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Immersion Pulmonary Oedema and Alveolar Hemorrhage in a Long-Distance Asthmatic Swimmer: A Case Report

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Abstract

This report describes recurrent immersion pulmonary oedema associated with alveolar hemorrhage in a female asthmatic swimmer during a long distance competition in open water. During the race, she had repeated haemoptysis, shortness of breath and thoracic pain. A chest radiograph showed alveolar infiltrates in the right upper lobe. An electrocardiogram showed right atrium hypertrophy and a right axis suggestive of pulmonary hypertension. A computed tomography pulmonary angiography demonstrated diffuse ground glass opacities. A bronchoalveolar lavage showed large numbers of red blood cells, neutrophilsand hemosiderin-laden macrophages, suggestive of alveolar hemorrhage. Chest radiograph, taken 4 days after the competition, showed an almost complete resolution of infiltrates. This report illustrates the various components of this problem in some competitive swimmers and the potential contribution of unstable asthma and it also stresses the need to recognize this entity to provide appropriate management.

Keywords

Immersion pulmonary oedema; asthma; exercise-induced bronchoconstriction; swimmers

Abbreviations

BAL: Bronchoalveolar Lavage; ECG: Electrocardiogram; EIB: Exercise-induced Bronchoconstriction; FEV_1:Forced Expiratory Volume in one second; FVC: Forced Vital Capacity; LTB_4: Leukotriene B4; IPO: Immersion Pulmonary Oedema; L: Liter; PC_{20}: Provocative Concentration of Methacholine inducing a 20% fall in FEV_1; Vo_{2max}: Maximal Oxygen Consumption

Introduction

Immersion in water, at the thorax level, can provoke numerous physiological effects. First, the exerted water pressure increases hydrostatic pressure from venous and lymphatic compression, which leads to a redistribution of the blood from the legs to the thorax, increasing central blood volume. This increased blood volume induces a rise in atrial and pulmonary arterial pressures, increases right and left ventricular stroke volumes as well as the cardiac output.

Water pressure compresses the thorax affecting the pulmonary system. Indeed, a decrease of about 10% of chest circumference increases airway resistance and decreases residual volume,

expiratory flows, expiratory reserve volume, and vital capacity [1-3]. A slight decrease in diffusion capacity also occurs due to pulmonary vascular bed distention [4]. In cold water, vital capacity will further decrease due to peripheral vasoconstriction, which further increases central blood volume. Chest and lung compliance also decrease as a consequence of water pressure [5]. The outcome of all these effects is an increased breathing burden for the swimmer [6].

Furthermore, immersion increases blood flow to the kidneys and the creatinine clearance. Sodium excretion is increased following atrial natriuretic peptide release due to atrial distension. Potassium excretion is also increased while aldosterone production is decreased, resulting in increased diuresis. Finally, immersion can reduce thirst. Altogether, these effects can promote dehydration.

Intense exercise increases cardiac output raising pulmonary capillary pressure which, during immersion, causes central redistribution and blood pooling [7]. This in addition to the effect of the body prone or supine position increases cardiac preload [8].The peripheral vasoconstriction resulting from cold water immersion increases cardiac preload and after load, as well as pulmonary artery pressure.

Immersion pulmonary oedema (IPO) occurs rarely in healthy individuals, but has been reported with an increasing frequency in recent years, particularly in scuba divers, free divers [9-13], and endurance swimmers [14-17]. IPO is considered to occur in 1.4-1.8% of participants in an open water swim event and haemoptysis can be observed in over 50% of all cases [18-19]. This condition is generally associated with cold water immersion [20,21], but also with a variety of conditions including swimming in warm water (29°C), using a wetsuit or dry suit, being immersed in depths as low as 2.9 meters, or performing an endurance surface swim [21]. Clinical symptoms such as dyspnoea, cough and pinkish sputum usually resolve within 24 hours. Follow-up chest radiographs and echocardiograms are usually normal, but one-third of patients will have repeat episodes [18].

A review on IPO showed that in 60 cases analyzed, 67% were male and had a mean age of 36 years (range 18-61 years) [21]. Pre-existing medical conditions were present in 16%, arterial systemic hypertension being the most common (5 cases) while only 2 reported having asthma. In 22% of the cases, subjects had a prior or recurrent episode of IPO. Some reports found a similar prevalence of IPO in both sexes [22,23], but recent studies reported an increased prevalence of pulmonary edema in women [24,25].

