Respiratory Medicine Case Reports 27 (2019) 100845

Contents lists available at ScienceDirect

Respiratory Medicine Case Reports

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

Case report

Pulmonary edema during the Boston Marathon

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A B S T R A C T

The Boston Marathon is the most popular marathon in the New England region and attracts some of the most qualified athletes participating from the United States and abroad. The race occurs in April, a month in the northeast characterized by unpredictable weather. While there are a number of well described weather-related medical complications that occur during exercise, less is known about noncardiogenic pulmonary edema (NCPE) in marathon runners, a condition that most physician are unfamiliar with. This phenomenon has been described in the literature as a complication of severe hyponatremia and cerebral edema. Here, we describe the case of a healthy athlete who took part in the Boston Marathon in 2018 and presented afterwards with hypothermia and NCPE. We also review the normal cardiopulmonary physiology along with the physiological changes and external factors impacting the respiratory system during exercise. The combination of significant physical exertion, cold and rainy weather and subsequent hypothermia, perhaps along with other less understood factors may have increased the permeability of his lungs and caused NCPE.

1. Background

The Boston Marathon is the world's oldest annual marathon and the most popular marathon of the New England region [1]. Athletes meeting qualifying standards fly each year from different continents to race from the town of Hopkinton to Boston's Copley Square. They endure a variety of weather conditions that have included extreme heat (> 90F), driving rain with gusting winds, and even snowfall [2]. The stress of running 26.2 miles (42.195 km) in less than optimal conditions is physically taxing even for a well-prepared endurance athlete, and more so for lesser trained participants. Unsurprisingly, marathon runners are at risk of experiencing a multitude of medical conditions affecting different organ systems, be it musculoskeletal, gastrointestinal, cardiac, renal, metabolic, or pulmonary [3]. Several reports of noncardiogenic pulmonary edema (NCPE) in marathon runners have been described in the literature, mostly in the setting of severe hyponatremia and cerebral edema [4–7]. Here, we describe a case of an otherwise healthy athlete who took part in the Boston Marathon and presented shortly before completing the race with hypothermia and NCPE.

2. Case presentation

A 55-year-old male athlete with no significant past medical history was brought to the Emergency Department after experiencing severe shortness of breath while nearing completion of a marathon. After running twenty miles in conditions of heavy rain, wind gusts of more than 25 miles per hour and temperatures in the 40s F, he developed progressive shortness of breath, productive cough with white sputum, lightheadedness, and rigors. He stopped running and sought medical assistance. Upon presentation to the emergency room, his vitals were significant for hypoxia (oxygen saturation 89% breathing room air) and hypothermia (94.4 F). His physical exam was significant for bibasilar crackles without wheezing and normal heart sounds. Laboratory studies revealed sodium of 141 mmol/L, NT- Pro-BNP of 184 pg/mL (upper limit of normal (ULN) 450), troponin of 0.01 ng/ml (ULN 0.03), creatine kinase of 441 U/L (ULN 397), PaO2 of 56.2 mmHg and PaCO2 of 37 mmHg on arterial blood gas measurements. A chest x-ray showed evidence of interstitial lung edema with Kerley B Lines and increased vascular markings in the upper lobes (Fig. 1). The patient subsequently underwent active core rewarming with warm IV fluids. A transthoracic echocardiogram showed a left ventricle ejection fraction of 63% without any diastolic dysfunction or valvular disease. The patient's oxygen needs decreased within hours without diuresis. The working diagnosis was NCPE/Interstitial lung edema, likely triggered by marathon running and/or hypothermia.

