Respiratory Medicine Case Reports 11 (2014) 16–17

Contents lists available at ScienceDirect

Respiratory Medicine Case Reports

journal homepage: www.elsevier.com/locate/rmcr

Case report

Lung physiology at play: Hemoptysis due to underwater hockey

Meghan Aversa [a], Stephen E. Lapinsky [b,*]

[a] Department of Medicine, University of Toronto, Canada
[b] Mount Sinai Hospital and Department of Medicine, University of Toronto, Canada

ARTICLE INFO

Article history:
Received 23 August 2013
Accepted 5 December 2013

Keywords:
Hemoptysis
Diving
Exercise

ABSTRACT

Hemoptysis can be a very concerning symptom, and the workup of a patient with hemoptysis may be expensive and invasive. Over the past decade, there has been increasing recognition of hemoptysis that occurs in highly trained athletes under conditions of extreme physical exertion and is explained by "pulmonary capillary stress failure". This report highlights the physiological mechanisms of pulmonary capillary stress failure in the highly trained athlete, with emphasis on the predisposition to develop this condition in underwater sports. We describe the case of an otherwise healthy 34 year-old competitive underwater hockey player who reported hemoptysis following particularly strenuous games. We postulate that the hemoptysis was a result of the pulmonary capillary stress failure caused by the cumulative hemodynamic effects of a markedly elevated cardiac output, the increased central blood volume caused by the hydrostatic effects of submersion in water, and the negative intrathoracic pressure produced by voluntary diaphragmatic contractions.

1. Introduction

We describe the case of an otherwise healthy male who presented to hospital with hemoptysis following a game of underwater hockey. We postulate that the hemoptysis was a result of pulmonary capillary stress failure caused by the combined hemodynamic effects of exertion, submersion, and diaphragmatic contractions.

2. Case report

A 34 year-old male presented to the Emergency Department with hemoptysis following a strenuous game of underwater hockey. Underwater hockey is played in a swimming pool at a depth of 2–4 m. Wearing a snorkel mask and fins, players pass a weighted puck from the bottom of the pool, during repeated apneic dives. The patient reported a cough productive of approximately four teaspoons of bright red blood. Similar episodes of hemoptysis had occurred twice previously, each time following a game of underwater hockey. He denied ever experiencing shortness of breath or chest pain with exercise. Review of systems was negative. There was no history of respiratory disease, and he was a non-smoker.

On presentation to hospital, vital signs were normal and physical examination was unremarkable.

Blood work was normal and included: hemoglobin 155 g/L, platelet count 182 $\times$ 10^9/L, INR 1.0, PTT 26.1 s, anti-nuclear antibody 1:40 (homogenous), rheumatoid factor <8 (negative), negative anti-neutrophil cytoplasmic antibody (ANCA) and anti-glomerular basement membrane antibody levels. Chest x-ray was normal. CT angiogram of the chest revealed normal lung parenchyma, no vascular abnormalities, and no evidence of pulmonary emboli. Trans-thoracic echocardiogram showed borderline concentric left ventricular hypertrophy but normal left ventricular diastolic function. The right ventricular systolic pressure (RVSP) was at the upper limit of normal at 35 mmHg. Stress echocardiogram was normal after 14 min of exercise, with an increase in RVSP from 40 to 50 mmHg. Bronchoscopy revealed slightly erythematous mucosa but no frank bleeding or endobronchial lesions. Bronchoalveolar lavage demonstrated no abnormalities.

The patient was discharged from our clinic with no clear diagnosis. Interestingly, he was referred back seventeen years later for an incidental finding of a pulmonary nodule. He reported that he had ultimately stopped playing underwater hockey and experienced no further episodes of hemoptysis. In retrospect, recent literature, reviewed below, quite clearly characterizes the cause of his previous hemoptysis.