This report presents the various clinical, imaging, and bronchoalveolar lavage features of a case of immersion pulmonary oedema associated with alveolar hemorrhage in a young asthmatic female swimmer during a long distance competition in open water. We discuss the mechanism that could lead to this complication of swimming, particularly in asthmatic subjects.

Case Presentation

On July 2013, a 23 year-old non-smoker female swimmer experienced acute haemoptysis, dyspnea and thoracic pain during a free open water swimming competition named "Traversée du Lac St-Jean", a 32 km swimming race with a duration of 7 to 8 hours. The water temperature during this competition was between 17-21°C (63-70°F). She has had asthma since childhood, has experienced exercise-induced asthma-like symptoms, and has had a concentration of inhaled methacholine causing a 20% decrease in the forced expiratory volume in one second (PC_{20}) of 5.4 mg/ml (May 2013). She

reported a respiratory tract infection in the days preceeding the race. Her medication included ciclesonide 200g with 2 daily inhalations, inhaled salbutamol 100g on demand, and pregabalin 150mg once a day. Despite reporting a mild exacerbation of asthma symptoms 4 days prior to the competition, she had not taken her asthma medication. She reported drinking approximately 4 liters (L) of water and sports beverages the evening before and 1.5L the morning of the competition. During the race, at 20 minute intervals, she drank around 0.5L of either a sports beverage that contained electrolytes, or hot chocolate.

Two and a half hours after the beginning of the competition, at the twelfth kilometer, she presented acute dyspnea, right thoracic pain, and haemoptysis. She reported no water aspiration. Despite the persistence of the symptoms, she finished the competition in seven hours and 41 minutes, after which she was rapidly brought to the regional hospital's emergency room for medical evaluation. It should be noted that the patient had reported a previous episode of mild haemoptysis, which had quickly resolved following the previous year's same event.

On her arrival at the hospital, the patient was alert, but dyspnoeic. At this time, her blood pressure was 114/75 mmHg, heart rate was 83 bpm, body temperature was 36.3°C (97.3°F), and oxygen saturation was 87%. The initial physical examination revealed diffuse crackles on pulmonary auscultation and a prolonged expiration time suggestive of increased airway resistance. Blood neutrophil count was increased (12.8×10^9/L). Hemoglobin was 142 g/L and platelet count 291×10^9/L.

A chest radiograph showed alveolar infiltrates in the right upper lobe (Figure 1). An electrocardiogram (ECG) showed a right atrium hypertrophy and a right axis deviation, suggestive of pulmonary hypertension. A computed tomography pulmonary angiography demonstrated ground glass opacities predominant within the right upper lobe of the lung, but also in the left superior lobe, the middle lobe, the lingula, and in the inferior lobes (Figure 2). This was compatible with alveolar hemorrhage. There was no pulmonary embolism, pleural, or pericardial effusion. The patient was considered too dyspnoeic to perform a spirometry. Initially, she was treated with inhaled salbutamol and intravenous methylprednisolone. The next day, she was transferred to the *Institut universitaire de cardiologie et de pneumologie de Québec*, a tertiary care center, for further evaluation.

A bronchoscopy was performed and the bronchoalveolar lavage (BAL) was increasingly haemorrhagic from the first to the third specimen obtained. The BAL showed 26.0×10^6 cells, including red blood cells, 33% neutrophils, and the presence of hemosiderin-ladden macrophages suggestive of alveolar hemorrhage. Exhaustive laboratory tests including antinuclear antibodies (C-ANCA , P-ANCA, anti-ENA) and cryoglobulinemia were all negative, as were anti-glomerular basement membrane (anti-GBM), anti-double-stranded DNA antibody (anti-dsDNA), complement proteins (C3 and C4), immunoglobulins (IgG, IgA, IgM), and serum protein electrophoresis. High-sensitivity C-reactive protein (CRP-hs) was 19.49 mg/L and fibrinogen 3.2 g/L. The urine analysis was normal, as were creatinine, urea, electrolytes, and complete blood count. These investigations excluded various conditions including vasculitis. Two days after the competition, an echocardiogram showed an abnormally dilated left atrium of 44ml/m^2 (normal < 28; mild dilation 29-33; moderate 34-39; severe > 40ml/m^2) and a tricuspid regurgitation (1/4). Pulmonary pressure was calculated at 25-30 mmHg. A control ECG was done the day after the competition and showed a complete resolution of signs of right atrium dilatation and right axis.