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https://doi.org/10.1016/j.rmcr.2019.100845
Received 3 January 2019; Received in revised form 14 April 2019; Accepted 14 April 2019

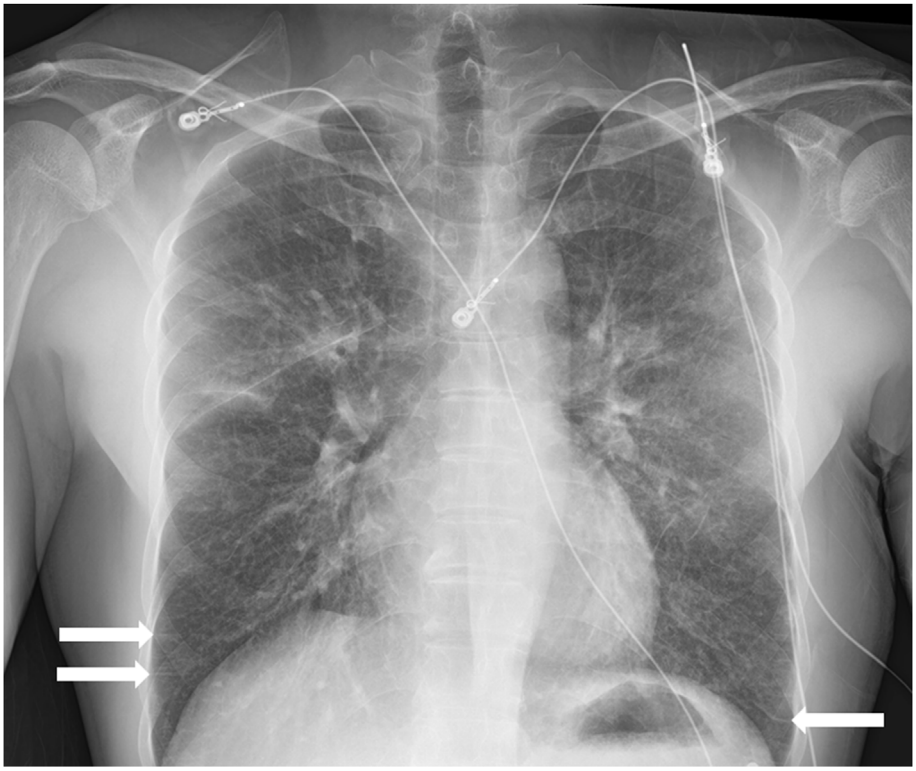

Fig. 1. Chest x-ray showing interstitial lung edema with Kerley B Lines (Arrows) and increased vascular markings in the upper lobe.

3. Discussion

Despite adequate training, unpredictable medical events may occur during marathon running such as dehydration, electrolyte abnormalities and even sudden cardiac death. Here, we review NCPE, a possibly underappreciated complication of marathon running, along with the respiratory physiological changes that occur while running and external factors that may also contribute.

4. Marathon induced NCPE

Pulmonary edema is traditionally attributed to either cardiogenic or noncardiogenic causes. Whereas well described cardiogenic causes can occur during long-distance endurance events, including myocardial infarction, papillary muscle rupture, and arrhythmias, the noncardiogenic causes are far less understood [2,3].

The concept of noncardiogenic pulmonary edema not related to hyponatermia was first introduced by Young et al., in 1987 [4]. A patient was described with pulmonary edema following a marathon that underwent a right heart catheterization demonstrating a low pulmonary capillary pressure and an initially depressed cardiac index which responded to rehydration. Zavorsky and colleagues expanded our understanding of this phenomenon by performing chest x-rays before and after a marathon on twenty-six runners. The post-race studies were done 19, 55 and 98 minutes after completion of the race [13]. Forty-six percent of the runners had findings consistent with mild to severe interstitial lung edema. Among women studied, more than half experienced pulmonary edema [5]. Other techniques have also been employed to assess for post-exercise pulmonary edema. Healthy athletes underwent chest ultrasonography following a race and the presence of comet-tail artifacts, an indicator of pulmonary edema, was assessed both before and after exercise. An increase in the presence of this finding supported greater amounts of extravascular lung water in the post-exercise examination [2]. Other cases of exercise-induced pulmonary edema have additionally been described [6–9].

