common, splenosis has been reported in the retroperitoneum and liver. In rare cases, presence of splenic tissue within the pulmonary parenchyma has been reported when injury to the lung had occurred along with injury to the spleen. Rarely, splenic tissue may gain access into the thorax either from a diaphragmatic tear or through diaphragmatic foramina, and seed the pleural space. Fewer than 30 cases have been reported in the literature; in most instances, the diagnosis was made only after surgery. However, intrathoracic splenosis is usually asymptomatic. Moreover, radiographic and CT findings associated with this disorder are nonspecific, and several alternative diagnoses exist. These include lymphoma, mesothelioma, invasive thymoma, and metastatic disease, as well as localized fibrous tumor of the pleura or pleural plaques. The concern that these findings might represent malignancy may subject the patient to unneeded follow-up or diagnostic procedures. In addition to the risks associated with surgery or biopsy, removal of thoracic splenic tissue in a patient without functioning abdominal splenic tissue may render the patient asplenic, and increase the risk for infection.

Scintigraphy with indium In 111-labeled platelets, 99mTc SC, and 99mTc heat-damaged RBCs all are capable of demonstrating splenosis because they are sequestered by splenic tissue. However, use of 99mTc heat-damaged RBCs is preferred because of reduced uptake by normal liver tissue, resulting in improved target-to-background ratio. A case has been reported in which a 99mTc heat-damaged RBC scan depicted additional foci of splenosis that were not demonstrated on a 99mTc SC scan. Our case is unique in that intrathoracic splenosis was seen on the 99mTc heat-damaged RBC scan following false-negative 99mTc SC scan results.

Scintigraphy with 99mTc heat-damaged RBCs is not only more sensitive than 99mTc SC, but is also superior to the peripheral blood smear. Howell-Jolly bodies represent nuclear material within the RBC, and are normally retained by the spleen. Their presence on a peripheral blood smear indicates functional asplenism. However, other patients with uptake in residual splenic tissue have also been shown to have Howell-Jolly bodies, presumably due to the inability of this tissue to function normally.

Although rare, the diagnosis of intrathoracic splenosis should be considered in any patient with pleural-based nodules in the left hemithorax with a history of thoracoabdominal trauma and splenectomy or splenic injury. Noninvasive confirmation of the diagnosis using 99mTc heat-damaged RBC scintigraphy can spare patients additional follow-up imaging and/or invasive procedures. This case demonstrates that reliance on either a negative 99mTc SC scan finding or the presence of Howell-Jolly bodies on a peripheral blood smear can result in a missed diagnosis. 99mTc heat-damaged RBC imaging should be considered the modality of choice to diagnose splenosis.

REFERENCES

Hemoptysis Provoked by Voluntary Diaphragmatic Contractions in Breath-Hold Divers*

Esen Kıyan, MD; Samil Aktas, MD; and Akin Savas Toklu, MD

Pulmonary barotrauma of descent (lung squeeze) has been described in breath-hold divers when the lung volume becomes smaller than the residual volume (RV), with the effect of increased ambient pressure. However, the ratio between the total lung capacity and the RV is not the only factor that plays a role in the lung squeeze. Blood shift into the thorax is another important factor. We report three cases of hemoptysis in breath-hold divers who dove for spear fishing in shallower depths than usual. All of the divers performed voluntary diaphragmatic contractions at the beginning of their ascent, while their mouths and noses were closed. We suggest that the negative intrathoracic pressure due to the forced attempt to breathe in with voluntary diaphragmatic contractions contributes to alveolar hemorrhage, since it may damage the pulmonary capillaries.

(CHEST 2001; 120:2098–2100)

Key words: alveolar hemorrhage; breath-hold diving; hemoptysis

Abbreviations: RV = residual volume; TLC = total lung capacity

Pulmonary barotrauma of descent (lung squeeze) occurs in breath-hold divers when the lung volume becomes smaller than the residual volume (RV). As the diver descends deeper, the gas in the lung will be compressed and the lung volume will be diminished, with the effect of the increased ambient pressure, according to Boyle’s law.

