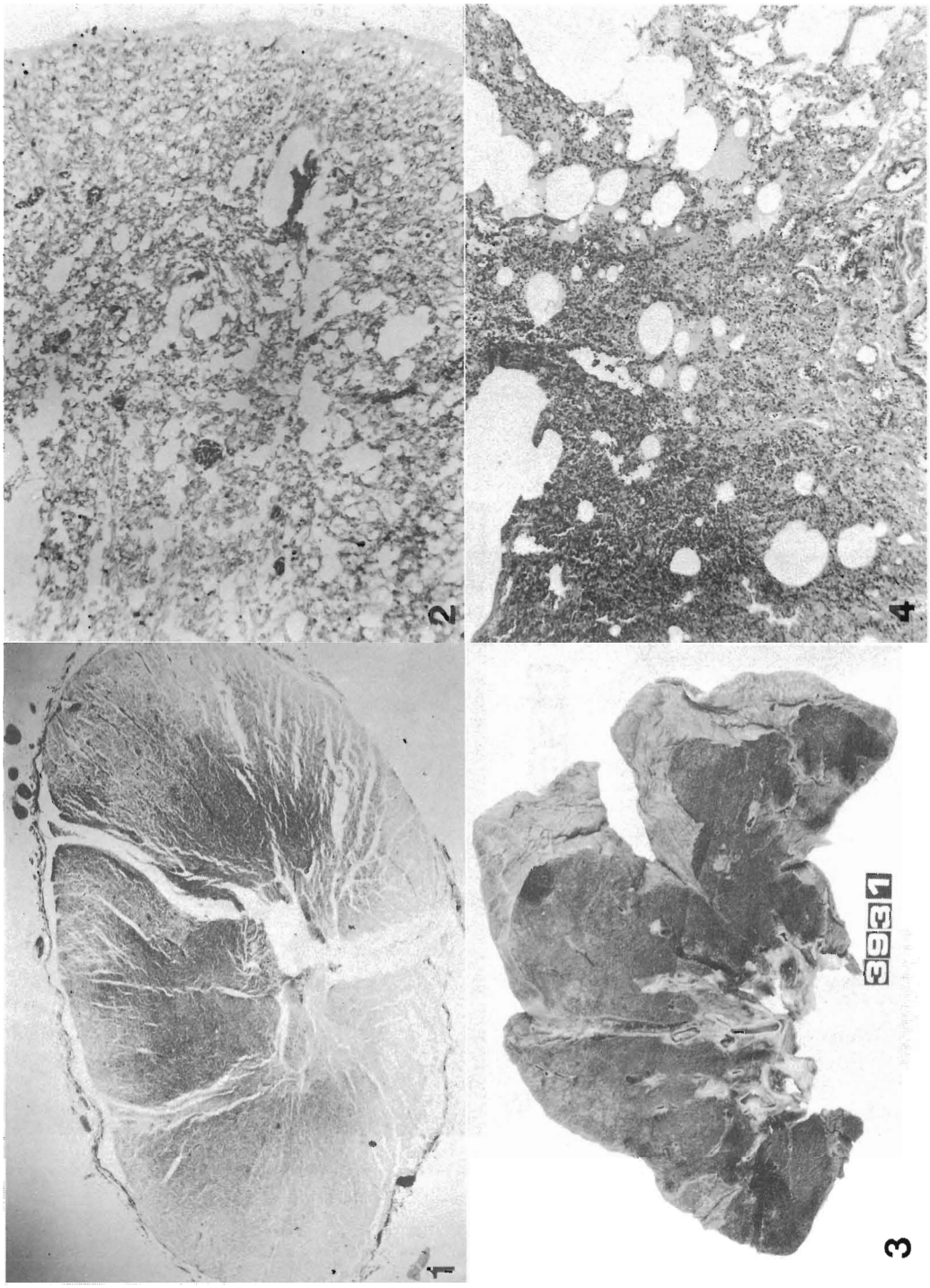
Fig. 3. Scattered hemorrhagic infarcts of the right lung.

Fig. 4. Hemorrhagic infarct and rupture of the alveolar walls of the lung. Hematoxylin and eosin stain, ×40.