* Corresponding author. Division of Critical Care, Mount Sinai Hospital, 600 University Avenue, Room 18-214, Toronto, Ontario M5G 1X5, Canada. Tel.: +1 416 586 4800x3211; fax: +1 416 586 8480.
E-mail addresses: meghan.aversa@mail.mcgill.ca (M. Aversa), stephen.lapinsky@utoronto.ca (S.E. Lapinsky).

2213-0071
http://dx.doi.org/10.1016/j.rmcr.2013.12.002

3. Discussion

Exercise-induced hemoptysis in otherwise healthy individuals has been described in strenuous swimming [1], SCUBA (self-contained underwater breathing apparatus) diving [2], and breath-hold diving [3,4], although it develops through somewhat different mechanisms in each sport. To our knowledge, we describe the first case of hemoptysis following underwater hockey. We postulate that the hemoptysis in our case was caused by a combination of the hemodynamic effects of strenuous exertion, submersion, and diaphragmatic contractions on the pulmonary capillaries. Underwater hockey is somewhat unique in that all of these mechanisms occur simultaneously.

In 2004, West described our reliance on the blood-gas barrier to be simultaneously both thin enough to facilitate gas exchange and strong enough to withstand the stress of exercise as a "basic dilemma" [5]. The blood-gas barrier (comprised of the capillary endothelium, an extracellular matrix, and the alveolar epithelium) is estimated to be as thin as 0.2 to 0.3 um, its strongest component being the type 4 collagen of its basement membranes. During exercise, pulmonary artery pressures rise and pulmonary transcapillary pressures can reach 40 mmHg. At a certain point, the pulmonary transcapillary pressures overwhelm the thin blood-gas barrier, the cells are disrupted, and the integrity of the barrier is temporarily compromised. With electron microscopy, West actually captured the disrupted pulmonary capillary endothelial cells of cannulated rabbits at higher transcapillary pressures [6], in what he referred to as "pulmonary capillary stress failure."

Evidence of pulmonary capillary stress failure is the leakage of proteinatous fluid (edema) and red blood cells (RBCs) into the intraalveolar space, which has been well documented after strenuous swimming [1]. Hopkins et al. described hemoptysis in elite athletes following strenuous cycling and documented higher concentrations of red blood cells and protein in their bronchoalveolar lavage samples as compared to a control population [7]. Larger studies of pulmonary capillary stress failure causing alveolar hemorrhage in humans after strenuous exertion on land are lacking, but the phenomenon is very well described in exercising thoroughbred racehorses [8]. In this case, our patient's exertion during this strenuous game of underwater hockey likely elevated his pulmonary pressures and contributed to the pulmonary capillary stress failure that produced post-game hemoptysis.

Significant cardiopulmonary effects related to increased ambient pressure have been described in breath-hold diving. Boussuges et al. described three cases of healthy males who presented to hospital with productive cough, hemoptysis, and evidence of alveolar hemorrhage on bronchoscopy shortly after a 25–35 m dive. [3] At these depths, divers experience "thoracic squeeze" where increasing pressure reduces lung volume potentially below residual volume, causing a shift of blood into the thoracic cavity [9]. At a depth of approximately 30 m, close to 1 L of blood is shifted into the thoracic cavity of a diver [9]. At a depth of 2–4 m, an underwater hockey pool is likely too shallow for these effects to apply. However, Aborelius et al. demonstrated in right-heart catheterized subjects that immersion, even to the level of the neck, causes enough hydrostatic pressure to shift peripheral blood into the thorax [10]. In a similar experiment, Begin et al. demonstrated a 20–40% increase in the calculated pulmonary capillary pressure within 30 min of immersion to the level of the neck [11]. It is likely that our patient also experienced a degree of pulmonary capillary stress failure from the hydrostatic pressure at the depth at which he played.