This suggested transient pulmonary hypertension.

The spirometry, completed 3 days after the race, showed a forced expiratory volume in one second (FEV_1) of 3.36L (90% of predicted), a FEV_1/forced vital capacity (FVC) of 80 %, and normal lung volumes & carbon monoxide diffusing capacity. In the following month, the FEV_1 further increased at 3.92 L (106% of predicted); the airway responsiveness was normal (PC_{20}> 16 mg/ml; and no fall in FEV_1 post Eucapnic Voluntary Hyperpnea), probably reflecting the intense asthma treatment initially received. When seen one month later, she used inhaled ciclesonide 200g two inhalations daily and rarely inhaled salbutamol.

Haemoptysis had persisted for 48 hours following the competition, but resolved rapidly. Dyspnea improved gradually over 48 hours, but she remained with cough and chest tightness during exercise until December 2013. A chest radiograph taken 4 days after the competition showed an almost complete resolution of infiltrates and was normal at one month. She reported no other episode of hemoptysis or exercise-induced dyspnea in the following months.

Discussion

IPO following swimming is an unusual event that has been reported more frequently in recent years [21,25]. The causes of IPO are not completely understood and several mechanisms may be involved. Moreover, it is not currently possible to predict who is at an increased risk of developing this condition, as many factors can trigger this phenomenon. As there is no specific diagnostic test for this condition, the diagnosis mainly stems from the clinical history, biological assessment (blood gases, cardiac enzymes, blood & urine catecholamine's), chest radiograph or computed tomography (CT-scan) (lung infiltrates), and cardiac echography [26].

In healthy subjects, a combination of factors including intense exercise, immersion, cold water, and over hydration can trigger a transient increase in the pulmonary capillary pressure resulting in oedema and hemoptysis. In this report, the combination of a previous episode, uncontrolled asthma, cold water and taking medication that promote fluid retention may be involved in the pulmonary oedema and alveolar hemorrhage of this young asthmatic female long-distance swimmer.

She had reported a previous episode of slight hemoptysis following the same competition the year before, suggesting a predisposition to this phenomenon. However, she reported no episode during training between the two events. This can be explained by the fact that the training sessions were performed in warmer water temperature, at lower intensity, shorter distance and smaller waves which represent conditions that are not as rigorous as the event itself. Furthermore, she had a history of asthma, but presented no other pre-existing medical condition associated with pulmonary oedema. A few days before the competition, she presented symptoms of the common cold, which could have affected her asthma, but in spite of this, she did not use her asthma medication at this time. The combination of a prolonged immersion in cold water and intense exercise in unstable asthma could have produced damage to the capillary-alveolar barrier [21,27].

During strenuous exercise, hyperventilation induced dehydration and heat loss of the airway mucosa may trigger bronchoconstriction and mucosal hypersecretion [28]. IPO can then be triggered by changes in respiratory mechanics. The forced inhalation against resistance, such as during a bronchospasm, may induce shear forces in the airway wall with mechanical and inflammatory injuries of

the bronchoalveolar structures [18,22,29] and may promote fluid leakage out of the capillaries and into the alveoli. Haemoptysis, observed in the present case, probably reflects such mechanical strain of capillary-alveolar network induced by strenuous exercise and bronchoconstriction [18,30]. Moreover, the vasculature of the lamina propria has been shown to be more prominent in asthmatic airways than in normal controls [31]. The bronchial circulation plays an important role in the regulation of heat and water loss in the airway, thus increased vascularity may predispose to exercise-induced bronchoconstriction (EIB) [32].

However, pulmonary oedema with acute asthma is an uncommon condition [33]. The negative pleural pressure and hyperinflation in asthma can increase the hydrostatic pressure of the pulmonary vasculature inducing damage to pulmonary capillaries causing leakage of plasma proteins and red cells into the interstitial space [34]. Intense exercise also can cause an increase in hydrostatic pressure because of the large filling pressure demanded by the left ventricle to develop the necessary cardiac output [35] adding an additional stress on the wall of the capillary, which can eventually break collagen fibers that are responsible for wall strength. Exercise-induced pulmonary hemorrhage was first described in race horses [36,37], but evidence of increased red blood cells in bronchoalveolar lavage fluid, increased lung opacities, and reduced pulmonary capacity was observed in triathletes, following a competition [17,38,39].

