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Case Report

Spontaneous Pneumomediastinum with Concurrent Pneumopericardium in a Young Woman with Asthma: Clinical-Radiological Correlation and Conservative Outcome

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Abstract: Spontaneous pneumomediastinum (SPM) is a rare and usually benign condition characterized by the presence of free air in the mediastinum without an evident traumatic or iatrogenic cause. Although classically described in young men, it may occur in women and in association with multiple precipitating factors. We report the case of a 32-year-old woman with poorly controlled asthma who presented with acute dyspnea and chest pain. Chest computed tomography confirmed extensive pneumomediastinum associated with pneumopericardium, without evidence of esophageal rupture or pneumothorax. Additional potential triggers included recent intensive weight training involving Valsalva maneuvers and the use of veterinary-origin anabolic steroids, suggesting a multifactorial mechanism of alveolar rupture. Transthoracic echocardiography excluded cardiac tamponade. The patient was managed conservatively with symptomatic support and optimization of asthma therapy, with favorable clinical and radiological evolution. This case highlights the importance of recognizing SPM and pneumopericardium in the differential diagnosis of acute chest symptoms, as well as the role of combined mechanical and inflammatory factors in its pathophysiology, reinforcing that conservative management is safe and effective in clinically stable patients.

Keywords: Chest pain; Dyspnea; Cough; Pneumomediastinum; Pneumopericardium.

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1. Introduction

Spontaneous pneumomediastinum (SPM), also known as Hamman's syndrome [1–4], is a rare condition defined by the presence of air in the mediastinal interstitium without a traumatic or iatrogenic cause. Its incidence is low, with a higher frequency in young adult males [1–2, 5–8]. A 22-year systematic review published in 2014 reported an incidence of 1 per 44,500 hospital admissions [6], a value like that reported in 2008, ranging from 1/7,000 to 1/45,000 depending on the population studied [7]. Predisposing factors include asthma exacerbations, intense physical exertion, and episodes of vigorous coughing or vomiting that increase intrathoracic pressure.

The pathophysiology of SPM is explained by the Macklin effect, described in 1944, in which increased intra-alveolar pressure leads to small alveolar ruptures, allowing air to track along the bronchovascular interstitium toward the mediastinum [1–4, 6]. Computed

tomography (CT) demonstrates air dissecting the bronchovascular sheaths, a typical finding of the Macklin effect. Chest radiography is often the initial imaging test; however, CT is the most sensitive method for confirming the diagnosis and excluding secondary causes [2–4, 7–11]. Most patients have a favorable course with conservative treatment based on rest, analgesia, and oxygen therapy, without the need for invasive intervention [2–3, 7–11]. Severe complications such as pneumothorax or cardiac tamponade are rare. The condition is self-limiting, with clinical and radiological resolution occurring within a few days [1–12].

SPM and pneumopericardium are rare conditions with low incidence and variable clinical presentation. Although classically described in young men, cases in women and in patients with asthma have been increasingly reported [1–2, 5–8]. The simultaneous association of both entities is uncommon and may be easily mistaken for severe thoracic emergencies, such as pneumothorax or acute coronary syndrome. In this context, the present study aims to report the case of a young patient with asthma who developed spontaneous pneumomediastinum and pneumopericardium, highlighting the clinical and radiological characteristics of the condition, the criteria guiding management, and the observed clinical course, in correlation with findings reported in current literature.

2. Case Report

A 32-year-old woman, residing in the suburban area of the city and employed at a swimming club, denied smoking and alcohol consumption. She had a history of bronchial asthma with irregular adherence to treatment, using inhaled formoterol and budesonide on demand, as well as a prior report of COPD, which is atypical given her age and non-smoking status and may represent a misclassification of chronic asthma rather than true chronic obstructive pulmonary disease. She practiced weight training and reported the use of a veterinary-origin anabolic steroid during hypertrophy training, although she could not recall the name of the substance or the exact date of the last dose. She also reported at least two hospitalizations in the past year due to pneumonia.

