Three Autopsy Cases of Acute Decompression Sickness
—Consideration of Pathogenesis about Spinal Cord Damage in Decompression Sickness—

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Abstract

The authors report three autopsy cases of acute decompression sickness. Two of three cases showed marked necrosis with edema in the spinal cord. Microscopic examination disclosed thrombus formation in the parenchymal, arachnoidal and epidural vein systems in associated regions.

It is suggested that the processes of the spinal cord damage were the result of two different stages of tissue injuries: the first is more or less tissue destruction due to autochthonous bubble formation and the second is severe circulatory disturbance especially the disturbance of venous return from the spinal cord.


Introduction

Decompression sickness is an illness most frequently seen in divers and Caisson workers that results from a reduction of environmental pressure sufficient to cause the formation of bubbles from gasses dissolved in blood and tissues. Clinically, almost 24 percent of all cases of decompression sickness due to diving manifest some spinal cord dysfunction. Tragic somatic and socioeconomic impacts by neurologic disabilities were laid down among these persons.

The pathogenesis of spinal cord damages in decompression sickness has been the subject of controversy. The conventional view would ascribe tissue damage to arterial bubble embolization with consequent obstruction of arterioles and capillaries. Recently, another viewpoint is proposed by Hallenbeck and associates. They described that the venous obstruction leading to venous infarction is the most causal factor of the spinal cord damage in decompression sickness.

This report is based upon three autopsy materials of acute decompression sickness. The purpose of this study is to consider about the pathogenesis of the spinal cord damage in decompression sickness.

Cases and Autopsy Findings

Case 1, a 38-year old male helmet diver. This man dived to a depth of 40 meters for four hours, surfaced in 20 minutes. While surfacing he lost his consciousness and expired soon after.

Autopsy findings: Autopsy was performed seven hours later. There were numerous gaseous bubbles in the blood in the right atrium and ventricle of the heart, vena cava, portal veins of the omentum, superficial veins of the brain and the epidural vein plexus in the vertebral canal. In addition, blood seemed somewhat concentrated. Severe cyanosis was noted in the body surface. Extensively marked
congestion, edema and intraalveolar hemorrhage were found in the lungs and remarkable congestion and edema were presented in the general visceral organs. There was microscopically characteristic vacuolization due to retardation of gaseous bubbles in the lumina of the sinusoids in the bone marrow cavities. One of the most interesting findings was platelet aggregation in the vicinity of the bubbles in the sinusoids.

Several, small, up to 1,000 microns diameter, round spaces which were unstainable by all dyes used in the laboratory were found in the brain and spinal cord, especially in the white matter. These spaces should be occupied by bubbles created by decompression.

Case 2, a 36-year-old male SCUBA diver. This case had a history of diving four times to a depth of 60 meters for 30-40 minutes each time accompanied by an interval between dives for 10-15 minutes. He complained of pains in both legs after surfacing from the last dive, and 20 hours later, he was transferred to Kyushu Rosai Hospital. Physical examination showed complete sensory loss and flaccid paralysis of the bilateral lower extremities and hypesthesia and weakness of muscular power of the bilateral upper extremities. Slight dyspnea was also noticed. He had marked hematemesis resulting in shock state while in recompression therapy and, in spite of various attempts, improvement from shock state has never occurred. He died five days after the onset of disease.

Autopsy findings: In gross inspection, no bubbles were visible in the blood vessels. Hemorrhagic erosive esophagitis and gastritis were found. Marked congestion, edema and aspiration of blood were noted in the lungs with slight bronchopneumonia. Other visceral organs including the brain showed congestion, edema and petechial bleeding, also.

Microscopically, some of the sinusoids in the bone marrow cavity of the femoral head were expanded by containing gaseous bubbles in relatively massive volume, accompanied with thrombus formation adjacent to the bubbles and with tissue necrosis around the sinusoids. Focal necrosis without marked inflammatory reaction was seen in the hepatic lamina of the cortex of the cerebrum and the granular layer and Purkinje cells of the cerebellar cortex.