Indeed, it seems that the integrity of the blood-gas barrier can be altered in athletes at intense levels of exercise. There are several reports of hemoptysis and/or hemorrhagic pulmonary oedema in elite athletes [15,17,38,40,41]. The combination of hemoptysis, oedema and rapid recovery (24 hours) strongly suggests stress failure of the pulmonary capillaries [42]. Hopkins *et al*. [39] showed that cyclists had higher concentrations of red blood cells, total protein, albumin, and leukotriene B_4 (LTB_4) in BAL fluid, following a maximal exercise (6 to 8 minutes at 90% VO_{2max}), compared with sedentary subjects who did not exercise. Except for LTB_4, the absence of activation of the proinflammatory process suggests that the mechanism for altered blood-gas barrier was mechanical stress. The increase in LTB_4 is possibly due to the activation of alveolar macrophages resulting from the disruption of alveolar epithelial cells [42]. However, following submaximal exercise (1 hour at 75-80% of VO_{2max}), there is no difference between athletes and controls, suggesting that submaximal exercise is not sufficient to impair the blood-gas barrier [43].

IPO might result from a mismatch between right and left stroke volumes, e.g. a limited increase in left ventricular stroke volume compared with a relatively greater rise in right ventricular stroke volume during intense exercise [19]. However, healthy individuals should be able to adapt to such fluid shift [21]. The lymphatic system may be overloaded due to increased flow, but, on the other hand, the lymphatic flow may be also reduced following respiratory pattern changes and supine position of swimming [44]. In this case, taking pregabalin may have contributed to increased circulatory volume, as it promotes fluid retention.

In the case reported here, cold water, prolonged exercise duration and its intensity may have also promoted IPO. The temperature of the water was around 17-21°C (63-70°F) and the swimmer wore an open water full body long leg open swimwear, which provides no cold protection. An increased cardiac preload from immersion and a supine position in swimmers may be augmented by wearing tight-fitting

wetsuits and increase peripheral vasoconstriction due to cold, all leading to an even greater increase in central shift of the blood volume [16,17, 19].

Overhydration can also be a trigger for pulmonary oedema by increasing pulmonary vascular permeability and pressure [45,46]. Alteration in fluid-electrolyte balance makes the blood hypotonic and fluid diffuses across an osmotic gradient into the brain to compensate for hyponatremia [47,48]. Several reports have considered pre-swim overhydration (e.g. 5L consumed 2h prior to swimming) as a risk factor of IPO [16,17]. The recommendations from the 2008 International Exercise-Associated Hyponatremia Consensus Development Conference states that athletes should drink a maximum of 0.4 to 0.8 L/h during an event [49]. In the present case, the swimmer had not been drinking large volumes of beverages before the competition (1.5 L in the morning before the race), but she had taken pregabalin which promotes fluid retention. However, she had a normal natremia (138mmol/L) on her arrival at the hospital.

Prediction of recurrent IPO in an athlete with a previous episode remains difficult. Whether or not an athlete should return to diving or swimming after an episode of IPO should be determined on a case by case basis. The decision should be based on the physical condition, history of hypertension or cardiovascular disease and the type of exercise being considered. However, athletes should be aware of the apparition of IPO symptoms during future exercise and should stop if they develop marked dyspnea or haemoptysis. Currently, no treatment has been shown to prevent the onset of IPO, but some precautions can be taken by athletes to avoid such an episode (Table 1). Ensuring adequate asthma control could reduce this risk as asthma is often undertreated in athletes.

In this case study, the swimmer continued to train and compete without any further episode of haemoptysis or dyspnea during the month following the IPO episode. Her asthma was well controlled, with a daily intake of inhaled corticosteroids and her chest radiograph remained normal. As the long-term consequences of repeated IPO episodes are unknown, further studies are necessary to better understand why this phenomenon occurs and to uncover how it could be prevented.

Conclusion

We report the case of a long-distance asthmatic swimmer who had evidences of IPO and alveolar hemorrhage during a long distance swimming competition in open water. In this case, it likely represents a second episode caused by a combination of prolonged swimming in cold water, uncontrolled asthma, and medication favoring fluid retention. This report reviews the general management of this condition and stresses the need to identify this problem in athletes, which can mimic or be associated with asthma.

