Introduction

Spontaneous pneumothorax (SP) refers to the presence of air or gas in the space between the chest and the lungs, in other words a “collapse” of the lungs which prevents complete inflation [1]. More than a half of pneumothorax cases are traumatic, either iatrogenic or accidental; the remaining are referred to as SP. There are two types of SP - primary and secondary. Primary spontaneous pneumothorax (PSP) is an idiopathic disease which appears in otherwise healthy person. Secondary spontaneous pneumothorax (SSP) is an underlying condition which appears in patients with various pulmonary disorders such as chronic obstructive pulmonary disease (COPD), cystic fibrosis, acquired immune deficiency syndrome and tuberculosis [2]. It mostly affects the elderly population and if it is not treated immediately, the condition can get worse and cause death. Chest pain and sometimes mild breathlessness are the most common symptoms in PSP. Some patients may wait for a few days before they seek medical help, although there are many cases when PSP is life threatening [1, 2]. The course of SP remains unpredictable with a recurrence rate ranging from 25 – 54%. An important risk factor for PSP is smoking. Also, patients with PSP tend to be taller than controls. Furthermore, PSP often oc-
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curs among young adults [3]. The rise of negative pleural pressure increases from the lung base to the apex, so that alveoli at the lung apex in tall persons are subject to remarkably greater distending pressure than those at the base of the lung, and the vectors in theory, predispose to the development of apical subpleural blebs [2]. Surprisingly, SP may occur even during sedentary activity, despite recognized association between the onset of pneumothorax and physical activity [3].

Case Report

A 49-year-old man (190 cm tall, 90 kg weight, body mass index 24.93 kg/m²) was admitted to our Emergency Department with a high level of suspicion for acute coronary syndrome. He was sweating, pale, with shortness of breath and chest pain that spread to the arms and back with no response to nitroglycerin. The patient felt a severe chest pain an hour before admission after a short rapid run to catch the bus to work. In his youth, he was an active professional footballer and after retirement he continued playing football three times a week and occasionally went hiking. At his previous medical check-up, he reported that he climbed Mount Everest 3 days before. After returning home, he had a dry cough followed by mild dyspnea without any treatment. He used some secretolytic bang drag to eliminate the symptoms, but without any improvement. There was no relevant personal, traumatic or psychiatric history or any basic lung disease. He was not a smoker and no allergies were reported. He had general medical examinations annually, and in his opinion he was in perfect condition without illness. On admittance, his blood pressure was 110/80 mmHg, pulse rate 106/min, respiratory rate 26/min, body temperature 36.7 °C, oxygen saturation 88% in room air and 96% with an oxygen mask at a flow of 8 L/min. On examination, he had breathing difficulties, with reduced breath sound on the right side. The physical examination showed no other abnormalities. Cardio sensitive enzymes (troponin, creatine kinase (CK), CK-MB) were within normal limits as well as complete blood count and biochemistry results. A 12-lead electrocardiogram showed sinus tachycardia with 110 beats/min and no ST-T changes or ischemic changes. Chest X-ray revealed a unilateral right sided pneumothorax with no mediastinal change (Figure 1). Based on radiographic findings, two emergency surgeons concurrently performed one-sided catheter drainage. Thereafter, his dyspnea and chest pain improved. The patient was hospitalized after consultation with the chest surgeon. Two days later, no blebs were found on chest computed tomography, which also showed that the right lung was fully expanded. On the third day after admission, after X-ray confirmation of pneumothorax absence, the catheter was removed. The patient was discharged from the hospital on the 7th day after admission without any complications. The six-month and annual follow-ups showed no recurrence. A written informed consent was obtained from the patient for publication of this case report and the accompanying images.