*From the Department of Chest Medicine (Dr. Kıyan), and Department of Undersea and Hyperbaric Medicine (Drs. Aktas and Toklu), Istanbul University, Istanbul, Turkey.

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Correspondence to: Samil Aktas, MD, Istanbul Üniversitesi, Istanbul Tip Fakültesi, Deniz ve Sualtı Hekimliği AD, 34390, Capa, İstanbul-Türkiye; e-mail: aktasmd@hotmail.com
During descent, total lung capacity (TLC) decreases and reaches the RV at a certain depth, and further descent may cause alveolar hemorrhage and pulmonary edema.\(^1\)\(^2\) The depth at which TLC is reduced to RV is the theoretical maximal depth for a breath-hold diver. The rise in water pressure with depth is equivalent to 0.1 atm/m. At 10 m of seawater, 1 atm of air and 1 atm of water will be pressing on the breath-hold diver, reducing the lung volume to one half of TLC. Since RV averages one fourth of TLC, a pressure of 4 atm at 30 m of seawater will reduce the lung volume to RV.

It is known that many breath-hold divers exceed their depth limits calculated on the basis of the TLC/RV ratio. Additional factors must therefore be considered. At greater depths, the high negative transthoracic pressure that develops as the diver passes through 30 m and the chest wall approaches its elastic limit draws about 1 L of blood into the thorax.\(^1\) As a result, pulmonary capillaries bulge prominently into the alveolar spaces, replacing air and resulting in a decrease of RV and thereby extending the depth limit.\(^1\)\(^2\) However, blood shift into the thorax may predispose to alveolar hemorrhage by causing an increase in the pulmonary capillary pressure. In this study, we report three cases of hemoptysis in breath-hold divers following voluntary diaphragmatic contractions.

**CASE REPORTS**

We observed three divers who had experienced hemoptysis while breath-hold diving. All three divers were nonsmoking healthy men aged 22 to 37 years and had no significant medical histories. They were also scuba divers, and they had no histories of pulmonary barotrauma of ascent or decompression sickness. They were free of diseases predisposing to pulmonary hemorrhage. None of the divers were receiving medications, including acetylsalicylic acid (aspirin), and none of them reported aspira-

**Case 1**

A 37-year-old, healthy, well-trained, male breath-hold diver performed several dives to the depth of 10 to 12 m. He then dove to 17 to 18 m and descended to 20 to 22 m for spear fishing. After 30 s at this depth, he developed shortness of breath while he was making voluntary diaphragmatic contractions. After returning to the surface, he had a sudden onset of coughing and expectorated bloody froth. He had no chest pain. Within an hour, he was comfortable and his symptoms resolved spontaneously. One week after this event, he was admitted to our clinic. Results of physical examination, lung function testing, oxygen saturation, and chest radiography were normal, but the thorax CT obtained 2 h after the diving event revealed images suggestive of alveolar hemorrhage (Fig 1). A diagnosis of alveolar hemorrhage was made based on his history and CT findings. Because of late hospital admission, we could not perform BAL to document alveolar hemorrhage. The patient had a history of five episodes of hemoptysis during breath-hold diving. When we obtained the detailed history of these episodes, we learned that he had performed voluntary diaphragmatic contraction during all episodes. Control CT findings were normal 3 weeks after the event. The follow-up examination of the diver revealed no evidence of lung, heart, or hematologic disease.

**Case 2**

The patient was a 22-year-old, healthy man with 10 years of breath-hold diving and 3 years of scuba-diving experience. His depth limit was >30 m. First, he made a dive to 5 m and ascended to the surface. Then he dove to 20 m, where he remained for 30 s. During a normal ascent, he experienced voluntary diaphragmatic contractions at 17 m. After surfacing, he coughed and expectorated bloody froth. He had no dyspnea and chest pain. His symptoms disappeared after 5 min, and he did not seek medical attention. Results of a medical evaluation performed 1 week after the event were normal, as were physical examination, chest radiography, lung function testing, and oxygen saturation findings.