Acknowledgments

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Figures

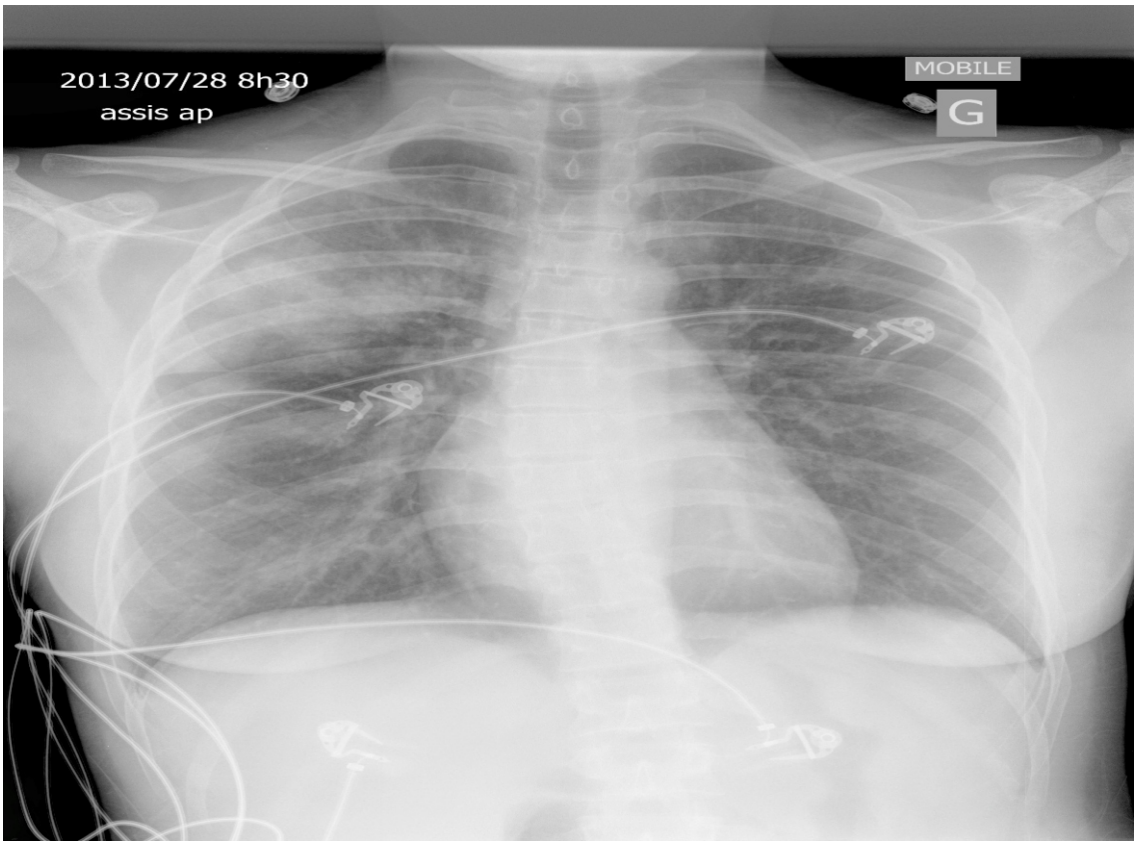

Figure 1: (28-07-2013 8h30am) Chest radiograph showed alveolar infiltrates within the right upper lobe.

