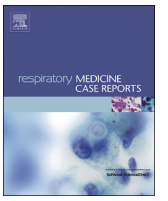
Respiratory Medicine Case Reports 39 (2022) 101722

Contents lists available at ScienceDirect

Respiratory Medicine Case Reports

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



Case Report

Rare case of occupational pulmonary hemorrhage in a firefighter

Sheiphali A. Gandhi [a, *], T.J. Welch [b], Kirk D. Jones [c], Kristin J. Cummings [d], Laura Styles [b, d], Paul D. Blanc [a], Robert J. Harrison [a, d]

[a] Department of Medicine, Division of Occupational and Environmental Medicine, University of California San Francisco, San Francisco, CA, USA
[b] Public Health Institute, Oakland, CA, USA
[c] Department of Pathology, University of California San Francisco, San Francisco, CA, USA
[d] Occupational Health Branch, California Department of Public Health, Richmond, CA, USA

A B S T R A C T

Alveolar hemorrhage associated with physical exertion, known as exercise-induced pulmonary hemorrhage (EIPH), is a rare condition linked to strenuous exertion. This can be an unusual form of respiratory and occupational illness. We present the case of a healthy firefighter who developed fatal pulmonary hemorrhage after participating in a strenuous physical training exercise regimen. This case represents a severe presentation of EIPH, which results from the disruption of the pulmonary blood-gas barrier as a result of strenuous exertion. Clinicians caring for those in vocations and recreation involving extremely vigorous exercise should be aware that such activities can cause EIPH.

1. Background

We present a case of a previously healthy 43-year-old firefighter who developed acute, fatal diffuse alveolar hemorrhage in the course of on-the-job cardiovascular physical training. Alveolar hemorrhage associated with physical exertion, known as exercise-induced pulmonary hemorrhage (EIPH), is a rare but well-documented event linked to athletic pursuits and other forms of exertion.

2. Case presentation

At the time of the incident, the 43-year-old firefighter was in excellent physical condition and without chronic health conditions. On the day in question, he was running as part of his regular physical training regimen. Witnesses reported that he appeared fatigued and breathless. A colleague trained in emergency medical services (EMS) witnessed the firefighter's collapse near his fire station. The firehouse crew activated outside EMS services. On initial assessment, paramedics noted intact pulses and shallow breathing. Initial oxygen saturation by pulse oximetry was 66% on room air; blood pressure was unmeasurable. Glucose was within normal range by a point-of-care test strip. During transport to a hospital facility, the paramedics performed bag-valve-mask ventilation, noting resistance to the delivery of inhaled breaths.

When the firefighter arrived at the hospital emergency department, he was found to be pulseless with an initial electrical rhythm of asystole. Cardiopulmonary resuscitation was initiated. Following intubation, gross quantities of bloody secretions in the endotracheal tube were observed, and, during continued resuscitation efforts, the secretions became progressively bloodier. Cardiac ultrasound demonstrated a lack of cardiac activity despite prolonged attempted resuscitation. Further resuscitative efforts were discontinued. Laboratory data demonstrated a coagulation profile, platelet, and troponin levels within the normal reference range and a negative COVID-19 polymerase chain reaction (PCR) test.

* Corresponding author. University of California San Francisco, Division of Occupational and Environmental Medicine, 2330 Post St, Ste 460, San Francisco, CA, 94117, USA.
E-mail address: sheiphali.gandhi@ucsf.edu (S.A. Gandhi).

https://doi.org/10.1016/j.rmcr.2022.101722
Received 19 June 2022; Accepted 25 July 2022
Available online 4 August 2022

On autopsy, the lungs exuded large amounts of pinkish frothy fluid, and the tracheobronchial tree contained bloody, watery fluid. The tracheobronchial mucosa was erythematous. The pulmonary vasculature did not demonstrate gross thrombi or emboli. Cardiac examination showed a mildly dilated right atrium and ventricle, but no fibrosis or necrosis were present. The coronary arteries were normal for age, and no other cerebrovascular abnormalities were found. Microscopic examination of the lungs revealed marked congestion with many alveoli containing blood (Fig. 1). No hallmarks of inflammatory capillaritis (as those seen in systemic lupus erythematosus or anti-glomerular basement membrane syndrome) were noted on pathology. Postmortem toxicology results were negative. The autopsy diagnosis was EIPH.

