CLINICAL

Histopathology of the early stage of osteonecrosis in divers

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Kawashima, M., K. Hayashi, T. Torisu, and M. Kitano. 1977. Histopathology of the early stage of osteonecrosis in divers. Undersea Biomed. Res. 4(4): 409–417.—This investigation was based on the histopathological examination of the femoral heads taken from four divers who died of decompression sickness. In Cases 1 and 2, where the clinical course was most acute, there was platelet aggregation adjacent to the air bubbles, slight-to-moderate bleeding with congestion of bone marrow, and sludging of erythrocytes in the dilated sinusoids. In Case 3, where the patient died 5 days after diving, rather extensive necrosis in the bone marrow surrounding expanding sinusoids included air bubbles. Platelet aggregation and agglutination or platelet thrombi were formed in the vicinity of the air bubbles. In Case 4 in which the diver died 14 days after the onset of decompression sickness, air bubbles and thrombosis were found in the sinusoids of the femoral head. Around them, phagocytic activation and fibrosis took place, which seemed to represent repair of necrotic bone marrow lesions. The sinusoid system of bone marrow can easily become obstructed by intravascular air bubbles after decompression. In the sinusoid system, bubbles probably exert direct mechanical effects and also indirect effects due to various thrombogenic activities. The early stages of osteonerosis in divers might be intimately related to circulatory disturbances, especially of the sinusoid system.

platelet aggregation
decompression sickness

Ostoenecrosis in workers exposed to compressed air was first described by Bornstein and Plate in 1911 and independently by Bassoe in 1913. In 1939, Kahlstrom, Burton, and Phemister reported on four cases of multiple osteonecrosis in caisson workers; one of these patients was examined at autopsy and the pathological features of bone were described. The first case of a diver with osteonecrosis was reported by Grutzmacher in 1941.

In our recent surveys in Japan, radiological investigation revealed 72 cases (53.3%) of osteonecrosis among 135 divers who had been admitted for the treatment of decompression sickness and 268 cases (59.5%) of osteonecrosis among 450 divers who had been examined radiologically at Ohura, Saga Prefecture, Japan. Osteonecrosis in commercial divers has become a serious medical problem in Japan.

The etiology of osteonecrosis in divers is not clearly understood. There is little information on the pathological changes in the early stages of osteonecrosis. The purpose of this study is to

throw light on the pathogenesis of osteonecrosis in divers. This investigation was based on the histopathological examination of the femoral heads taken from four autopsy cases in which the divers died of acute decompression sickness (Table 1).

TABLE 1
AUTOPSY CASES, DECOMPRESSION SICKNESS

Case	Age, yr	Sex	Interval between last dive and death
1	38	Male	Immediate
2	28	Male	10 hours
3	36	Male	5 days
4	20	Male	14 days

CASES AND HISTOPATHOLOGICAL FINDINGS

Case 1, a 38-year-old diving fisherman

Clinical diagnosis of the Case 1 diver was decompression sickness with chokes (pulmonary and cardiac shock). He had dived and stayed on the sea bottom at 40 m for 4 h and then ascended to the surface in 20 min. When he surfaced, he was already in a syncopic state; he died soon after.

At autopsy, there were so many air bubbles in the right cardiac lumen and veins of the entire body that the blood looked like foamy beer. Remarkable congestion and hemorrhagic infarction were seen in the lung. Most parts of the bone marrow cavity of the femoral head were replaced by fatty marrow and hemopoietic foci were scarce. Multiple, round-shaped air bub-

