ALVEOLAR HEMORRHAGE AFTER SCUBA DIVING: A CASE REPORT

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Self-contained underwater breathing apparatus (scuba) diving is increasingly popular in Taiwan. There are few references in the literature regarding pulmonary hemorrhage as the sole manifestation of pulmonary barotrauma in scuba divers, and no study from Taiwan was found in the literature. We present the case of a 25-year-old man who suffered alveolar hemorrhage related to pulmonary barotrauma as a complication of scuba diving. To our knowledge, this is the first case report describing a Taiwanese subject suffering from non-fatal pulmonary hemorrhage after scuba diving.

Key Words: alveolar hemorrhage, computed tomography, hemoptysis, scuba diving (*Kaohsiung J Med Sci* 2010;26:389–92)

Self-contained underwater breathing apparatus (scuba) diving is increasingly popular in Taiwan. There is, however, little information in the literature regarding pulmonary hemorrhage as the sole manifestation of pulmonary barotrauma in scuba divers, and no study from Taiwan was found in the literature. We present this case to alert physicians to the risk of alveolar hemorrhage as the main complication of scuba diving.

CASE PRESENTATION

A previously healthy, obese 25-year-old man (body weight 98 kg, height 174 cm; body mass index 32.4 kg/m²) was transferred to our hospital with hemoptysis. He had been well before a scuba diving accident at



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a depth of about 11 meters in a subtropical area, Peng-hu. When he tried to move a rock to collect shell-fish under the water, the rock slipped from his hand and hit him on the anterior chest wall. Being startled, he ascended to the surface quickly. Immediately after reaching the surface, he repeatedly expectorated some fresh blood over a few minutes and also noted otor-rhea with otalgia and hearing loss of his left ear. He denied aspirating sea water or blood, but he had some breathlessness. His father reported that he expectorated about 100 mL of fresh blood in total. After 20 mg furosemide and 30 mg ketorolac were given intravenously, he was transferred to our hospital as suggested by the doctor in the clinic of the local health center.

He presented to the emergency department of our hospital with a pulse rate of 109 beats/min and respiratory rate of 14 breaths/min. He was afebrile and denied having chills. His consciousness was clear, without any neurological deficit. Mild local tenderness on the midline of his anterior chest wall was noted, while no ecchymosis or wound was found. His breath sounds were mildly decreased bilaterally, especially in the bilateral lower lung fields, but no crackle was

heard. Gastric irrigation found no evidence of upper gastrointestinal bleeding. Laboratory tests revealed leukocytosis with $20,630/\mu L$ and elevated serum aspartate aminotransferase ($225\,U/L$; normal, $10-35\,U/L$), serum alanine aminotransferase ($252\,U/L$; normal, $10-40\,U/L$), and creatine phosphokinase ($274\,U/L$; normal, $26-174\,U/L$). A chest radiograph (Figure 1) revealed a patchy lesion with ground-glass appearance in the retrocardiac region, highly consistent with bronchopneumonia in the left lower lung. Thoracic computed tomography (CT) obtained a few hours after the event (Figure 2) revealed a ground-glass appearance in the left lower lobe, several small patches in



Figure 1. Chest radiograph revealed a patchy lesion with groundglass appearance in the retrocardiac region.

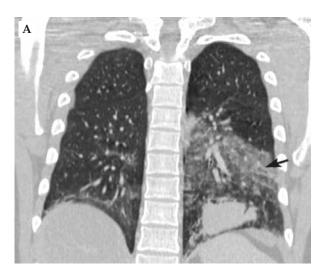
the right upper and lower lobes, and a small amount of left pneumothorax. Because of the nonspecific findings, he was then admitted to the isolation ward for further evaluation to exclude pulmonary tuberculosis.

He was initially treated with amoxicillin plus clavulanate for 7 days. Perforation of his left tympanic membrane with otorrhea was noted, and topical ofloxacin solution was applied.

Bronchoscopy on the 4th hospital day revealed thick mucus secretion with blood in the airway and diffuse ecchymosis in the left lower segmental bronchi. Iron staining of the bronchoalveolar lavage revealed hemosiderin deposition in macrophages, which was compatible with pulmonary hemorrhage. After three sets of sputum acid-fast stain and a bronchial wash failed to find acid-fast bacilli, he was transferred to the ordinary ward for further care. Follow-up chest CT obtained 10 days later (Figure 3) showed partial resolution of the pulmonary hemorrhage. As he was in a stabilized clinical condition, he was discharged on the 11th hospital day. Follow-up in the chest clinic and later in the otorhinolaryngologic clinic was uneventful.