The firefighter had been in the service for 19 years, holding the job title of Fire Suppression Technician EMT-Basic. As part of his employment, he underwent annual health surveillance examinations, being repeatedly cleared for duty. Examinations included exercise testing with an elevated ramp in which he achieved 11.80 METS in 7:02 minutes with appropriate blood pressure and heart rate elevations. Prior screening laboratory testing demonstrated normal total cholesterol with elevated high-density lipoprotein, while glucose and renal function tests were within the normal range. His annual screening spirometry results were unremarkable and without obstructive or restrictive deficits. There was no evidence of excess spirometric decline. There was no reported history of previous hemoptysis or hypoxia with exercise.

3. Discussion

Although rare in humans, EIPH is familiar in veterinary medicine as a syndrome following intense exertion in thoroughbred racehorses [1]. Human cases of EIPH, over a range of severity, have been reported in both athletes and nonathletes following strenuous exertion. In one case report, two ultramarathoners developed hemoptysis and shortness of breath. Chest radiographs demonstrated bilateral alveolar consolidations. They improved within 24 hours, without residual findings [2]. Other reports, including experimental exposure data, provide further evidence of pulmonary hemorrhage following exercise in runners based on bronchoscopic findings [3,4]. Exercise-induced pulmonary hemorrhage also has been identified in military personnel participating in vigorous training exercises. For example, eight military personnel developed hemoptysis and shortness of breath during an aquatic training exercise [5]. Another case of EIPH occurred in a member of the Australian Defense Forces who developed recurrent hemoptysis reproducible by vigorous training exercises [6]. Aside from exercise, extremes of intrathoracic pressure may also induce EIPH. An avid saxophone player developed hemoptysis and bilateral patchy infiltrates following 6 h of instrument playing. Bronchoscopy, in that case, was consistent with alveolar hemorrhage [7].

Our case is an example of EIPH at the severe end of the spectrum, confirmed clinically and pathologically. Prior to the incident, the firefighter had been healthy and met cardiopulmonary fitness for duty requirements. Thus, this case underscores the difficulty in predicting EIPH risk. An experimental study, previously cited, produced mild disease in six elite cyclists who sprinted at maximal effort, with bronchoscopy performed within 1 h of exercise [3]. The bronchoalveolar lavage (BAL) samples obtained, when compared to sedentary subjects, demonstrated higher concentrations of red blood cells and total protein. These findings support the hypothesis that strenuous exercise can result in impairment of the blood-gas barrier integrity [3]. The same investigators also examined the effect of more prolonged but submaximal exercise in a similar group of athletes: no changes in BAL composition were seen [8].

In a 26-year-old healthy male who underwent regular research bronchoscopies, researchers noted that coincidentally, on the day following a marathon, his unrelated research bronchoalveolar lavage showed gross evidence of bleeding, which was increasingly evident with serial lavage aliquots [4]. Analysis of the lavage fluid demonstrated markedly elevated concentrations of hemoglobin, iron, and ferritin when compared to prior bronchoscopic samples from the same subject. A follow-up bronchoscopy two months later and without such exercise demonstrated resolution of the abnormal findings. A relevant case of pulmonary hemorrhage in nonathletes was reported following a vigorous game of handball. Chest tomography demonstrated diffuse, bilateral, patchy airspace disease;