Diaphragmatic contraction, the practice of generating an inspiratory effort against a closed glottis, is routinely employed by underwater athletes to prolong their apneic period. By generating negative intrapleural pressure, this maneuver increases venous return and has been cited as a likely cause of hemoptysis in breath-hold diving [12]. Our patient generated diaphragmatic contractions routinely during games and we postulate that it also contributed to his hemoptysis.

Considering that our patient had borderline concentric left ventricular hypertrophy and an RVSP at the upper limit of normal, it is possible that he may have had mild cardiac or pulmonary vascular disease that increased his susceptibility to developing pulmonary capillary stress failure. Some of these findings may occur in highly trained athletes [13]. It is unlikely that this patient had any other significant cardiorespiratory condition, given his stable status over the seventeen years following his initial presentation.

This specific case demonstrates that pulmonary capillary stress failure in underwater sports is multifactorial. Underwater hockey requires simultaneous exertion, submersion, and often, diaphragmatic contraction, all of which increase pulmonary transcapillary pressure through different mechanisms. We postulate that these mechanisms all contributed to the hemodynamic changes that lead to pulmonary capillary stress failure and our patient's hemoptysis.

4. Conclusion

When evaluating hemoptysis in underwater athletes, the clinician must consider how much exertion, depth of submersion, and diaphragmatic contraction may be contributing to the patient's symptoms. To avoid more significant episodes of hemoptysis, it may be prudent to advise the patient to discontinue playing the underwater sport.

References

[1] Adir Y, Shupack A, Gil A, Peled N, Keynan Y, Domachevsky L, et al. Swimming-induced pulmonary edema: clinical presentation and serial lung function. Chest 2004;126:394–9.
[2] Pons M, Blickenstorfer D, Oechslin E, Hold G, Greminger P, Franzeck UK, et al. Pulmonary oedema in healthy persons during scuba-diving and swimming. Eur Respir J 1995;8:762–7.
[3] Boussuges A, Pinet C, Thomas P, Bergmann E, Sainty JM, Vervloet D, et al. Haemoptysis after breath-hold diving. Eur Respir J 1999;13:697–9.
[4] Koehle M, Lepawsky M, McKenzie D. Pulmonary oedema of immersion. Sports Med 2005;35:183–90.
[5] West B. Vulnerability of pulmonary capillaries during exercise. Br J Sports Med 2006;40:821.
[6] West JB, Tsukimoto K, Mathieu-Costello O, Prediletto R. Stress failure in pulmonary capillaries. J Appl Physiol 1991;70:1731–42.
[7] Hopkins SR, Schoene RB, Henderson WR, Spragg RG, Martin TR, West JB. Intense exercise impairs the integrity of the blood-gas barrier in elite athletes. Am J Respir Crit Care Med 1997;155:1090–4.
[8] Hinchcliff KW, Jackson MA, Brown JA, Dredge AF, O'Callaghan PA, McCaffrey JP, et al. Trachobronchoscopic assessment of exercise-induced pulmonary hemorrhage in horses. Am J Vet Res 2005;66:596–8.
[9] Schaefer KE, Allison RD, Dougherty JH, Carey CR, Walker R, Yost F, et al. Pulmonary and circulatory adjustments determining limits of depths in breath-hold diving. Science 1968;162:1020–3.
[10] Arborelius Jr M, Ballidin UI, Lilja B, Lundgren CE. Hemodynamic changes in man during immersion with the head above water. Aerosp Med 1972;43:592–8.
[11] Begin R, Epstein M, Sackner MA, Levinson R, Dougherty R, Duncan D. Effects of water immersion to the neck on pulmonary circulation and tissue volume in man. J Appl Physiol 1976;40:293–9.
[12] Kiyan E, Aktas S, Toklu AS. Hemoptysis provoked by voluntary diaphragmatic contractions in breath-hold divers. Chest 2001;120:2098–100.
[13] Pavlik G, Olexó Z, Osváth P, Sidó Z, Frenkl R. Echocardiographic characteristics of male athletes of different age. Br J Sports Med 2001;35:95–9.