On 04/16/2024, she was admitted to a public emergency care unit with complaints of sudden nocturnal awakening accompanied by intense dyspnea, retrosternal chest pain, and dry cough, without fever. On admission, her blood pressure (BP) was 120/80 mmHg, heart rate 75 beats per minute (bpm), respiratory rate 20 breaths per minute (rpm), peripheral oxygen saturation (SpO_2) 93% on room air (RA), and axillary temperature 36.5°C. She was diagnosed with severe asthma exacerbation and started treatment with bronchodilators and systemic corticosteroids. The patient had a recent spirometry test (04/13/2024) demonstrating mild obstructive ventilatory disorder, with a positive bronchodilator response and no symptoms during or after the examination. On 04/18/2024, she was transferred to Ronaldo Gazolla Municipal Hospital (HMRG), with persistent mild dyspnea and ventilatory-dependent chest pain, exacerbated by coughing. She also reported an intense holocranial headache of a previously unreported pattern.

On physical examination, there were no noteworthy findings on general inspection or neurological examination. Cardiac auscultation revealed no murmurs or crackles, and Hamman's sign was not identified. Pulmonary auscultation revealed scattered areas of intense wheezing and diffuse crackles. The abdomen was unremarkable, and the lower limbs showed no circulatory abnormalities. Vital signs were as follows: BP 130/98 mmHg, HR 75 bpm, RR 20 rpm, temperature 36.2°C, and SpO_2 93% on room air.

Considering the initial hypothesis of decompensated asthma, treatment was initiated with nil per os, beclomethasone 200 mcg every 12 hours, prednisone 40 mg/day for 4 days, salbutamol 400 mcg on demand (minimum interval of 6 hours), in addition to inhalation therapy with ipratropium 0.25 mg/mL (40 drops) via nebulization every 8 hours. Comfort measures were prescribed, including analgesia with dipyrone 1 g every 4 hours and oxygen therapy as needed. A chest radiograph was requested but not performed. During the subsequent routine evaluation, chest and brain computed tomography (CT) scans were requested.

Laboratory tests performed on 04/19/2024 revealed hemoglobin 12.9 g/dL, leukocytes 9,690/mm³, platelets 293,000/mm³, urea 32 mg/dL, creatinine 1.06 mg/dL, C-reactive protein 9.2 mg/L, sodium 136 mEq/L, and potassium 3.56 mEq/L. Chest CT demonstrated extensive pneumomediastinum (Figures 1A and 1B) and pneumopericardium (Figures 2A and 2B), as well as subcutaneous emphysema in the left cervical region (Figures 3A and 3B) and discrete ground-glass opacities in the right upper lobe. No esophageal rupture, lobar atelectasis, or free air between the visceral and parietal pleurae was observed, ruling out secondary pneumothorax, Boerhaave syndrome, and pulmonary thromboembolism. Brain CT showed no significant abnormalities.

Figure 1. Axial chest computed tomography images demonstrating the presence of free air in the middle (A) and upper mediastinum (B), outlining vascular and tracheal structures, consistent with extensive pneumomediastinum. There is no evidence of pneumothorax or associated fluid collections.

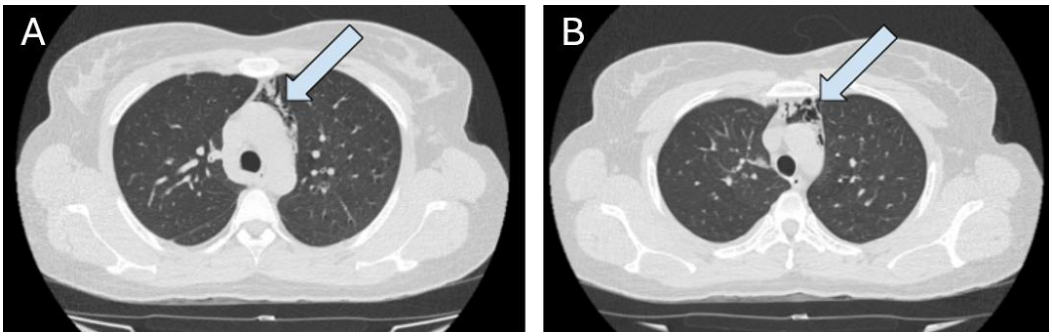


Figure 2. Computed tomography with axial slices reveals air confined within the pericardial space (A), appearing as a radiolucent halo surrounding the heart, and absence of pericardial effusion or signs of cardiac compression (B).