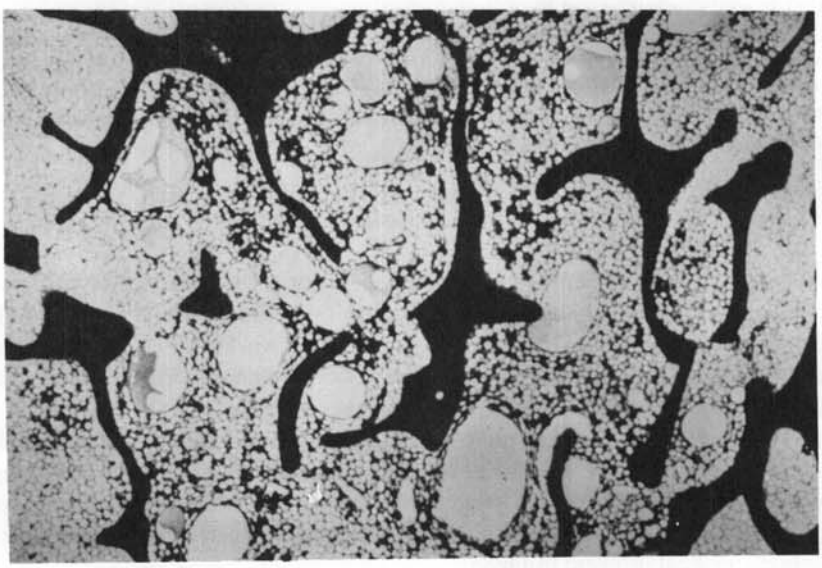

Fig. 1. Femoral head. Large, dilated sinusoids including air bubbles are noted in the bone marrow cavity. Case 1, hematoxylin and eosin, × 15.

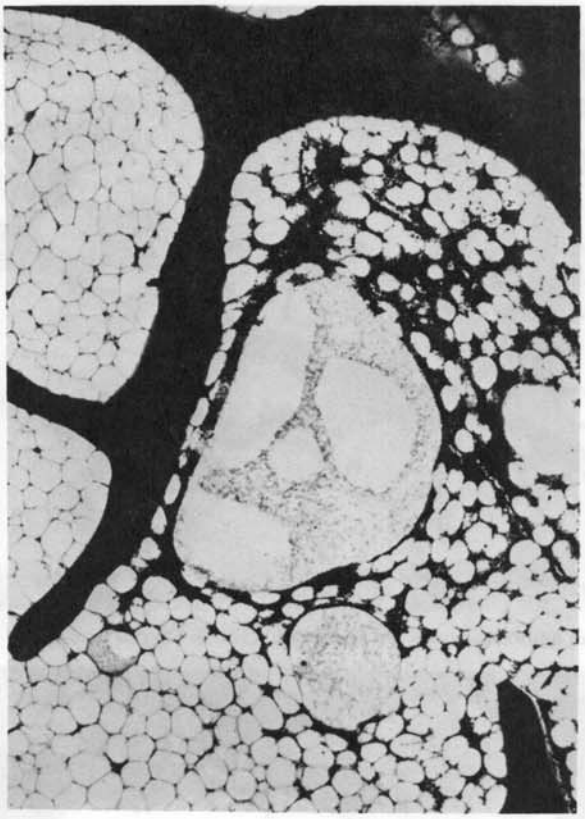

Fig. 2. Dilated sinusoids in femoral head. Platelet aggregation in vicinity of air bubbles is seen in lumina. Case 1, hematoxylin and eosin, × 50.

bles, up to 1500 μm in diameter, unstainable by all dyes used in the laboratory, were found in the lumina of sinusoids, especially around the hemopoietic foci (Fig. 1). A characteristic finding was platelet aggregation in the vicinity of the bubbles (Fig. 2). Marked congestion and aggregation of erythrocytes, which seemed to be the result of blood-cell sludging, were also frequently seen.

Case 2, a 28-year-old diving fisherman

Clinical diagnosis of Case 2 was decompression sickness with chokes and spinal cord paralysis. This fisherman dived repeatedly (seven times) to a depth of 30 m for a total of about 6 h. When he surfaced after the last dive, he complained of a slight paralysis of the extremities and dyspnea. The dyspnea gradually became severe. He died 10 h after onset of these symptoms.