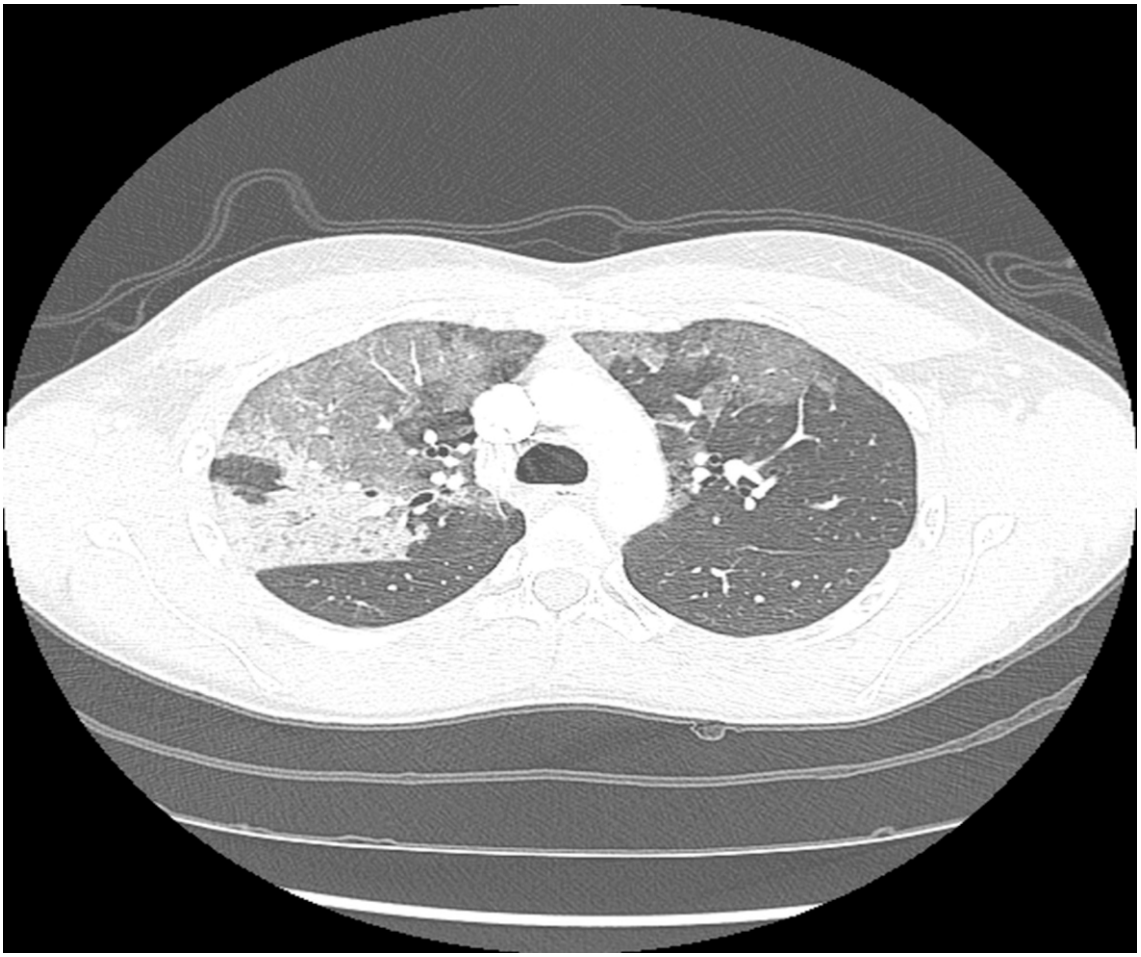

Figure 2: (27-07-2013 6h14pm) Computed tomography pulmonary angiography demonstrated ground glass opacities predominant within the right upper lobe of the lung, but also in the left superior lobe and the middle lobe.

Table

Potential contributors or risk factors to IPO	Potential preventive measures
Exercise in open water	Warm-up swim immediately before the competition Request assistance during the swim if symptoms of unusual breathlessness or hemoptysis develop
Water temperature	Avoid training or competing in cold water (16-30°C) Acclimate to the water conditions and temperature
Chest wall compression	Avoid using a swimsuit/wetsuit/drysuit which is too tight
Previous IPO episodes	Search for risk factors Stop exercise if symptoms of unusual breathlessness or hemoptysis occur
Asthma worsening	Ensure control of asthma before the competition (perform spirometry)
Viral respiratory infection	Avoid high intensity exercise
Specific medical condition (Systemic hypertension)	Ensure treatment and control of the condition
Overhydration	Avoid drinking too much before the event (less than 5L in 2 hours) Limit hydration to 400 to 800 ml/h during the event Drink beverages that contain electrolytes (at least 10% of Na and K). *Note: Na: Sodium, K: Potassium*

References

1. Taylor NA and Morrison JB. Static and dynamic pulmonary compliance during upright immersion. Acta Physiol Scand. 1993;149(4):413-417.

2. Dressendorfer RH, Morlock JF, Baker DG and Hong SK. Effects of head-out water immersion on cardiorespiratory responses to maximal cycling exercise. Undersea Biomed Res. 1976;3(3):177-187.

3. Romer LM, McConnell AK and Jones DA. Inspiratory muscle fatigue in trained cyclists: effects of inspiratory muscle training. Med Sci Sports Exerc. 2002;34(5):785-792.

4. Manier G, Moinard J, Techoueyres P, Varene N and Guenard H. Pulmonary diffusion limitation after prolonged strenuous exercise. Respir Physiol. 1991;83(2):143-153.