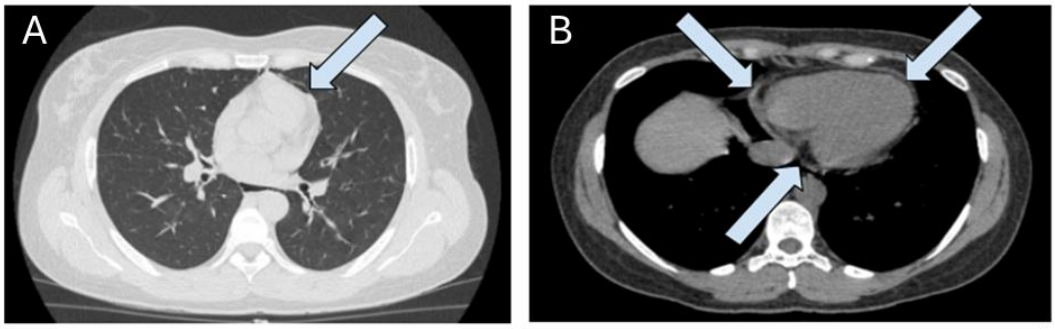
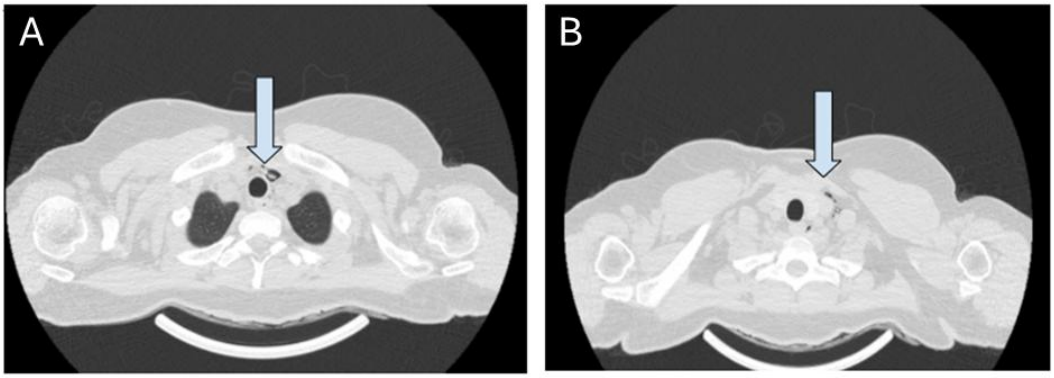


Figure 3. Axial computed tomography images demonstrating left cervical subcutaneous emphysema (A), with air dissemination through the superficial muscular planes and subcutaneous tissue (B). This finding is associated with spontaneous pneumomediastinum, with no signs of tracheal or esophageal rupture.



As a complementary examination, a transthoracic echocardiogram was performed to assess function and exclude cardiac tamponade. The study revealed a left ventricular ejection fraction of 70% according to Teichholz criteria, with normal chamber diameters and wall thicknesses, preserved left ventricular systolic function at rest, and no segmental wall motion abnormalities. The right ventricle was normokinetic, left ventricular diastolic function was normal, and the cardiac valves showed preserved morphology and mobility. The inferior vena cava was normally distended, with adequate respiratory variation.

After discussion with the pulmonology team, conservative management was maintained. Oral diet was reintroduced, and the inhaled corticosteroid dose was optimized to 200 mcg every 8 hours. Systemic prednisone was gradually tapered to 25 mg/day, with planned discontinuation within 48 hours, as it had been initiated for the management of asthma. As the patient remained clinically stable, clinical observation continued for six days. Follow-up CT performed on 04/24/2024 demonstrated regression of mediastinal air (Figure 4A) and pericardial air (Figure 4B), as well as complete resolution of subcutaneous emphysema (Figures 5A and 5B). On physical examination, wheezing was no longer present, and the patient remained eupneic, without respiratory effort or cough, although peripheral oxygen saturation fluctuated between 91% and 93% on room air.