Case 2 revealed multiple gas bubbles in the veins of the cerebral surface and in the abdominal mesentery at autopsy. Multiple gas bubbles were found in the large joint cavities. Marked congestion, edema, and hemorrhagic infarction were seen in the lung. Fat emboli were seen in the lung vessels (Fig. 3). Necrosis of the spinal cord was another major finding. The lesions in the femoral head were almost the same as in Case 1. Air bubbles, platelet aggregation, and an accumulation of fat were seen in the dilated sinusoids (Fig. 4).

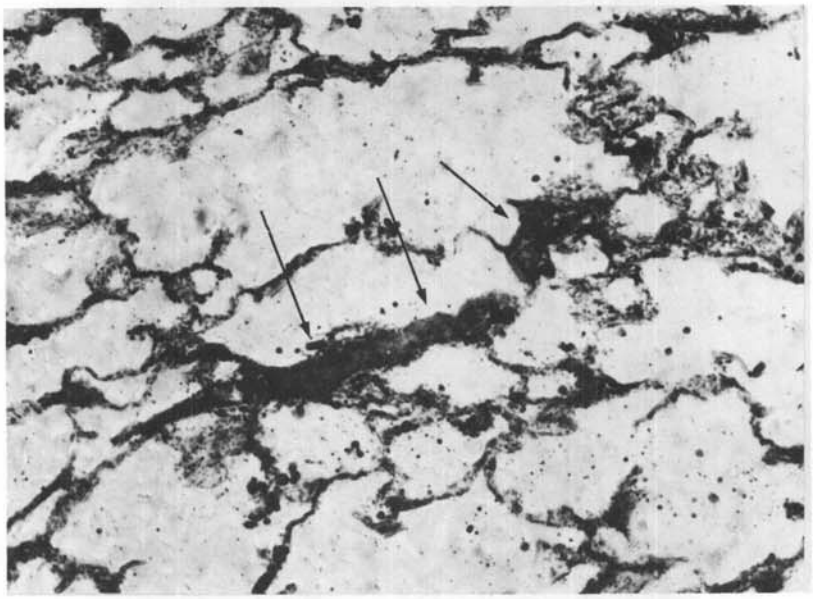

Fig. 3. Lung. Fat emboli (arrows) are scattering. Case 2, Oil red 0, × 50.

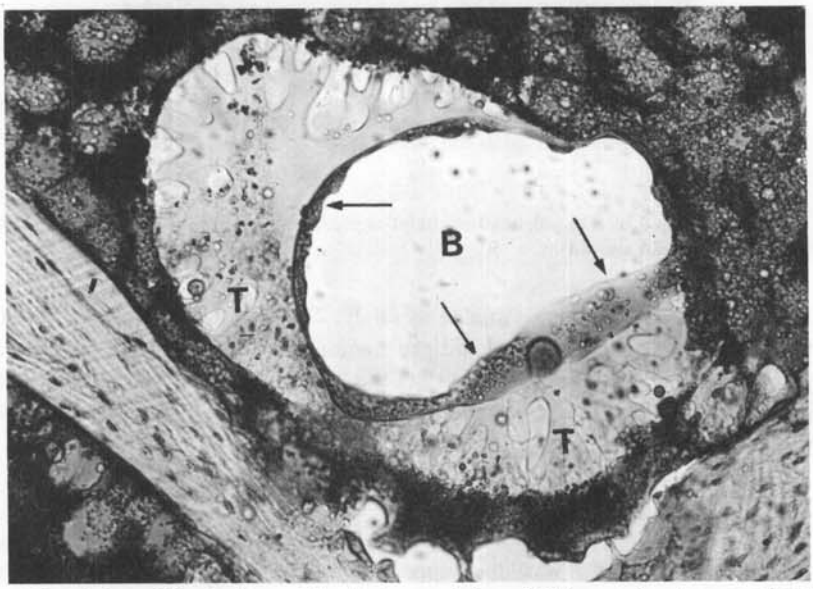

Fig. 4. Dilated sinusoid in the femoral head. Accumulation of fat (arrows) and aggregation of platelets (T) are noted in vicinity of air bubbles (B). Case 2, Oil red 0, × 50.