**Case 3**

The patient was a 31-year-old, well-trained man with 9 years of breath-hold diving and 11 years of scuba-diving experience. His depth limit was 40 to 45 m. First, he performed 20 dives to a depth of 15 m. For each dive, he spent 2 min at this depth. He had no diaphragmatic contractions during these dives. Later, he made another dive to 15 m, where he remained for 2.5 min; however, this time he had diaphragmatic contractions just before the ascent (because of short surface interval). After surfacing, he started to cough and expectorated bloody froth. He had dyspnea but no chest pain. He recovered after 10 min, and he did not seek medical attention. On admission to our clinic 2 weeks after the diving event, physical examination, chest radiography, lung function testing, and oxygen saturation findings were normal. He had a history of hemoptysis during breath-hold diving after voluntary diaphragmatic contractions 3 years previously.

**DISCUSSION**

The pulmonary blood-gas barrier needs to be extremely thin for efficient gas exchange. However, it also needs to

<table>
<thead>
<tr>
<th>Variables</th>
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<th>Case 2</th>
<th>Case 3</th>
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<tr>
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<td>Symptoms disappeared after 1 h</td>
<td>Symptoms disappeared after 5 min</td>
<td>Symptoms disappeared after 10 min</td>
</tr>
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</table>
Figure 1. Bilateral alveolar infiltration suggestive of intra-alveolar hemorrhage.

Three factors can affect the pulmonary blood-gas barrier and cause alveolar hemorrhage or edema. The first is increased blood volume and BP in the pulmonary capillaries (eg, immersion, exercise, exposure to cold water, high altitude, and overhydration). The second is decreased elasticity and resistance of the respiratory membrane (eg, Goodpasture’s syndrome, or the overdistention of the lung by mechanical ventilation). The third is negative pressure in the alveoli (eg, upper-airway obstruction by tumor, foreign body, spasm, etc). During diving, immersion causes central blood pooling, thus increasing cardiac preload. Exercise causes an increase in cardiac output. Cold exposure increases both preload and afterload by vasoconstriction.

A combination of these mechanisms, as occurs during diving, together with an increase in intrathoracic blood volume could be responsible for an excessive increase in pulmonary capillary pressure. As a result, increased capillary pressure can disrupt the blood-gas barrier and cause alveolar edema or hemorrhage.

Boussges et al published a report of three cases of nonfatal alveolar hemorrhage in breath-hold divers. They speculated that factors such as immersion, exercise, and exposure to cold, and an increase in ambient pressure could account for the hemoptysis. In that study, all of the divers had ingested acetylsalicylic acid (aspirin) a few hours before diving. The author reported that acetylsalicylic acid usage might have aggravated the bleeding through its antiplatelet effect, but it is known that this drug does not cause spontaneous bleeding from intact vessels.

Most of the factors that can cause alveolar hemorrhage were not obvious in our patients, who experienced hemoptysis when they dove to a shallower depth than their limit. They had no known pulmonary or cardiac diseases, and they were not receiving any medications, including acetylsalicylic acid. They dove in Mediterranean waters during the spring, when the water temperature is not very low. They were not overhydrated and had no water aspiration. However, all of the divers did the same maneuver: voluntary diaphragmatic contraction at the depth. This maneuver is used by breath-hold divers to increase the depth-hold time. Diaphragmatic contractions cause markedly negative intrapleural pressure generated by a forceful inspiratory effort against an obstructed extrathoracic airway. Markedly negative intrapulmonary pressure increases venous return, pulmonary blood volume, and pulmonary capillary hydrostatic pressure while lowering the perivascular interstitial hydrostatic pressure.

We suggest that voluntary diaphragmatic contraction could be the main contributing factor for the alveolar hemorrhage in our divers, in addition to relatively low water temperature, exercise, and immersion, which increase the pulmonary capillary pressure. Although hemoptysis in breath-hold divers has been reported before, this maneuver has not been cited previously as a contributing factor for hemoptysis in breath-hold divers.

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