Figure 4. Axial chest computed tomography images from follow-up examination showing regression of mediastinal and pericardial air, with restoration of the anatomical planes.

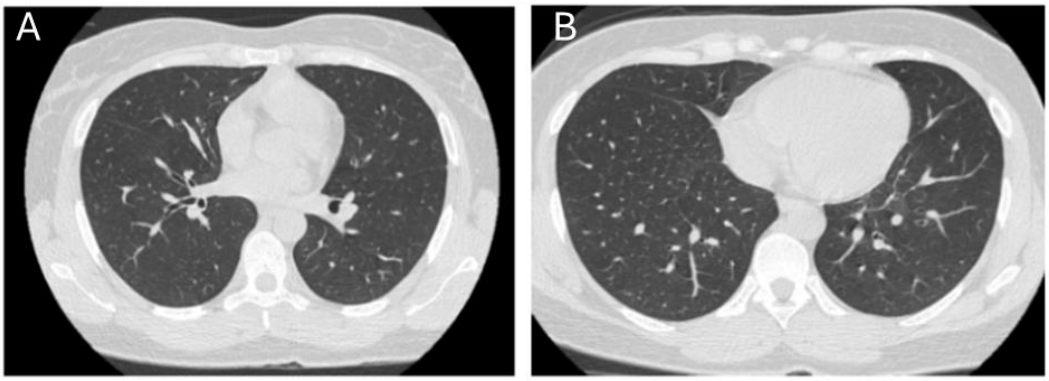
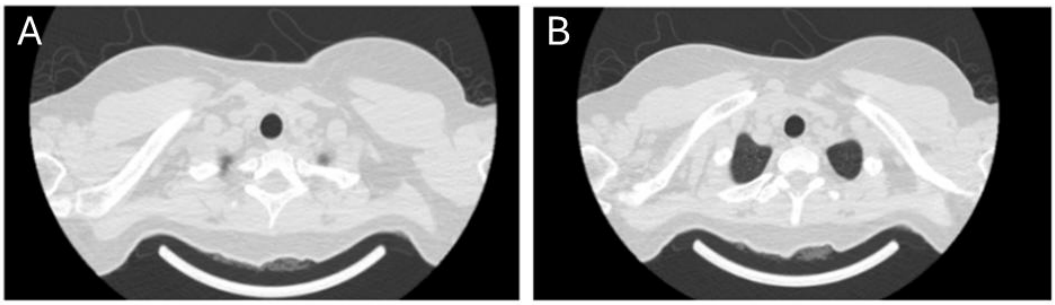


Figure 5. Comparative follow-up computed tomography images demonstrating complete regression of the previously identified subcutaneous emphysema, with no recurrence of air in the cervical soft tissues.



Serological tests for HIV, hepatitis B and C, and syphilis (VDRL) were negative. In view of the clinical and radiological improvement, hospital discharge was recommended on 04/25/2024, with prescription of beclomethasone 200 mcg (two inhalations every 12 hours, continuous use), salbutamol 100 mcg (four inhalations as needed, with a minimum interval of 6 hours), and an oral prednisone tapering regimen starting at 20 mg, with dose reduction of 5 mg every five days until discontinuation. The patient was referred to as a shared follow-up at the Family Health Clinic and scheduled to return to the Internal Medicine outpatient clinic at HMRG after 30 days.

3. Discussion

PM is a rare clinical entity, predominantly described in young men [4, 5, 12–15, 19, 20], but it should also be considered in the differential diagnosis of dyspnea and chest pain in young women without comorbidities [7]. The Macklin effect, illustrated in Figure 11, represents the classic pathophysiological mechanism of SPM, characterized by the dissection of air along the bronchovascular planes toward the mediastinum [4,5,8,13]. In this report, the image is presented as a didactic illustration, as the typical radiological pattern was not clearly identifiable in this patient. This process may extend to the pericardial, retropharyngeal, and retroperitoneal spaces [19] and can also be observed in secondary pneumomediastinum of various causes [20]; therefore, the diagnosis of SPM cannot rely solely on the presence of this imaging finding.