Case 3, a 36-year-old diving fisherman

Decompression sickness with spinal cord paralysis was the clinical diagnosis of the Case 3 diver. He dived repeatedly (4 times) to a depth of 60 m for a total of 40 min. After the last dive he complained of paralysis and dyspnea. He died 5 days after the onset of the disease, in spite of treatment with recompression in the hyperbaric chamber.

Marked congestion and edema were seen in the lung. There was widespread necrosis associated with multiple venous thrombi in the thoracic spinal cord. No air bubbles were found in the large vessels at autopsy. Sinusoids, especially of the subchondral layer of the femoral head, were expanded by air bubbles. A rather extensive necrosis was noted around these sinusoids (Fig. 5). Platelet aggregation or platelet thrombosis was seen in the dilated sinusoids (Fig. 6).

Case 4, a 20-year-old diving fisherman

Clinical diagnosis of Case 4 was decompression sickness with spinal cord paralysis. This fisherman had dived to a depth of 50 m for 20 min. Twenty minutes after surfacing, he complained of paralysis and dyspnea. In spite of recompression therapy, he died 14 days after diving. Cervical spinal cord necrosis was observed. Air bubbles and thrombus formation were found in the sinusoids, especially of the subchondral layer, of the femoral head. Slight hemorrhage, migration of phagocytes, and fibrosis were found around the involved sinusoids (Fig. 7).

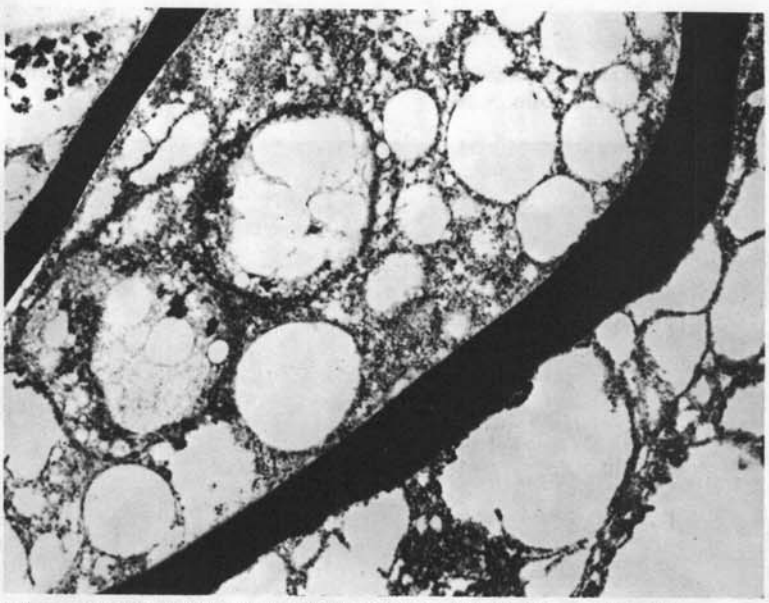

Fig. 5. Necrotic area of femoral head. Many dilated sinusoids, including air bubbles, are seen in it. Case 3, hemotoxylin and eosin, × 50.

DISCUSSION

Beginning in the first half of the nineteenth century, there have been many attempts to advance a theory that would explain the symptoms after decompression. These theories may be grouped by cause as follows: first, exhaustion and cold; second, mechanical congestion; third, today's theory of gas embolism (Bell 1942).

The gas-embolism theory actually dates from the work of Robert Boyle, about 1662, who was the first to observe gas bubbles in the blood of animals subjected to suddenly decreased air pressure. The work of Paul Bert (1878) demonstrated that the symptoms of decompression