Spinal cord involvement was the most conspicuous, but the middle half of the cervical cord (C-3, 4, 5 & 6) was not available for examination. Congestion and edema, coincidental hemorrhage were marked in the entire parts of it. Small wedge-shaped edematous histolytic foci were seen in the lateral and posterior funiculi in the lower cervical segments (C-7 & 8). In the lower lower segments of thoracic cord (T-7, 8, 9, 10 & 11), wide-spread edematous histolytic foci with central cavity formation existed. The anterior funiculus was involved at these levels. Neither gliosis nor mesenchymal cell infiltration was noted in and around the foci. Another important finding confirmed by serial sections was thrombosis in the perifocal venules, arachnoidal veins and epidural venous plexus. Thrombosis was relatively frequent finding in the regions of the thoracic level of the thoracic cord. The grey matter was maintained in relatively good condition.

Case 3, a 20-year-old male SCUBA diver. This case had a history of diving to a depth of 50 meters for 20 minutes. Twenty minutes after surfacing, he complained of numbness of both legs and dyspnea. Then he was transferred to Kyushu Rosai Hospital. Physical examination clarified complete sensory loss till the 3-4th cervical dermatome. Pyramidal si-
signs were seen in all extremities showing flaccid paralysis and in the respiratory muscles resulting in inability of automatic respiratory movement. His consciousness was maintained in rather good condition. He showed some improvements by recompression therapy, however, 15 days after the onset of the symptoms, he expired.

Autopsy findings: Intravascular bubble was not visible to the naked eye at the time of the autopsy. Slight to moderate congestion was noted in the general visceral organs Cystitis was marked.

Histological examinations revealed marked congestion with associated thrombus formation in the small vessels in the lungs. Fibrosis with slight hemorrhage was seen in the bone marrow cavity of the femoral head in association with stagnation of some amounts of air bubbles in the sinusoidal lumina accompanied by thrombus formation.

Marked edematous rarefaction with petechial bleeding was seen in the parenchyma of the spinal cord, especially severe in the cervical cord (C-2, 3, 4, 5, 6, 7, 8) and 10th segment of thoracic cord. Histolysis with cavitation in the lateral and posterior funiculi was extensive in the third cervical segment. Grey matter, however, relatively well preserved its essential tissue construction. Foam cell accumulation, in moderate degree, in the peripheral areas was seen. But basic nature of his-
Photo. 1: Dilated sinusoids including air bubbles in the bone marrow cavity of femur of Case 1. Platelets aggregation is marked adjacent to the bubbles. Ceroidin section, H & E, x 50.

Photo. 2: Relatively large vacuole supposed as autochthonous air bubble in the white matter of the parietal lobe of cerebrum. Case 1. Ceroidin section, H & E, x 250.

Photo. 3: Dilated sinusoids including air bubbles. Thrombus formation is predominant in the vicinity of the bubbles. Necrotic process is observed in the surrounding layer. Femur head of Case 2. Ceroidin section, H & E, x 50.

Photo. 4: Transverse section of the spinal cord at T-10 level of Case 2. Refraction due to edema and histolysis is marked in the lateral and posterior funiculi. Ceroidin-paraffin section, Klüver-Barrera, x 11.

Photo. 5: Thrombosis in the venule of the posterior funiculus of the thoracic cord (T-3) of Case 2. Edema and histolysis are marked in the surrounding tissue. Ceroidin-paraffin section, Elastica Van Gieson, x 250.

tological changes in the spinal cord and in the vein system was the same as in Case 2.

Discussion

Spinal cord involvement in divers and Caisson workers has been a commonplace. The changes in the spinal cord have usually been confined to the white matter. The thoracic segments were the sites of predilection, with the lateral and posterior funiculi suffering that order of frequency. Grey
matter of the spinal cord was said to be affected only by extension of the lesions emanating from the white matter.

In the white matter of the three cases described, there were two kinds of lesions representing different stages of duration of illness. The one is autochthonous bubble formation in the white matter of the spinal cord as observed in Case 1. Another is remarkable edema and petechial bleeding with subsequent tissue necrosis and cavity formation as observed in Cases 2 and 3.

