CASE REPORT



Cycling-induced recurrent spontaneous pneumomediastinum and pneumopericardium in a young female patient

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Abstract

We present an exceptional case of recurrent cycling-induced spontaneous pneumomediastinum and pneumopericardium in a female patient without any trauma. Radiological and endoscopic examinations were carried out to exclude other differential diagnoses. Decision for in-hospital observation and conservative treatment was made. No symptoms were reported 12 months after return to sports activity.

KEYWORDS

Hamman's syndrome, spontaneous pneumomediastinum, spontaneous pneumopericardium, sport medicine

1 | INTRODUCTION

Spontaneous pneumomediastinum (SPM) and pneumopericardium are important differential diagnoses in patients presenting with chest pain. Incidences of SPM range between 7–28 per 100,000 in male patient and 1–6 per 100,000 in female patient. Risk factors contributing to the development of SPM are smoking, thin stature, male sex, and young age (healthy adolescents,

young men, and parturient women).³ Further, history of physical exertion was recently reported in 21% of patients presenting with SPM.⁴ Examples for sports activities previously linked with occurrence of SPM are soccer, rugby, track and field athletics, scuba diving, and weight lifting.^{2,5-8} To the best of our knowledge, this is the first case report, which illustrates cycling as an unusual trigger for spontaneous pneumomediastinum and pneumopericardium.

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2 | CASE REPORT

A 21-year-old female patient presented to the Emergency Department (ED) with retrosternal chest pain, coughing, pharyngeal globus sensation, and dysphonia. She reported gradual progress of these symptoms within the last hours after she had initially noticed episodes of tachycardia and decline of performance during endurance training on her racing bike. She denied recent chest trauma, and medical history was unremarkable. The patient was not taking medications, did not smoke, and had no significant family history.

Physical examination revealed a Caucasian female patient in good general and nutritional status in no distress. Right-sided cervicothoracic swelling was visible. She was afebrile, and vital signs were in the normal ranges. The cardiac examination revealed a normal rate and a regular rhythm. There were bilaterally equal breath sounds in pulmonary examination. Cervicothoracic crepitus was noticed due to an underlying subcutaneous emphysema. The remainder of the examination was unremarkable.

An electrocardiogram (ECG) and chest radiograph were obtained. The ECG was normal. The radiograph showed presence of mild pneumomediastinum and pneumopericardium (Figure 1).

In consensus with the patient, decision for conservative treatment was made. This treatment consisted of inhospital surveillance for 48 h and constant monitoring of vital signs during the first 24 h after hospital admission. Retrosternal chest pain was sufficiently treated by ibuprofen. Follow-up radiograph on Day 8 after discharge showed near-complete resolution of the air, and the patient returned to sports activity.

Two years later, she presented to the ED with a second episode of chest tightness, after having done endurance training on her racing bike the same day. Post medical history was unchanged, and she again denied specific triggers for pneumomediastinum. Physical examination revealed good general status. Except for mild tachycardia, general cardiac and pulmonary examinations were unremarkable. There was no subcutaneous emphysema. Laboratory results showed mild leukocytosis.

Due to recurrence of symptoms, further diagnostic workup contained low-dose chest computed tomography (CT), bronchoscopy, and esophagogastroduodenoscopy. The CT revealed a second episode of pneumomediastinum and pneumopericardium, but again no pneumoperitoneum (Figure 2). Bronchoscopy and esophagogastroduodenoscopy showed no evidence of pathologies.

As 2 years before, decision for conservative management without any surgical intervention was made. Vital signs were monitored for 24 h before she was discharged the next day. Finally, the patient returned to regular bike training two weeks later and had not reported any further problems during a 12-month follow-up period.

3 DISCUSSION

Spontaneous pneumomediastinum (SPM), also known as Hamman's syndrome, 9 and spontaneous pneumopericardium are defined as the presence of gas in the mediastinal structures and in the pericardial cavity without an identifiable cause, respectively. Various cases are published, which link different sports activities with development of SPM. In fact, a recent review by Morgan et al. reported history of physical exertion in 21% of patients. 4 However, the role of treatment options and recommendations regarding return to activity for athletes remain uncertain.