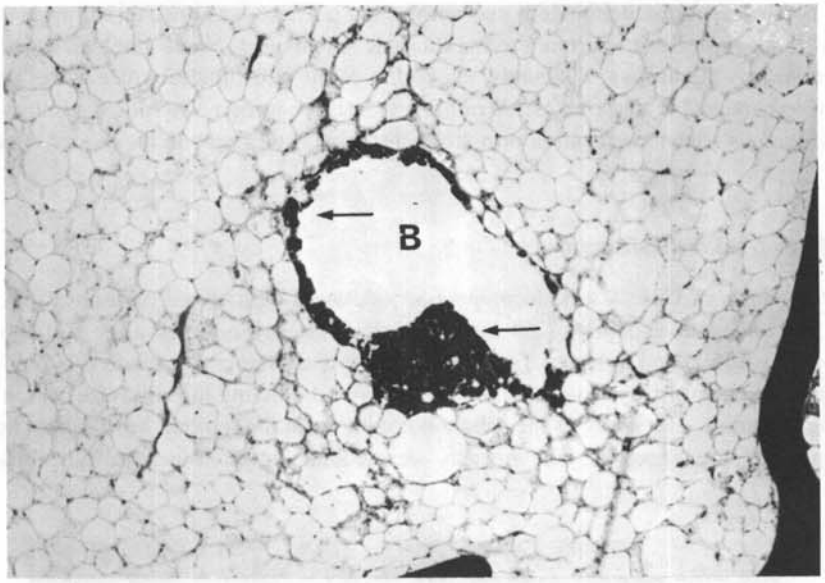

Fig. 6. Dilated sinusoid in femoral head. Air bubble (B) and agglutination thrombus (arrow) are seen in lumen. Case 3, hematoxylin and eosin, × 50.

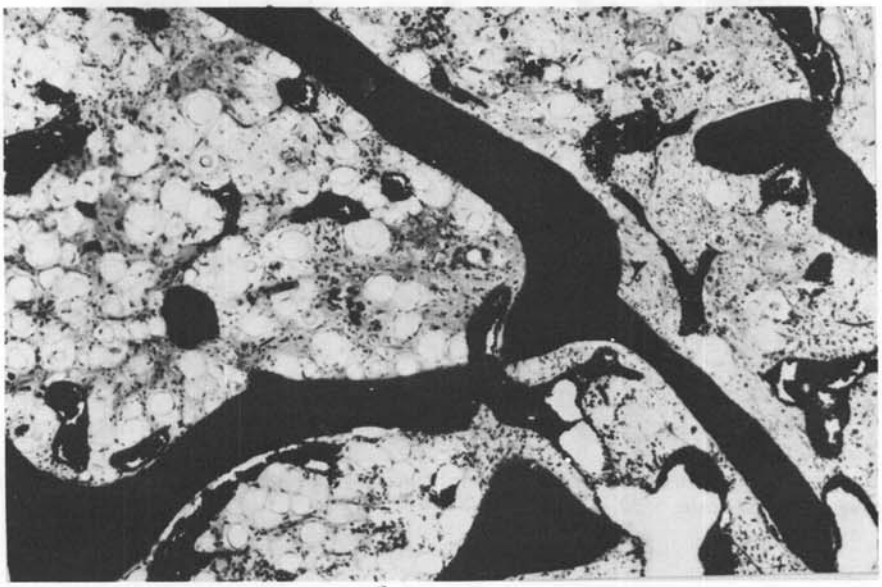

Fig. 7. Fibrotic area of femoral head. Slight hemorrhage and phagocytic migration are seen in it. Case 4, hematoxylin and eosin, × 50.

sickness are produced by air or gas emboli. He was the first to describe decompression sickness clinically and to advance a theory of the etiology of decompression sickness.

It is generally agreed that all symptoms and lesions after decompression are the result of actual bubble formation in the circulating blood or in various tissues.

In addition to the air bubble, other etiological factors have been proposed in the disturbance of the blood circulation after decompression. These factors are hemoconcentration (Behnke

1942; Cockett, Nakamura, and Kado 1965), vasospasm (Chryssanthou, Tiechner, Goldstein, Kalberer, and Antopol 1970), agglutination of erythrocytes (Swindle 1937; End 1938), aggregation of platelets (Philp 1964; Philp, Schacham, and Gowdey 1971; Hallenbeck, Bove, and Elliott 1975), and fat embolism (Cockett, Pauley, Saunders, and Hirose 1971).