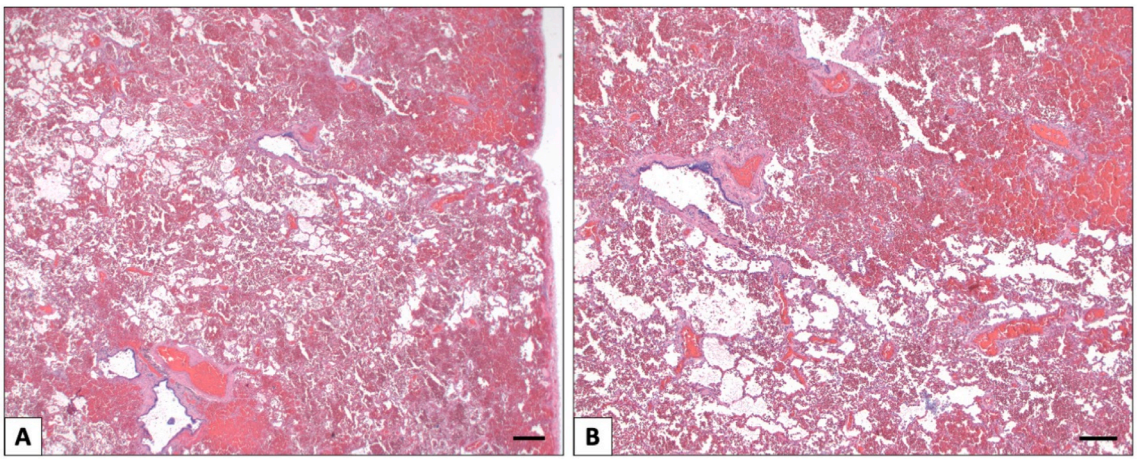


Fig. 1. Autopsy section from the lung shows alveolar filling and expansion by numerous red blood cells and sparse filbrin there is no under lyinging vasuilities or alveloar capillarities (hemotoxylin and eosin,(A)scale bar = 200 μm (B)scale bar = 500 μm. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.).

bronchoscopy measured increasing red blood cell concentrations in serial lavage aliquots. One month after presentation, the patient's signs and symptoms had completed resolved [9].

The scenario of exercise-induced pulmonary edema without frank hemorrhage is more commonly recognized than EIPH and may reflect a related phenomenon along a pathophysiologic spectrum of response. Such pulmonary edema, for example, is particularly evident in water-based sports [10,11]. A spectrum of such adverse effects presumes that the shared mechanism underpinning both exercise-related pulmonary edema and EIPH is increased pulmonary capillary transmural pressure affecting the blood-gas barrier of the lungs, resulting in vascular permeability, pulmonary edema, and in severe cases, progression to pulmonary hemorrhage [4,12,13]. This progression is exemplified in the case discussed above, in which military personnel developed hemoptysis while swimming but had radiographic findings and additional symptoms consistent with pulmonary edema [5].

The blood-gas barrier of the mammalian lung is potentially fragile, measuring only 0.2–0.4 μm to allow for the diffusion of oxygen and carbon dioxide across the tissues. Structural changes or damage to the capillary walls can cause increased permeability and influx of both proteinaceous fluid and red blood cells into the alveolar space [12,13]. Severe increases in capillary pressures have been demonstrated to cause structural changes to the capillary wall, which can be associated with high-permeability edema and/or alveolar hemorrhage [13].

Studies in humans and animal models have demonstrated increased pulmonary capillary pressures during intense exercise due to the high left ventricular filling pressures required to maintain cardiac output. Pulmonary limitations to exercise have been recognized in the elite athlete, but the mechanisms involved have not been established [14]. Clinical evidence of exercise-induced pulmonary edema and hemorrhage plausibly represent the severe end of the spectrum as vascular pressures rise [12,15].

Clearly recognized syndromes of occupational illness and injury, especially those linked to athletic pursuits, may not be adequately considered as potential adverse outcomes in the range of pulmonary diseases. This case underscores that EIPH can be caused by vigorous exercise in work-related conditions in addition to recreational activities.