5. Bondurant S, Hickam JB and Isley JK. Pulmonary and circulatory effects of acute pulmonary vascular engorgement in normal subjects. J Clin Invest. 1957;36(1 Part 1):59-66.

6. Navarro RR, Romero L and Williams K. Nasal issues in athletes. Curr Sports Med Rep. 2013;12(1):22-27.

7. West JB, Tsukimoto K, Mathieu-Costello O and Prediletto R. Stress failure in pulmonary capillaries. J Appl Physiol (1985). 1991;70(4):1731-1742.

8. Norsk P, Bonde-Petersen F and Warberg J. Central venous pressure and plasma arginine vasopressin in man during water immersion combined with changes in blood volume. Eur J Appl Physiol Occup Physiol. 1986;54(6):608-616.

9. 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(5):762-767.

10. Boussuges A, Pinet C, Thomas P, Bergmann E, Sainty JM and Vervloet D. Haemoptysis after breath-hold diving. Eur Respir J. 1999;13(3):697-699.

11. Boussuges A, Succo E, Bergmann E and Sainty JM. [Intra-alveolar hemorrhage. An uncommon accident in a breath holding diver]. Presse Med. 1995;24(25):1169-1170.

12. Kiyan E, Aktas S and Toklu AS. Hemoptysis provoked by voluntary diaphragmatic contractions in breath-hold divers. Chest. 2001;120(6):2098-2100.

13. Strauss MB and Wright PW. Thoracic squeeze diving casualty. Aerosp Med. 1971;42(6):673-675.

14. Lund KL, Mahon RT, Tanen DA and Bakhda S. Swimming-induced pulmonary edema. Ann Emerg Med. 2003;41(2):251-256.

15. Mahon RT, Kerr S, Amundson D and Parrish JS. Immersion pulmonary edema in special forces combat swimmers. Chest. 2002;122(1):383-384.

16. Shupak A, Weiler-Ravell D, Adir Y, Daskalovic YI, Ramon Y and Kerem D. Pulmonary oedema induced by strenuous swimming: a field study. Respir Physiol. 2000;121(1):25-31.

17. Weiler-Ravell D, Shupak A, Goldenberg I, Halpern P, Shoshani O, Hirschhorn G, et al. Pulmonary oedema and haemoptysis induced by strenuous swimming. BMJ. 1995;311(7001):361-362.

18. Adir Y, Shupak A, Gil A, Peled N, Keynan Y, Domachevsky L, et al. Swimming-induced pulmonary edema - Clinical presentation and serial lung function. Chest. 2004;126(2):394-399.

19. Casey H, Dastidar AG and MacIver D. Swimming-induced pulmonary oedema in two triathletes: a novel pathophysiological explanation. J R Soc Med. 2014;107(11):450-452.

20. Wilmshurst PT, Nuri M, Crowther A and Webb-Peploe MM. Cold-induced pulmonary oedema in scuba divers and swimmers and subsequent development of hypertension. Lancet. 1989;1(8629):62-65.

21. Koehle MS, Lepawsky M and McKenzie DC. Pulmonary oedema of immersion. Sports Med. 2005;35(3):183-190.

22. Slade JB, Jr., Hattori T, Ray CS, Bove AA and Cianci P. Pulmonary edema associated with scuba diving : case reports and review. Chest. 2001;120(5):1686-1694.

23. Coulange M, Rossi P, Gargne O, Gole Y, Bessereau J, Regnard J, et al. Pulmonary oedema in healthy SCUBA divers: new physiopathological pathways. Clin Physiol Funct Imaging. 2010;30(3):181-186.

24. Zavorsky GS, Milne EN, Lavorini F, Rienzi JP, Lavin KM, Straub AM, et al. Interstitial lung edema triggered by marathon running. Respir Physiol Neurobiol. 2014;190(137-141.

25. Miller CC, 3rd, Calder-Becker K and Modave F. Swimming-induced pulmonary edema in triathletes. Am J Emerg Med. 2010;28(8):941-946.

26. Ware LB and Matthay MA. Clinical practice. Acute pulmonary edema. N Engl J Med. 2005;353(26):2788-2796.

27. Biswas R, Shibu PK and James CM. Pulmonary oedema precipitated by cold water swimming. Br J Sports Med. 2004;38(6):e36.

28. Fontanari P, Burnet H, ZattaraHartmann MC and Jammes Y. Changes in airway resistance induced by nasal inhalation of cold dry, dry, or moist air in normal individuals. J Appl Physiol. 1996;81(4):1739-1743.