In addition, osteonecrosis was found in caisson workers and divers (Bornstein and Plate 1911; Bassoe 1913; Grutzmacher 1941). In our postmortem surveys, inadequate decompression was related to the occurrence of osteonecrosis in divers.

The pathogenesis of osteonecrosis has not been clear. Recently, Smith and Stegall (1972) and Smith, Stegall, and D'Aoust (1975) succeeded in producing osteonecrosis in miniature pigs after repeated decompression. They noticed increased platelet aggregation and suggested that microthrombi may cause osteonecrosis. The histopathological findings of platelet aggregation, erythrocyte sludging, and microthrombus formation accompanied by bubbles may provide a clue to the pathogenesis of osteonecrosis in divers.

In Cases 1 and 2, which had the most acute clinical course, there was platelet aggregation adjacent to the bubbles, slight-to-moderate bleeding with congestion of bone marrow, and sludging erythrocytes in the dilated sinusoids of the femoral head.

In Case 3, where the patient died 5 days after diving, rather extensive necrosis in the bone marrow surrounding expanding sinusoids included air bubbles. Platelet aggregation and agglutination or platelet thrombi occurred in the vicinity of the air bubbles.

In Case 4, air bubbles and thrombi were found in the sinusoids of the femoral head. Around them, phagocytic activation and fibrosis took place and these events seemed to indicate repair of the necrotic bone marrow lesions.

Furthermore, fat was found in the dilated sinusoids in Case 2. We considered that the fat was the result of rupture of fat cells secondary to bubble formation. Fat enters the blood stream coincidentally with other products of tissue disintegration, such as fatty acids, serotonin, and other tissue products. These substances, along with bubbles, tend to alter the secondary and tertiary configuration of blood proteins, leading to activation of the blood-clotting system, platelet aggregation, release of vasoconstrictive substances, and finally, disturbance of blood circulation.

While the sinusoid system of bone marrow is a large venous blood pool in which there are many anastomoses, the volume of the sinusoid system is abundantly greater than that of the arteries supplying the regions it drains. Therefore, the sinusoid system differs from other vein systems. It is not simply a conduit transporting blood toward the heart, but a relatively stagnant pool in which the rate of flow is ordinarily sluggish. As a result, after decompression, the sinusoid system of bone marrow can easily become obstructed by intravascular bubbles that collect, coalesce, and grow—as Hallenbeck said, "by analogy with freezing water, lakes freeze, rivers do not freeze."

In the sinusoid system, bubbles probably exert direct mechanical effects and also indirect effects due to various thrombogenic activities.

In conclusion, the early stage of osteonecrosis of divers might be intimately related to circulatory disturbances, especially in the sinusoid system.

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l'étude anatomopathologique des têtes fémorales de 4 plongeurs morts de maladie de décompression. Chez les cas 1 et 2, dont l'évolution clinique a été plus rapide, nous avons trouve une agrégation plaquettaire à côté des bulles d'air, des hémorrhagies légères, et hyperémie de la moelle osseuse, et une agrégation erythrocytaire dans les sinusoïdes dilatés. Chez le cas 3, dont l'autopsie a eu lieu 5 jours après la plongée, nous avons trouvé une nécrose étendue de la moelle autour des sinusoïdes; des bulles d'air, des agrégations plaquettaires, et des agglutinations ou thrombi plaquettaires y étaient présents aussi. Chez le cas 4, des bulles d'air et des thromboses ont été trouvés dans les sinusoïdes. L'activation phagocytaire et la fibrose qui les entouraient semblent représenter la réparation des lésions nécrotiques de la moelle. Les système des sinusoïdes de la moelle se laisse facilement bloquer par des bulles d'air intravasculaires après la décompression. Les bulles exercent sans doute des effets mécaniques directes en même temps que des effets indirectes thrombogènes. Les premiers étapes de l'ostéonécrose chez le plongeur sont peut-être apparentés aux troubles circulatoires, surtout du system capillaire sinuosïde.

agrégation plaquettaire
maladie de décompression

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