Demonstrated by Macklin in animal studies, ¹⁰ the underling pathophysiology for spontaneous pneumomediastinum and pneumopericardium is an abruptly increasing pressure gradient between the intra-alveolar and interstitial space. As a consequence, preexisting air leaks

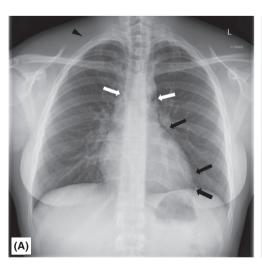
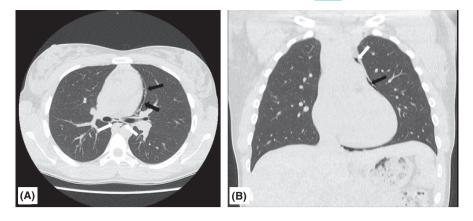




FIGURE 1 (A) Anterior–posterior and (B) lateral chest radiograph at first presentation demonstrating pneumomediastinum (white arrows), pneumopericardium (black arrows), and subcutaneous emphysema (arrowhead)

FIGURE 2 (A) Transversal and (B) coronal image of chest CT at second presentation demonstrating pneumomediastinum (white arrows) and pneumopericardium (black arrows)



are enhanced or terminal alveoli rupture occurs with consecutive air dissection along the pulmonary vasculature toward the hilum and into the structures of the mediastinum. Depending on local pressure conditions and anatomical situation, gas extension may occur along the deep cervical fascia into the neck (subcutaneous emphysema) or along blood vessel sheaths into the pericardium (pneumopericardium). Spontaneous pneumopericardium requires much higher pressure gradients than SPM and occurs less frequently. Further, development of pneumothorax is possible when mediastinal pressure suddenly rises. Causes for initial increase in intra-alveolar pressure are alveolar obstruction (e.g., by asthma, foreign body), excessive coughing (e.g., in respiratory infection), emesis, or Valsalva maneuver (e.g., during delivery or sports activity). In addition, smoking is a common history in patients with SPM.4,11

Bike training most likely caused recurrent spontaneous pneumomediastinum and pneumopericardium in our patient. To the best of our knowledge, no report described this trigger so far. We hypothesize that excessive cycling could be a risk for patients, prone to develop SPM. Intrathoracical pressure gradients may rise by both intense breathing activity and forward bent position of the upper body impairing lung expansion. In addition, cycling with high speed on a racing bike causes relevant air drag which may further impede sufficient expiration and may contribute to the pathomechanism.

Even if the majority of SPM presumably remains undiagnosed, athletes may present with chest, neck or throat pain, cough, dyspnea, dysphagia, or dysphonia. Physical examination may reveal crepitus or respiratory distress and ultimately signs of pericardial tamponade. Mild fever and leukocytosis may be detected as well.^{4,11}

To rule out life-threatening differential diagnoses such as tension pneumothorax, mediastinitis, esophageal perforation, or tracheal avulsion, diagnostic methods may contain chest radiograph, chest CT, bronchoscopy, esophagogastroduodenoscopy, and esophagram.

Questions remain regarding optimal therapeutic management for athletes. There is growing body of evidence that spontaneous pneumomediastinum and pneumopericardium are self-limiting and can be successfully treated by conservative management. A short period of in-hospital observation to perform diagnostic workup, confirm cardiopulmonary stability, and treat pain or nausea seems reasonable. Because up to ten percent of patients with SPM present with concurrent pneumothoraces, further interventions may be necessary in these individuals. When pneumothorax can be ruled out and symptoms recede, we assume that no additional testing is indicated to return to sports activity.

Athletes and coaches should be educated that the chance of SPM recurrence is approximately 1%. ⁴ Careful monitoring of symptoms such as chest pain and dyspnea after sports activity is vital to detect a possible recurrence.

This case report has limitations. We describe two episodes of SPM in one single person after cycling on a racing bike, which makes it difficult to draw general conclusions about clinical management in these patients. Our patient showed mild symptoms overall. Diagnostic workup and therapeutic options may contain more invasive approaches, if more serious symptoms are present.

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CONFLICT OF INTEREST

None.

AUTHOR CONTRIBUTIONS

AH, MH, and JH conceived the study and wrote the manuscript. All authors critically revised the manuscript.

ETHICAL APPROVAL

None.

CONSENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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