Asthma is frequently cited as a precipitating factor; however, a systematic review evaluating 1,771 articles published between 1990 and 2012, including 27 studies with 600 patients, found that asthma was present in only 9% of cases, being surpassed by unidentified factors (34%), intense physical activity (14%), drug abuse (14%), and vigorous coughing (10%) [13]. Other recognized triggers include upper airway infections, pneumonia, asthma exacerbations, persistent cough, vomiting, forced defecation, spirometry, use of inhaled drugs such as cocaine and methamphetamines, vigorous exercise, and the Valsalva maneuver, all of which are associated with abrupt increases in intrathoracic pressure [3–6, 8, 10, 12, 14, 19].

In the present case, additional triggers should be considered beyond asthma alone. The patient reported recent engagement in intensive weight training, involving repeated Valsalva maneuvers capable of significantly increasing intra-alveolar pressure [3–6,8,10,12,14,19]. Moreover, the use of veterinary-origin anabolic steroids may exert pro-inflammatory and structurally deleterious effects on pulmonary tissue, potentially increasing susceptibility to injury. It is therefore plausible that mechanical stress from exertion, combined with underlying airway inflammation and possible drug-related tissue vulnerability, acted synergistically as the primary driver of alveolar rupture and subsequent air dissection.

The presence of ground-glass opacities in the right upper lobe was interpreted as a nonspecific finding, possibly related to recent inflammatory or infectious processes. Given the patient's history of recurrent pneumonia, it is plausible that an underlying structural or inflammatory vulnerability may have contributed to alveolar fragility, acting as a locus minoris resistentiae for rupture.

The classic triad of SPM [17, 18] — chest pain, dyspnea, and subcutaneous emphysema—is well described in the literature, although it may not present in its complete form. Additional, less frequent symptoms include intense cough [4–6, 12–14, 17–20], odynophagia [4, 5, 12, 13, 19, 20], wheezing [4, 5, 16], dysphonia [4, 12, 19, 20], tachycardia [1, 4, 17], and fever, with hypoxemia being uncommon even in cases in which the patient presents with dyspnea [19]. Hamman's sign, although considered classic, is infrequent [5, 8, 10, 17, 20]. Among the main differential diagnoses to be excluded are Boerhaave syndrome, characterized by spontaneous esophageal perforation [4, 5, 11–15], often accompanied by left-sided unilateral pleural effusion, primary pneumothorax, acute coronary syndrome, pericarditis, isolated asthma exacerbation, pulmonary embolism, and pneumoperitoneum [4, 5, 19]. Severity markers include the presence of pneumoperitoneum, extensive pneumothorax, and hemodynamic instability.

In this patient, the presence of diffuse wheezing on auscultation, prior spirometry demonstrating reversible airflow obstruction, and clinical improvement following bronchodilator therapy supported the diagnosis of true bronchospasm. However, it is important to acknowledge that symptoms related to mediastinal air may overlap with asthma manifestations, and a component of mechanical airway compression cannot be entirely excluded.

The association between pneumomediastinum and pneumopericardium is uncommon but well documented [1–5, 7, 9, 13, 14, 16]. Echocardiography is the imaging modality

of choice for evaluating pericardial air, although it may miss small volumes [9], and remains an important tool for excluding cardiac tamponade and assessing hemodynamic repercussions [1, 5, 9]. Although tamponade is a potential and severe complication, none of the reported cases of SPM associated with pneumopericardium have progressed to this outcome, suggesting it remains a theoretical possibility. In the present case, the radiolucent halo observed on CT was consistent with air within the pericardial space, without signs of cardiac compression. A follow-up echocardiogram was not performed, as the patient remained clinically stable and follow-up CT demonstrated resolution of pericardial air; however, serial echocardiographic evaluation could have provided additional confirmation of sustained hemodynamic stability.