DISCUSSION

The initial differential diagnosis for our patient included pulmonary hemorrhage, bronchopneumonia, aspiration, and pulmonary edema. The large amount of fresh blood expectorated made pulmonary hemorrhage the most possible diagnosis, and this was eventually



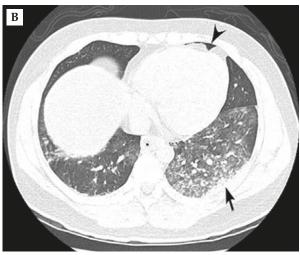


Figure 2. Chest computed tomography showed a ground-glass appearance in the left lower lobe (arrow), and several small patches in the right upper and lower lobes. A small amount of left pneumothorax (arrowhead) was also noted.

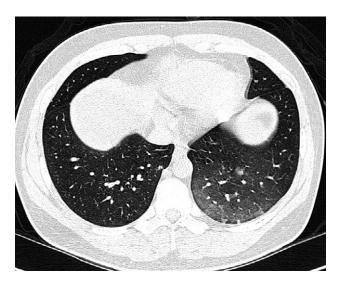


Figure 3. Follow-up chest computed tomography obtained 10 days later showed partial resolution of the ground-glass appearance.

confirmed by bronchoscopy and cytological examination of the bronchoalveolar lavage. Because of the presence of ear barotrauma and pneumothorax, and the absence of an obvious lesion in the thoracic cage, chest barotrauma was the most probable cause of pulmonary hemorrhage according to the patient's history.

During breath-hold diving, air in the lungs is compressed by increasing the ambient pressure according to Boyle's law. After descending below a "squeeze" depth, at which alveoli are compressed to their smallest noncollapsible volume, the blood pressure in the pulmonary capillaries exceeds the intra-alveolar pressure [1]. The increased transpulmonary capillary wall pressure leads to "stress failure" of pulmonary capillaries, resulting in leakage of fluid and blood into the lungs, i.e. pulmonary edema and even hemorrhage [1–3]. During a rapid ascent in scuba diving, increased gas volume in a confined space can produce tissue disruption and rupture, which may lead to pulmonary barotrauma, manifesting as pneumothorax, pneumomediastinum, subcutaneous emphysema, arterial gas embolism, etc. [4]. Pulmonary hemorrhage is not a very common single manifestation of pulmonary barotrauma in scuba divers. Because our patient had dived to 11 meters of depth and surfaced rapidly, barotrauma arising from the rapid ascent was the most probable mechanism inducing alveolar hemorrhage.

Numerous causes of hemoptysis originating from the lower respiratory tract have been reported, including tuberculosis, bronchiectasis, lung abscess, lung cancer, and pulmonary embolism. In an endemic area, tuberculosis should be ruled out first. A complete history with adequate examination is the keystone for making the correct diagnosis. In our patient, no other associated medical problem was found as the cause of pulmonary hemorrhage.

Pulmonary hemorrhage is not a common manifestation of pulmonary barotrauma after scuba diving. To our knowledge, this was the first case report describing a Taiwanese subject with non-fatal pulmonary hemorrhage after scuba diving. In addition, although thoracic CT of pulmonary hemorrhage of a breathhold diver had been published before [1,3], there has been little reported on thoracic CT of pulmonary hemorrhage in a scuba diver in the literature to date. Alveolar hemorrhage is also an important complication of diving accidents associated with barotrauma [4,5]. We report this case with alveolar hemorrhage as the main clinical manifestation of barotrauma arising from scuba diving. Physicians, regardless of practice location, may encounter patients with pulmonary barotrauma and should be familiar with its clinical manifestations.

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使用水肺潛水後的肺胞出血:一病例報告

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水肺潛水在台灣地區越來越普遍。肺部壓力性傷害以肺出血為主要表現是很少被報告 於文獻上的,在文獻上尤其找不到台灣的病例。我們在此報告一個 25 歲男性因水肺 潛水所致的壓力性傷害而產生肺胞出血。據我們所知,這是文獻上第一個台灣人因水 肺潛水導致非致死性肺胞出血的案例。

> **關鍵詞:**肺胞出血,電腦斷層,咳血,水肺潛水 (高雄醫誌 2010;26:389-92)

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