

A CASE OF CEREBRAL AIR EMBOLISM AS A LATE COMPLICATION OF PULMONARY BAROTRAUMA IN A DIVER

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ABSTRACT

The article describes a correctly diagnosed lung pressure injury in a diver - amateur. The case was initially asymptomatic. Then the symptoms of the central nervous system appeared. A patient effectively treated with the use of air recompression tables - healing decompression and then hospitalized at the 7th Naval Hospital in Gdańsk.

Keywords: diving accident, quick exit to surface, lung pressure injury, arterial gas embolism, neurological symptoms.

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INTRODUCTION

The cause of gas (air) embolism in blood vessels may be decompression sickness or pulmonary barotrauma, or they may occur as a complication of medical or self-harming procedures [2,4,5,14]. Regardless of the cause, the treatment of choice is therapeutic recompression in a high-pressure chamber [3,8,14,15].

Pulmonary barotrauma (pneumonia barotrauma, pseudocaesonic disease) is a rare but serious accident in underwater human activity. The cause of this injury is the pressure difference between the gas (air) pressure in the respiratory tract and the external pressure, exceeding 60-80 Hg. This situation occurs as a result of the obstruction of the exit of gas (air) from the lungs [7,9,11-13]. The time spent in overpressure does not play a significant role in the pathomechanism of pulmonary barotrauma (generally one breath is enough), and the depth of immersion can be only 2 m, so it can occur in almost all hyperbaric conditions [1,3,7,8,12].

Air (gas) embolism is caused by bubbles of air or other gas that most often pass from the alveoli of the lungs into the small blood circulation of the left half of the heart and then through the arteries to blood vessels throughout the body.

The location of the air escaping from the lungs causes specific clinical symptoms, but most often (in 50% of cases) it is arterial gas embolism. If it causes clinical symptoms, it is usually located in vital brain centers [8].

In typical cases, symptoms of pulmonary barotrauma appear early, and loss of consciousness occurs suddenly, in 30% of cases while still underwater. In milder cases, in which arterial embolism has not occurred, symptoms develop slowly. Typical symptoms include chest pain, shortness of breath, cough with blood-tinged sputum, and general weakness. The severity of these symptoms can increase over minutes or hours [1], and physical exertion can accelerate their development [11]. Such cases require immediate recompression. In atypical cases and without symptoms of arterial embolism, therapeutic recompression may be significantly delayed due to diagnostic difficulties (in one case, the delay was 30 hours) [6,8,11,13].

The case below is an example of an atypical pulmonary barotrauma with an initially asymptomatic course.

CASE DESCRIPTION

M. J., an 18-year-old participant of a youth scuba diving course, lost his weight belt and quickly surfaced from a depth of 2-3 m, holding his breath. He did not lose consciousness, no symptoms of pulmonary barotrauma were observed, and he did not complain. Since he felt good, he went underwater again and completed the activities planned in the training plan until the end. After a lunch break, together with other course participants, he swam a distance of about 2 km on the surface of the water as part of improving his swimming style in ABC equipment. He felt good the whole time, and did not complain. The next day (after a good night's sleep) during a test dive, he felt a headache. He was released from the classes and ordered to rest. After 2-3 hours, the headache went away, and he did not complain. The next day during a test dive, he had another headache, with which he went to see a doctor. Apart from a headache in the occipital

region, he did not complain. The physical examination did not reveal any deviations from the norm. Pulse regular 80/min, well tensed and filled, RR 120/80 mmHg, temp. 36.7°C. History of head injury negative. He received painkillers and was told to rest. After a few hours the headache decreased but did not disappear completely.

Despite the lack of typical symptoms of pulmonary barotrauma in the trainee, it was decided to perform therapeutic recompression. He was transported to Gdynia. During transport to the chamber, the patient's condition deteriorated rapidly. The following occurred: pain behind the sternum, shortness of breath, initially rapid breathing and tachycardia, then loss of consciousness, convulsions and respiratory and circulatory disorders. These symptoms indicated the displacement of air emboli in the cerebral vessels, probably formed 24 hours earlier, after pulmonary barotrauma (rapid emergence to the surface of the water with a held breath after losing the ballast belt), without clinical symptoms.