A few, round, small spaces which were unsustainably for all dyes used in the laboratory were found in the white matter of the spinal cord of Case 1. In the literature one finds full accounts of presumed autochthonous bubble formation in the white matter and also in the grey matter of the spinal cord. Berg (1878) and Hayashi (1974) claimed to have seen the bubbles in the interstitium of the spinal cord. Catsaras and other investigators also observed them, by stating that they were in vessels.

The spinal cord especially the white matter is more subject to autochthonous bubble formation by its richer lipid content. The solubility of nitrogen gas is greater in the fat and lipoids. They are capable of holding five times as much in solution as the non-fatty tissues. The solubility of nitrogen within all tissues increases as the atmospheric pressure rises. During decompression, massive bubble are created in the fat- or lipid-laden tissues.

So, it is appropriate to consider that the autochthonous bubble formation should directly result in more or less tissue destruction of the spinal cord, although the authors have no any other detailed data in direct support of this view.

Another point to be considered is the development of widespread profound circulatory disturbances in the spinal cord regions in decompression sickness. There were striking engorgement of meningeal veins and epidural venous plexus accompanied with edematous swelling of the entire parts of the spinal cord in all cases described. In Case 1, marked retardation of numerous gaseous bubbles was found in the epidural veins. In Cases 2 and 3, there were lesions of an edematous histolytic nature chiefly in the lateral and posterior funiculi of the spinal cords. The spacial disposition of the parenchymal lesions in Cases 2 and 3 seemed to be closely related to the venous thrombosis of intramedullary, meningeal and epidural vein systems. It was un-
able to detect the arteriolar or arterial alteration in and around the spinal cords in these cases.

Hallenbeck and associates (1975) found out the fact that in the experimental animals, regions of the epidural vertebral venous plexus become obstructed by intravascular air bubbles during the course of severe decompression leading to spinal cord injuries. The results strongly suggested that venous obstruction at this level and the consequent impairment of venous drainage of the spinal cord have a causal role in the production of spinal cord lesions in decompression sickness.

It is generally accepted that bubbles are initially liberated from tissues and blood during decompression. The bubbles that arise in the interstitium create tissue injury and are also delivered into the circulation. Vascular permeability increases due to intimal damage by intravascular bubbles resulting in plasma loss and interstitial edema. In the vessels the bubbles begin to exert direct mechanical effects including above-mentioned intimal damage and indirect effects due to surface activity at bubble-blood interfaces. The bubble-blood interaction as discussed by many investigators tends to alter the secondary and tertiary configuration of blood proteins, leading platelet aggregation, activation of the clotting system, release of vasoconstrictive substances, and finally disturbance of blood circulation due to hemococoncentration and red cell clumping. When the bubbles that arise in the interstitial spaces enter the circulation, other products of cellular and tissue disintegration such substances as lipid free fatty acid, peptides, histamine-like substances, potassium ion, etc. also enter. They all may play a role as agents of circulatory disturbance, together with intravascular air bubbles.

There are many fatty tissues in the epidural spaces in the region of the spinal cord. Massive nitrogen bubbles liberated from the epidural fatty tissue may gain the epidural venous plexus, and through obstructive action together with blood clotting phenomena retardation of venous return from the spinal cord may take place.

Under the extreme condition, the retardation of the venous return from the spinal cord may be aggravated through back pressure from bubble-laden pulmonary vessels and/or through right cardiac failure.

As for the cause of the spinal cord damage in Cases 2 and 3, the most important factor should be the increase in intramedullary venous pressure, which is mainly due to disturbance of venous return from the spinal cord.

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References

7) Haymaker, W, and Johnston, A. D.: Pathology of decompression sickness A co-
We treated 3 cases having “vertigo”, which seemed to occur by intermittent vertebral artery compression syndrome.

In the 3 cases, a 34 years old female, a 39 years old female and a 45 years old male, all patients complained mainly vertigo at extention of the neck, nausea and headache.

We observed central vestibular disorder in neuro-otological examination. And also vertebral angiography showed abnormality.

As treatment to 2 cases we performed cervical anterior spinal fusion and on vertebral artery performed Powers and Hardin & Poser operations.

At 1 case we adopted conservative therapy mainly by stellate ganglion block.

In 3 cases the symptoms occured by intermittent vertebral artery compression syndrome were improved.