29. West JB and Mathieu-Costello O. Stress failure of pulmonary capillaries: role in lung and heart disease. Lancet. 1992;340(8822):762-767.

30. Cochard G, Arvieux J, Lacour JM, Madouas G, Mongredien H and Arvieux CC. Pulmonary edema in scuba divers: recurrence and fatal outcome. Undersea Hyperb Med. 2005;32(1):39-44.

31. Orsida BE, Li X, Hickey B, Thien F, Wilson JW and Walters EH. Vascularity in asthmatic airways: relation to inhaled steroid dose. Thorax. 1999;54(4):289-295.

32. Gilbert IA, Fouke JM and McFadden ER, Jr. Heat and water flux in the intrathoracic airways and exercise-induced asthma. J Appl Physiol (1985). 1987;63(4):1681-1691.

33. Stalcup SA and Mellins RB. Mechanical forces producing pulmonary edema in acute asthma. N Engl J Med. 1977;297(11):592-596.

34. O'Callaghan MW, Pascoe JR, Tyler WS and Mason DK. Exercise-induced pulmonary haemorrhage in the horse: results of a detailed clinical, post mortem and imaging study. VIII. Conclusions and implications. Equine Vet J. 1987;19(5):428-434.

35. Manohar M. Pulmonary vascular pressures of thoroughbreds increase rapidly and to a higher level with rapid onset of high-intensity exercise than slow onset. Equine Vet J. 1994;26(6):496-499.

36. West JB and Mathieu-Costello O. Stress failure of pulmonary capillaries as a mechanism for exercise induced pulmonary haemorrhage in the horse. Equine Vet J. 1994;26(6):441-447.

37. Whitwell KE and Greet TR. Collection and evaluation of tracheobronchial washes in the horse. Equine Vet J. 1984;16(6):499-508.

38. McKechnie JK, Leary WP, Noakes TD, Kallmeyer JC, MacSearraigh ET and Olivier LR. Acute pulmonary oedema in two athletes during a 90-km running race. S Afr Med J. 1979;56(7):261-265.

39. Hopkins SR, Schoene RB, Henderson WR, Spragg RG, Martin TR and West JB. Intense exercise impairs the integrity of the pulmonary blood-gas barrier in elite athletes. Am J Respir Crit Care Med. 1997;155(3):1090-1094.

40. Stefanko G, Lancashire B, Coombes JS and Fassett RG. Pulmonary oedema and hyponatraemia after an ironman triathlon. BMJ Case Rep. 2009;2009

41. Luks AM, Robertson HT and Swenson ER. An ultracyclist with pulmonary edema during the Bicycle Race Across America. Med Sci Sports Exerc. 2007;39(1):8-12.

42. West JB. Vulnerability of pulmonary capillaries during exercise. Exerc Sport Sci Rev. 2004;32(1):24-30.

43. Hopkins SR, Schoene RB, Henderson WR, Spragg RG and West JB. Sustained submaximal exercise does not alter the integrity of the lung blood-gas barrier in elite athletes. J Appl Physiol (1985). 1998;84(4):1185-1189.

44. MacIver DH and Clark AL. The Vital Role of the Right Ventricle in the Pathogenesis of Acute Pulmonary Edema. Am J Cardiol. 2015;

45. Smith WS and Matthay MA. Evidence for a hydrostatic mechanism in human neurogenic pulmonary edema. Chest. 1997;111(5):1326-1333.

46. McClellan MD, Dauber IM and Weil JV. Elevated intracranial pressure increases pulmonary vascular permeability to protein. J Appl Physiol (1985). 1989;67(3):1185-1191.

47. Noakes TD, Goodwin N, Rayner BL, Branken T and Taylor RK. Water intoxication: a possible complication during endurance exercise, 1985. Wilderness Environ Med. 2005;16(4):221-227.

48. Ayus JC, Varon J and Arieff AI. Hyponatremia, cerebral edema, and noncardiogenic pulmonary edema in marathon runners. Ann Intern Med. 2000;132(9):711-714.

49. Hew-Butler T, Ayus JC, Kipps C, Maughan RJ, Mettler S, Meeuwisse WH, et al. Statement of the Second International Exercise-Associated Hyponatremia Consensus Development Conference, New Zealand, 2007. Clin J Sport Med. 2008;18(2):111-121.

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