Therapeutic recompression was performed in accordance with the applicable regulations according to regime III. The maximum pressure in the chamber was 8 ata. The total time of therapeutic recompression was 31 hours. In the chamber at a pressure of 3-5 ata the patient regained consciousness, all symptoms receded.

After the recompression was completed, the patient was transferred in good general condition to the Neurology Department of the Naval Hospital for observation and further treatment. Upon admission to the Department, he complained only of pain in the heart area. Physical examination did not reveal any deviations from the norm.

Neurological examination: negative meningeal signs, no focal signs, abdominal skin reflexes weaker on the right side, superficial and deep reflexes lively, equal. No pathological signs are found. The patient sits, walks, is calm.

EEG examination: basic activity with a voltage of 20-30 mV, symmetrical. Rz – marked. Hv – does not reveal pathology. Recording within normal limits. Chest radiograph: lungs and heart normally.

ECG examination after admission to hospital: right EKG, alpha angle = +100°, negative T in aVL, a few days later: sinus rhythm, regular 82/min, right EKG, alpha angle = +95°. ECG normal. Ophthalmological examination: VO – 1.0. Fundus of the eyes normally. ENT examination: ears, nose, throat unchanged.

Used: vit. A + E 3 times a day 2 tablets, P mixture 3 times a spoon, vit. C 3 times a day 2 tablets, vit. B complex 3 times a day 2 tablets.

The patient stayed in hospital for 14 days. Discharged from hospital in good general condition with a recommendation for outpatient check-up and EEG examination after 3 months.

EEG examination after 3 months: basic activity with voltage 30 mV, symmetrical, Rz - marked. Hv - no effect, few. Theta waves in 3 minutes. Conclusion: recording within the norm.

DISCUSSION

Based on the interview and clinical picture, air embolism of the central nervous system was diagnosed as a complication of pulmonary barotrauma despite the lack of typical symptoms in the initial period. Despite the late

application of therapeutic recompression (several dozen hours after pulmonary barotrauma) and the deterioration of the patient's general condition before the patient was placed in a pressure chamber, the treatment applied proved effective.

According to literature data, when treatment is significantly delayed, permanent consequences in the nervous system occur most often [1,3,7,8,15]. Therefore, in the case of suspected pulmonary barotrauma (even without clinical symptoms), as indicated in this case by

recurrent headache, therapeutic recompression should be performed early.

According to the current regulations, decompression sickness and the consequences of pulmonary barotrauma are treated by exposing the patient to a pressure of 5-10 atm (therapeutic recompression). Regime III, according to these principles, consists in compressing the patient in a chamber to a pressure of 8 ata (equivalent to 70 meters) [3].

REFERENCES

1. Davies W.: Royal Naval Diving Magazine, 1963, 10, 31.
2. Dolatkowski A.: Lek. Wojsk., 1952, 28, 793.
3. Dolatkowski A., Ulewicz K.: Zarys Fizjopatologii Nurkowania. Warszawa 1973.
4. Erde A.: Proc. 2nd Symp. Underwater Physiol. Washington 1963.
5. Hołyst J.: Niedostateczność Krążenia Krwi w Mózgu. Warszawa 1971.
6. Intelson G. M., Niessirio B.: Woj. Med. Żurn., 1965, 7, 63.
7. Lanpier E. H.: w Handbook of Physiology, section 3: Respiration, vol II, pod red W. O. Fenny i H. Rahn. Washington 1965.
8. Łokucijewski B. i wsp.: Lek. Wojsk., 1971, 6, 577.
9. Miasukow A. P.: Medicinskoje Obiespieczenie Liegkowodolazow i Akwalangistow. Moskwa 1967.
10. Miasnikow W. A., Maszkow G. W.: Woj. Med. Żurn., 1961, 5, 77.
11. Nazarkin W. J.: Woj. Med. Żurn., 1960, 3, 65.
12. Schaefer K. E. i wsp.: J. Appl. Physiol., 1958, 13, 15.
13. Seemann K., Wandel A.: Munch. Med. Wschr. 1967, 109, 1.
14. Teresiński S. i wsp.: Neur. Neurochir. Pol., 1975, 2, 281.
15. US Navy Diving Manual, Washington 1963.