Fig. 5. Multiple shallow ulcers of the colon.

Fig. 6. Submucosal bleeding and edema around the ulcer of the colon. Hematoxlyin and eosin stain, ×40.

Fig. 7. Focal acellular area of th thoracic vertebra. Hematoxylin and eosin stain, ×8.5.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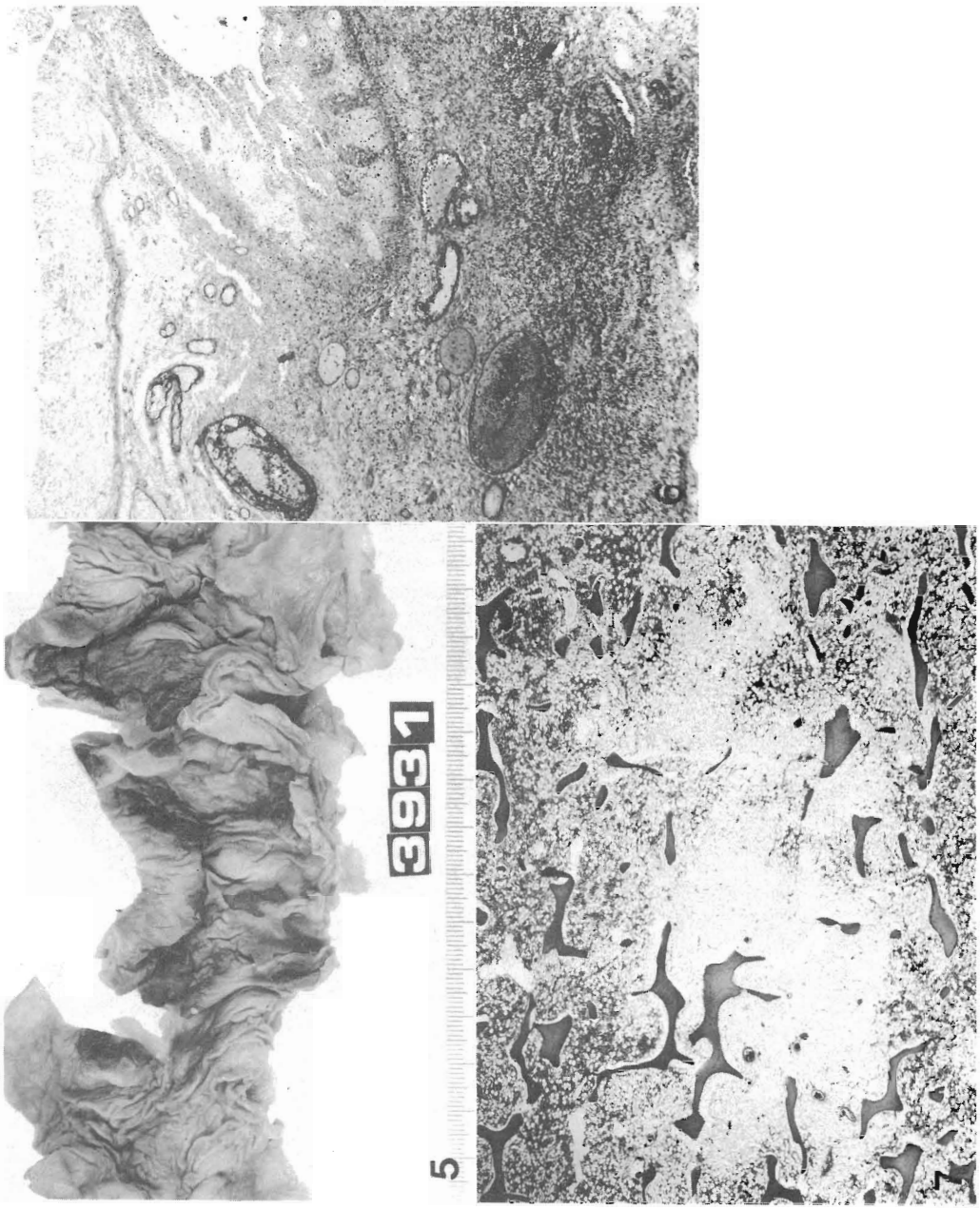
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