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Scuba diver deaths due to air embolism: two case reports

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SUMMARY
Barotraumas and decompression sickness are the two most well-known complications of diving. First presented case was 32 year-old male with recreational diver, who was found floating prone position on the bottom of sea in a depth of 33 m. He had been carried to the surface in a controlled ascent. Second case was a 39 year-old male experienced dive instructor in a diving school, after following an uneventful duration of dive was found unconscious with a floating supine position in a depth of 30 m and there were no signs of life when they were transported to the hospital. Extensive subcutaneous emphysema of the extremities was detected by palpation of the skin. In the autopsy diffuse gas bubbles like beads were seen in the coronary arteries and in ventricles, basilar artery and all of the cerebral arteries. The cause of death was attributed due to gas embolism and drowning.

Keywords: barotraumas – diving – air embolism – autopsy

Smrt potápěče v důsledku vzduchové embolie: dvě kazuistiky

SOUHRN
Barotrauma a dekompresní nemoc patří mezi dvě nejznámější komplikace potápění. Prezentujeme případ muže ve věku 32 let, rekreačního potápěče, který byl nalezen v poloze na břiše na dně moře v hloubce 33 metrů. Muž byl vyzvednut na břeh s kontrolou času vynořování. Ve druhém případě se jedná o muže stáří 39 let, instruktora potápění, který byl po ponoru nepřesahujícím běžnou délku času nalezen v bezvědomí v poloze na břiše v hloubce 30 m.
Oba muži byli bez známek života transportováni do nemocnice. Palpačně byl patrný výrazný podkožní emfyzém končetin. Při pitvě byla zjištěna přítomnost drobných bublinek plynu v koronárních arteriích, srdečních komorách, bazilární arterii a všech cerebrálních arteriích. Příčinou smrti byla stanovena plynová embolie a utonutí.

Klíčová slova: barotrauma – potápění – vzduchová embolie – pitva

Soud Lek 2013; 58(2): 26–28

Barotraumas and decompression sickness are the two most well-known complications of diving (1). A diver is breathing gas at increased pressure after descending, which often leads to tissue gas super saturation (2,3). When the ambient pressure decreases quickly following uncontrolled ascent to the surface, barotraumas and excessive formation of gas bubbles, which can enter into circulation, occurred in supersaturated tissues (2,3). Another form or unavoidable consequence of the decompression sickness is air embolism which is caused by breach of a vascular wall that allows entering of air to circulation (2). The gas bubbles, presented in the venous system, can enter the systemic arterial circulation by several different mechanisms such as intracardiac right-to-left shunt (usually patent foramen ovale) or pass through the pulmonary capillary due to pulmonary barotraumas or intrapulmonary passage after massive bubble formation and directly into arterial circulation (2,4-9). We presented two cases of diving fatality due to arterial air embolism and discussed with a review of the literature.

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CASE REPORTS

Case 1

A 32 year-old male with unknown signicant medical history was a recreational diver. He was found floating on prone position on the bottom of sea in a depth of 33 m. He had been carried to the surface in a controlled ascent. There was no vital signs when he arrived to the hospital. Despite resuscitation attempts, he did not revive. There was no information about his equipment. Post-mortem external examination showed hemorrhagic foams around the mouth and nostrils. Extensive subcutaneous emphysema of the extremities was detected by skin palpation. Except these findings no significant injuries were observed on external examination. X-ray

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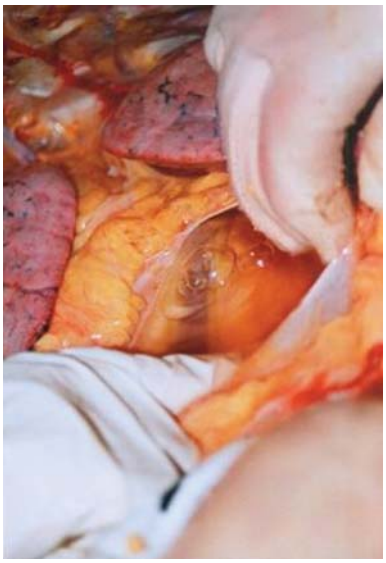


Figure 1. Gas bubbles seen in the heart spaces.