5. Normal cardiopulmonary physiology and changes observed during strenuous exercise

Gas exchange occurs across the blood gas barrier (BGB) between the capillaries and alveoli in the lung. The barrier is required to be thin enough to allow for gas diffusion, yet strong enough to endure the pressures imposed on it by the mechanics and hemodynamics of exercise. The diffusion capacity of the BGB is affected by exercise. Manier et al. studied patients before and during the early recovery phase after running a marathon. The runners underwent a measurement of diffusing capacity of the lungs for carbon monoxide and nitric oxide, which were both decreased [10]. The generation of pulmonary edema during exercise is thought to be primarily responsible for the diffusing capacity changes noted. The explanation for the edema, when not cardiogenic in nature, appears related to both mechanical compromises in the BGB as well as an increase in pulmonary capillary pressure.

Between 1990 and the early 2000s, John West conducted multiple experiments to study the stress failure of capillaries. A broncho-alveolar lavage was performed on athletes after 7 minutes of cycling simulation. Athletes had higher red blood cells, total protein, albumin and leukotriene B4 compared to sedentary comparators. On the other hand, they had lower proportion of lymphocytes and similar proportion of neutrophils. These findings suggest that extreme exercise causes disturbances in the blood-gas barrier through mechanical mechanisms as opposed to inflammatory pathways [11].

The BGB must continuously endure multiple stressors as described by West et al. These include circumferential tension which stretches the layer of capillary endothelial cells, surface tension of the alveolar lining cell layer which acts to support the capillary wall, and longitudinal tension of tissue elements in the alveolar wall transmitted by collagen and associated with inflation of the lung [12]. In a normal state, these three forces are thought to counterbalance one another which promotes BGB stability. However, during exercise, all three are thought to intensify which may result in the counterbalancing relationship leading to BGB stress failure.

Of these described stressors, the best understood challenge to BGB stability during exercise is the elevation in the intra-vascular pressure which translates into increases in capillary circumferential tension. Gaar et al. (1967) devised a series of studies resulting in the generation of a formula to calculate pulmonary capillary pressure (PCP) [Gaar Equation: PCP = PAWP + 0.4 x (mPAP – PAWP)] [13]. It has been shown by Wagner et al. that mean pulmonary arterial wedge pressure (mPAWP) in normal subjects during severe exercise climbs to greater than 20 mmHg [14]. Thus, during severe rigorous exercise, the pulmonary capillary pressure may exceed 25 mmHg, setting the stage for hydrostatic pressures that promote interstitial edema.

6. External factors affecting NCPE

The respiratory system can also be affected by environmental factors. Hypothermia can impair ciliary activity and cause bronchorrhea or even NCPE [18,19]. Running in cold weather has also been associated with higher expiratory H2O2 concentrations, suggesting an increase in inflammation within the airways [20]. This inflammation may play a role in increasing respiratory tract vascular leak, mucosal permeability and secretions. Moreover, hypothermia has been described as an inhibitor of the active sodium transporter at the alveolar level. This inhibition is responsible of decreased alveolar fluid clearance, thus contributing to NCPE [21,22]. Cold weather can also stimulate the sympathetic nervous system and shift blood volume from the systemic to the pulmonary circulation thus potentially augmenting pulmonary capillary pressure.

Interestingly, a recent study demonstrated a circadian variation in the permeability of the lungs during endurance training in the cold weather. Morning exercise in cold weather causes higher physiological strain on the lungs than evening exercise [23]. The Boston Marathon start time is shortly after 9:00 a.m.

In summary, we present the unusual case of a patient who developed noncardiogenic pulmonary edema while running the Boston Marathon under challenging weather conditions. The combination of significant physical exertion and cold weather likely resulted in stress failure of the BGB compounded by elevations in pulmonary capillary pressure due to the severity of the physical challenge and centralized redistribution of blood volume. While exercise-induced bronchospasm is commonly considered when evaluating dyspnea of unexpected severity during or after exercise, physicians should also be aware of NCPE in marathon runners and when recognized provide appropriate supportive care to facilitate recovery.

Conflicts of interest

No conflicts, approved by all coauthors.

Funding

There was no financial support for this project.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.rmcr.2019.100845.

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