Discussion

The pathophysiology of PSP still remains unknown [4, 5]. There are several theories explaining the etiology of PSP. The first is that subpleural blebs and bullae that are found at the lung apices at thoracoscopy and on computed tomography scan in up to 90% of cases of PSP are considered to play a role [6]. The second theory asserts that pulmonary blebs are small subpleural thin walled spaces with presence of air, not larger than 1 – 2 cm in diameter; it is thought that bleb rupture permits the air to enter into pleural space causing pneumothorax. The third theory states that blebs are also observed in cell apoptosis which is a cells self-execution plan to guided rupture. There is a theory that the pleural lining cells are performing apoptosis leading to SP [6]. However, the location of unique or diffuse sites of air leakage in PSP is generally unknown [7, 8]. Interestingly, literature data show that 40% of SP cases are found in the left lung [9, 10], like in our patient. The potential contributing factors for PSP

Abbreviations

SP – spontaneous pneumothorax
PSP – primary spontaneous pneumothorax
SSP – secondary spontaneous pneumothorax
COPD – chronic obstructive pulmonary disease
CK – creatine kinase
PaO2 – partial pressure of oxygen
HVR – hypoxic ventilatory response

Figure 1. Chest X-ray showing a large unilateral pneumothorax on the right side
Slika 1. Radiogram grudnog koša sa velikim unilateralnim pneumotoraksom sa desne strane
are climate changes and cold temperatures with decreased atmospheric pressure [11]. Nowadlys, more and more people are looking for adventure travels, so they hike to high altitudes, over 2000 meters with hypobaric hypoxia environment [12]. Whether or not they have an underlying lung disease, acute exposure to hypoxia causes several critical changes in respiratory physiology that affects persons hiking at high altitudes. After the rise to high elevation, the low partial pressure of oxygen (PaO2) takes to an increase in minute ventilation, known as hypoxic ventilatory response (HVR). Mediated by the carotid bodies, the HVR varies among persons and tends to increase PaO2 [12]. The HVR also causes a decrease in the partial pressure of carbon dioxide (PaCO2); the resulting respiratory alkalosis causes a leftward shift of the oxyhemoglobin dissociation curve, which improves alveolar oxygen uptake and, to a lesser degree, reduces oxygen delivery to the tissues. This shift on left side is balanced by increased production of 2,3-di-phosphoglycerate by red blood cells after prominent increase elevation; in vivo P50 stays approximately the same as at the sea level, and the mechanism in turn produces a compensatory rightward shift in the curve [13, 14]. Theoretically, there is a concern that in patients with COPD, bullous emphysema may expand or/and rupture during exposure to lower atmospheric pressure, but these patients belong to a group at risk and they should avoid sudden changes in atmospheric pressure, until proven otherwise. Statistics suggest that this concern is not warranted, because it is found in limited available literature. In a research of nine non-COPD patients fast decompressed to a simulated altitude of 13,100 m, bleb and cyst size increased in just one patient without development of pneumothorax [15]. These discoveries were supported by other COPD patient-based studies: persons quickly decompressed to lower atmospheric pressure showed no pneumothorax nor clinical or radiographic evidence of bullae expansion, but it should be taken with reserve, because there is a limited number of studies analyzing this issue, only some case reports or studies including a small sample of respondents that focus on narrow end-points [16, 17].

It is difficult to draw concrete conclusions on understanding the pathophysiology of SPP from the previous studies and how it interacts with the high-altitude environment. However, it is possible to conclude what kind of patients should undergo medical examination before hiking and going to high-altitudes.

Given that increasing numbers of people are traveling to high altitudes for pleasure or work, and certain health conditions are frequent in the whole population, there is a high probability that many high altitude travelers have underlying medical diseases. While other organ systems, such as kidneys, heart and hematological system undergo important adaptations, we have to emphasize that lungs play a primary role in the early and late responses to high altitudes [18].

Conclusion

We suggest that patients with any pulmonary disorders who go hiking or travel to high altitudes should be examined before these activities. The evaluation of pre-travel health check-ups, in order to accurately predict the risk of high altitude travel, should be the aim of future research.

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