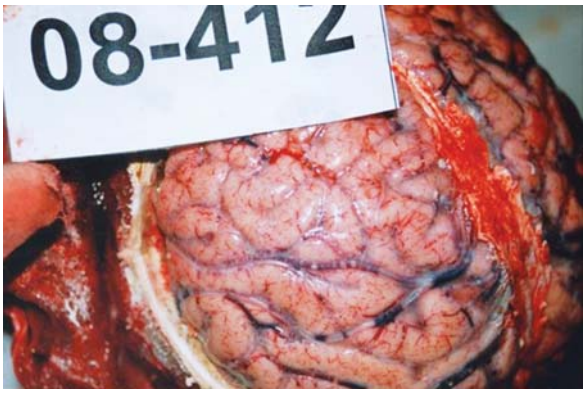


Figure 2. Gas bubbles present in the superficial cerebral blood vessels.

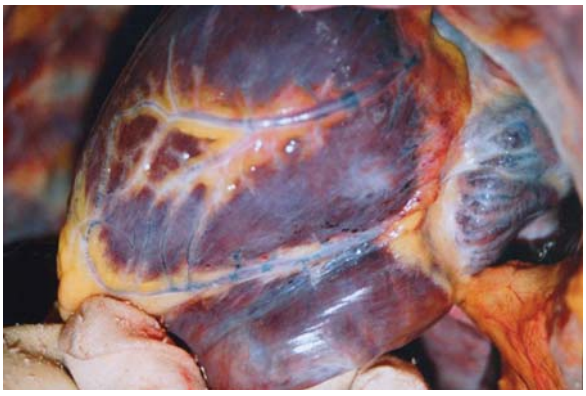


Figure 3. The coronary arteries and their branches filled with gas bubbles.

images, performed before autopsy, supported subcutaneous emphysema and showed extensive gas bubbles in the great vessels and heart spaces. Performed autopsy and diffuse gas bubbles like beads were seen in the coronary arteries and in both ventricles, basilar artery and all of the cerebral arteries and veins (Figure 1,2 and 3). Subarachnoid hemorrhage was observed on the right temporoparietal region. Also the lungs were very extremely heavy (right; 950 g, left; 900 g), markedly edematous and also bloody fluid content was observed in the stomach and duodenum. Histological examination of lungs showed edema, congestion and ruptured alveoli. Toxicological analysis revealed that no toxic agents or alcohol components were detected in the blood or urine specimens. The cause of death was given as gas embolism and drowning.

Case 2

The presented case was a 39 year-old male who was experienced as dive instructor in a diving school. The performed dive was technical, to a depth of 30 m using a self contained underwater breathing apparatus (SCUBA). There was no problem in the initial stage of diving. Following an uneventful duration of dive, he was found unconscious on the depth of the sea with a floating supine position with no signs of life. X-ray images, performed before autopsy, supported subcutaneous emphysema and showed extensive gas bubbles in the great vessels and heart spaces. Post-mortem external examination showed hemorrhagic foams around the mouth and nostrils, subcutaneous emphysema over the chest and extremities was detected. The autopsy revealed that intravascular spaces consistent with gas bubbles were seen in the pulmonary and gastric vascular structures, coronary arteries, in both ventricles and also within all cerebral arteries and veins (Figure 1,2 and 3). White foams were detected around the vocal cords and more intensively on the upper part of the trachea than the other parts of trachea. The surface of the lungs were tensed, voluminous (right; 900 g, left; 700 g), brightly and petechial hemorrhages on the interlobular regions were observed. Water was also observed in the stomach and duodenum. Histological examination of lungs showed edema, congestion and ruptured alveoli. No toxic substances were found in the toxicological analysis of the blood and urine. The pathologic cause of death was given as gas embolism and drowning for each case. Air embolism, associated with decompression sickness or pulmonary barotraumas during diving, can lead to various tissue and vital organ damages and may result in undesirable consequences if it enters cerebral or coronary circulation. After obtained X-ray images, medico legal autopsy was performed to clarify the manner and cause of death. The samples, including parts of organs, blood and urine, were collected after the macroscopic examination for histopathological and toxicological examination. Tissue analysis included hematoxylin-eosin staining. The slides examined with the light microscope. Headspace Gas Chromatography (GC/HS) technique was used for blood alcohol analysis, Spot Test, Thin Layer Chromatography (TLC) and Cloned Enzyme Donor Immunoassay (CEDIA) techniques were used for drug screening in the organ, blood and urine samples.