From a diagnostic standpoint, chest radiography is traditionally considered the initial examination of choice due to its wide availability and rapid performance. However, computed tomography is the most sensitive modality, allowing for a precise assessment of the extent of involvement, identification of the Macklin effect, and exclusion of complications such as pleural effusion, pneumothorax, or esophageal perforation, in addition to improved detection of cardiac involvement [4,5,8,14]. In the present case, although chest radiography was requested, it was not performed due to logistical constraints, and CT was prioritized given the persistence of symptoms and the need for a more comprehensive evaluation of differential diagnoses.

Treatment is predominantly conservative and symptomatic, based on rest, analgesia, and oxygen therapy [4–6, 12, 17, 18, 19]. Oxygen therapy may relieve dyspnea and has been proposed to accelerate reabsorption of mediastinal air by increasing the nitrogen diffusion gradient [3, 4, 20]; however, there is limited evidence supporting this effect in spontaneous pneumomediastinum, unlike in pneumothorax [19]. In this context, its use should be interpreted primarily as supportive rather than disease-modifying therapy. In the presence of identifiable precipitating factors, specific treatment should be added, such as cough suppressants [4] and bronchodilators and systemic corticosteroids during asthma exacerbations [16, 20].

There are also reports of prophylactic antibiotic use to prevent mediastinitis [3, 17]; however, this approach is not routinely recommended [5, 11, 19] and should be reserved for situations with an increased risk of respiratory tract infection [6, 12, 16–17]. The prolonged prednisone taper prescribed at discharge was guided by the management of asthma rather than the pneumomediastinum itself. Nevertheless, extended corticosteroid use carries potential risks, particularly in a patient with prior anabolic steroid exposure, and this aspect should be carefully considered in clinical decision-making.

The prognosis of SPM is generally favorable, with clinical regression observed within two to five days [12, 14, 18], radiological resolution within up to seven days [3, 6, 17], and a mean hospital stay ranging from two to ten days [1, 3–6, 11–15, 18]. Recurrences are rare, and long-term follow-up is not required [11–15, 19]. These findings reinforce that conservative management is safe and effective in the absence of complications or significant clinical instability.

This report has some limitations that should be acknowledged. Alpha-1 antitrypsin deficiency was not investigated, despite the atypical history suggesting possible underlying susceptibility. Serial echocardiographic follow-up was not performed, which could have provided additional confirmation of sustained cardiac stability after resolution of pneumopericardium. Additionally, the ground-glass opacities identified on CT were not further investigated, limiting the ability to fully exclude an underlying infectious or inflammatory process contributing to alveolar fragility.

4. Conclusion and future perspectives

This case reinforces the importance of recognizing spontaneous pneumomediastinum and pneumopericardium as differential diagnoses of acute chest pain and dyspnea, even in young women without classic risk profiles. A comprehensive evaluation integrating clinical findings and imaging allowed exclusion of life-threatening conditions and

supported a conservative approach, which proved safe and effective. Notably, the coexistence of asthma, intensive physical exertion with Valsalva maneuvers, and the use of anabolic steroids suggests a multifactorial pathophysiological mechanism that should be actively investigated in similar cases.

Future studies are needed to better define the role of combined mechanical and inflammatory factors in the development of SPM, as well as to establish clearer indications for imaging strategies and follow-up protocols. Additionally, further investigation into the impact of exogenous substances, including non-prescribed anabolic agents, may help clarify their contribution to pulmonary vulnerability and guide preventive strategies.

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Research Ethics Committee Approval: This manuscript complies with CNS Resolution 466/12 and related regulations. The protocol was approved by the CEP/SMS-RJ, opinion 7.816.274, CAAE 91082425.9.0000.5279, on September 3, 2025. All data were anonymized in accordance with the LGPD (Brazilian General Data Protection Law). The patient read and signed the Informed Consent Form and authorized the publication of clinical information and images. There were no interventions beyond the usual care. Any amendments will be submitted to the CEP beforehand.

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Conflicts of Interest: All other authors declare no conflicts of interest.

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