Key learning points

What is already known about this subject

- Strenuous exercise may disrupt the blood-gas barrier of the lung, potentially causing an influx of proteinaceous fluid and red blood cells into the airways. In its most severe form, this can result in fatal pulmonary hemorrhage

What this study adds

- This care represents, to our knowledge, the first reported fatal case of EIPH as an occupational injury.

What impact this may have on practice or policy

- Clinicians caring for those in vocations and recreation involving very vigorous exercise (including emergency services workers, athletes, and the military) need to be aware of EIPH, including the specter of severe adverse outcomes.

CDPH publication policy disclaimer

''The findings and conclusions in this article are those of the author(s) and do not necessarily represent the views or opinions of the California Department of Public Health or the California Health and Human Services Agency.''

Declaration of competing interest

None. The authors declare no conflicts of interest.

References

[1] K.W. Hinchcliff, L.L. Couetil, P.K. Knight, et al., Exercise induced pulmonary hemorrhage in horses: American college of veterinary internal medicine consensus statement, J. Vet. Intern. Med. 29 (2015) 743–758.
[2] J.K. McKechnie, W.P. Leary, T.D. Noakes, et al., Acute pulmonary oedema in two athletes during a 90-km running race, S. Afr. Med. J. 56 (1979) 261–265.
[3] S.R. Hopkins, R.B. Schoene, W.R. Henderson, et al., Intense exercise impairs the integrity of the pulmonary blood-gas barrier in elite athletes, Am. J. Respir. Crit. Care Med. 155 (1997) 1090–1094.
[4] A.J. Ghio, C. Ghio, M. Bassett, Exercise-induced pulmonary hemorrhage after running a marathon, Lung 184 (2006) 331–333.
[5] D. Weiler-Ravell, A. Shupak, I. Goldenberg, et al., Pulmonary oedema and haemoptysis induced by strenuous swimming, BMJ 311 (1995) 361–362.
[6] A. Kruavit, M. Jain, D. Fielding, et al., Exercise-induced endobronchial hemorrhage: a rare clinical presentation, Respirology Case Reports 4 (2016) e00163.
[7] Y.-C. Ko, M.-P. Dai, C.-C. Ou, Playing saxophone induced diffuse alveolar hemorrhage: a case report, Ir. J. Med. Sci. 179 (2010) 137–139.
[8] S.R. Hopkins, R.B. Schoene, W.R. Henderson, et al., Sustained submaximal exercise does not alter the integrity of the lung blood-gas barrier in elite athletes, J. Appl. Physiol. 84 (1998) 1185–1189.
[9] A. Diwakar, G.A. Schmidt, Exercise-induced pulmonary hemorrhage in a nonathlete: case report and review of physiology, Lung 192 (2014) 329–331.
[10] E. Hohmann, V. Glatt, K. Tetsworth, Swimming induced pulmonary oedema in athletes – a systematic review and best evidence synthesis, BMC Sports Sci Med Rehabil 10 (2018), https://doi.org/10.1186/s13102-018-0107-3.
[11] S. Spencer, J. Dickinson, L. Forbes, Occurrence, risk factors, prognosis and prevention of swimming-induced pulmonary oedema: a systematic review, Sports Medicine - Open 4 (2018) 43.
[12] J.B. West, K. Tsukimoto, O. Mathieu-Costello, et al., Stress failure in pulmonary capillaries, J. Appl. Physiol. 70 (1991) 1731–1742.
[13] B. West John, Mathieu-Costello Odile, Vulnerability of pulmonary capillaries in heart disease, Circulation 92 (1995) 622–631.
[14] D.B. Phillips, M.K. Stickland, Respiratory limitations to exercise in health: a brief review, Current Opinion Physiol. 10 (2019) 173–179.
[15] J.B. West, Invited review: pulmonary capillary stress failure, 2000. J. Appl. Physiol. 89 (1985) 2483–2489 ;discussion 2497.