DISCUSSION

Barotraumas and decompression sickness are the two most well-known complications of diving (1). A diver is breathing gas at increased pressure after descending, which often leads to tissue gas super saturation (2,3). When the ambient pressure decreases quickly following uncontrolled ascent to the surface, barotraumas and excessive formation of gas bubbles, which can enter into circulation, occurred in supersaturated tissues (2,3). Another form or unavoidable consequence of the decompression sickness is air embolism which is caused by breach of a vascular wall that allows entering of air to circulation (2). The gas bubbles, presented in the venous system, can enter the systemic arterial circulation by several different mechanisms such as intracardiac right-to-left shunt (usually patent foramen ovale) or pass through the pulmonary capillary due to pulmonary barotraumas or intrapulmonary passage after massive bubble formation and directly into arterial circulation (2,4-9). Air embolus, another form of decompression illness and

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arterial gas embolization is the second most common seen cause of death among divers, with sequels dependent on the final destination of the emboli with mortality rate from 7 to 14% (1,9,10). In scuba diving, these gas bubbles most commonly occur in uncontrolled ascents with decreasing partial ambient pressure which results in gas coming out of supersaturated tissues into the intra-vascular space. Air embolism, associated with decompression sickness or pulmonary barotraumas during diving, can lead to various tissue and vital organ damages and may result in undesirable consequences after invasion of cerebral, coronary, renal or gastrointestinal circulation (2). The clinical presentation of arterial embolism can be varying from acute renal insufficiency to sudden death depending on the occlusion of the end arteries, associated organs or tissues. The gas bubbles can enter the systemic arterial circulation via right to left shunt, pulmonary capillary due to pulmonary barotraumas or intrapulmonary passage (2,4-9). Although there was no evidence of right to left shunt, severe subcutaneous emphysema was detected in both of our cases. According to this finding we thought that the arterial air embolism was occurred due to pulmonary barotraumas and this consideration was supported by the histopathological investigation. Approximately, in the 60% of the victims, sustained fatal diving accidents, the cause of death was reported as drowning (12,13). Although, drowning was not attributed as a single cause of death of the presented cases, it had been contributed to death with arterial air embolism. Both of the victims were experienced divers and it was not known how the accident was occurred. There was no technical information about the diving equipment. Finally, the divers must take various precautions and precise equipment check before diving, with strictly following of all the procedures learned in diving instruction. Diving and decompression sickness can cause life threatening consequences if the warnings were not taken into consideration.

Report was presented in 22nd IALM Congress, Istanbul 5-8 July 2012.

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ZPRÁVY Z KONFERENCE

IV. mezinárodní kongres úrazové chirurgie a soudního lékařství



Ústav soudního lékařství LF MU a Fakultní nemocnice u sv. Anny v Brně, Klinika úrazové chirurgie LF MU a Traumacentrum Fakultní nemocnice Brno ve spolupráci s Aesculap Akademií uspořádali ve dnech 26. – 27. září 2012 v Mikulově, v konferenčních prostorách hotelu Galant, IV. mezinárodní kongres úrazové chirurgie a soudního lékařství. Hlavním tématem bylo *„Polytrauma a komplikace v chirurgické péči"*. Kongres byl pořádán pod záštitou předsedkyně Nejvyššího soudu JUDr. Ivy Brožové, děkana Lékařské fakulty Masarykovy univerzity prof. MUDr. Jiřího Mayera, CSc., ředitele Fakultní nemocnice Brno MUDr. Romana Krause, MBA a ředitele Fakultní nemocnice u sv. Anny v Brně Ing. Petra Košky, MBA. Garanci převzala Česká společnost pro úrazovou chirurgii ČLS JEP. Akce byla určena pro lékaře a sestry chirurgických oborů, urgentních příjmů, zdravotnických záchranných služeb, soudní lékaře a laborantů. Cílem kongresu bylo uspořádat vědeckou mezioborovou akci, která přinese účastníkům větší přehled a orientaci v dané problematice. Hlavními tématy byly přednemocniční péče, urgentní příjmy, komplikace v chirurgii a forenzní problémy.

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Hlavní pořadatelé si vás zároveň dovolují **pozvat** na podobný kongres, v pořadí již V., který se koná ve dnech **12. – 13. září 2013** v Mikulově v konferenčních prostorách hotelu Galant. Doufáme, že tentokrát bude spektrum účastníků rozšířeno i o hojnou přítomnost policistů a státních zástupců z celé republiky.

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MUDr. Eva Daňková