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Case Report

Pneumomediastinum associated to asthma exacerbation in adults: Two case reports and bibliographic review

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ABSTRACT

Introduction: Pneumomediastinum (PM) is defined as the presence of air in the mediastinal cavity. It has been initially described by Laennec in 1819 as secondary to trauma. Among the non-traumatic causes, asthma exacerbations are included. The following report depicts two PM cases in the context of an asthma exacerbation. **Case 1**: 18-year-old male, diagnosed with asthma since childhood. The patient presented with an asthma exacerbation, associated with subcutaneous emphysema and PM as a complication. **Case 2**: 37-year-old male, asthmatic athlete. He presented with an asthma exacerbation, associated with Salmonella bacteremia and PM as a tomographic finding. **Conclusion**: The real incidence of PM associated with asthma attacks is currently unknown. It is generally an asymptomatic and self-limited entity, but it must be suspected in patients presenting with dyspnea, subcutaneous emphysema and hypoxemia that does not improve with oxygen therapy. Controlling asthma exacerbation and monitoring the patient are the main bases of the treatment.

Keywords: Pneumomediastinum, asthma, air leak syndromes, subcutaneous emphysema, hypoxemia, chest Computed Tomography scan, Macklin effect.

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Abbreviations: PM, Pneumomediastinum; **ALS,** air-leak syndromes; **CT,** computed tomography; **mMRC,** Modified Medical Research Council dyspnea scale; **FiO_2,** fraction of inspired oxygen, **mcg,** micrograms.

INTRODUCTION

Pneumomediastinum (PM), also known as mediastinal emphysema, is defined as the presence of air in the mediastin. It is frequently an underdiagnosed entity, whose incidence in asthmatic patients varies in the literature from 0.3% in pediatrics to 1% (Stack and Caputo, 1996) in adults (Vianello et al., 2018). It is caused by an air leak from the airway, or to a lesser extent from the digestive tract, towards the thoracic cavity, and it is encompassed within the spectrum of *air leak syndromes* (ALS). These are possible complications in asthma exacerbations, with PM being the most widely recognized (Takeishi et al., 1989). The objective of the following report is to present two cases of PM associated with exacerbation asthma, highlighting the clinical features, diagnosis and evolution of both patients.

Case 1

An 18-year-old male patient, a non-smoker, consulted the emergency service for a 7-day history of non-productive cough and dyspnea, progressing to mMRC III/IV in the last 48 hours. As relevant medical history, he referred being diagnosed with asthma when he was 11 years old, had no previous hospitalizations, and is in self-treatment with one (1) canister of inhaled salbutamol as needed, 100 mcg/dose per month (total doses = 200). He denied the use of corticosteroids and declared that he had not received any follow-up by the pulmonology service since his diagnosis.

The patient was admitted to another center for severe asthma exacerbation with hypoxemia 48 hs previus. In

the other hospital, a chest computed tomography (CT) scan was performed (Figure 1), which confirmed the presence of pneumomediastinum extending to the base of the neck, associated with subcutaneous emphysema in the anterior chest wall.

Systemic and inhaled corticosteroids combined with bronchodilators were indicated as treatment. He evolved with partial clinical improvement.

The patient was referred to our center. At the initial examination, he was lucid, afebrile, and normotensive, with a heart rate of 100 beats/minute. He was tachypneic (30 breaths/minute) and with saturation (SpO2) of 90% at FiO_2 21%. On physical assessment, diminished bilateral air entry was auscultated, with generalized wheezing, predominantly during expiration. Subcutaneous emphysema was evident with bilateral palpation of the upper pectoral, supraclavicular and cervical regions.

The initial laboratory test results showed leukocytosis of 20520/mm_3 with no other particularities. Arterial blood gases (ABG) test was performed on room air, which showed hypoxemia with respiratory failure (PO_2 55 mmHg), normocapnia (PCO_2 43 mmHg), pH 7.40; HCO_3 26mEq/L; base excess 0.8 and lactate (2.4 mmol).

Oxygen therapy was administered at a flow rate of 2 L/min, achieving a SpO_2 level of 96%, combined with bronchodilators, inhaled and systemic corticosteroids, according to the institutional protocol for asthma attacks. Hospitalization was decided to monitor the patient due to the pneumomediastinum. He evolved with a frank improvement of the oximetry and his clinical features. The patient was discharged after 5 days, followed by controls done by the pulmonology service in the outpatient clinic.

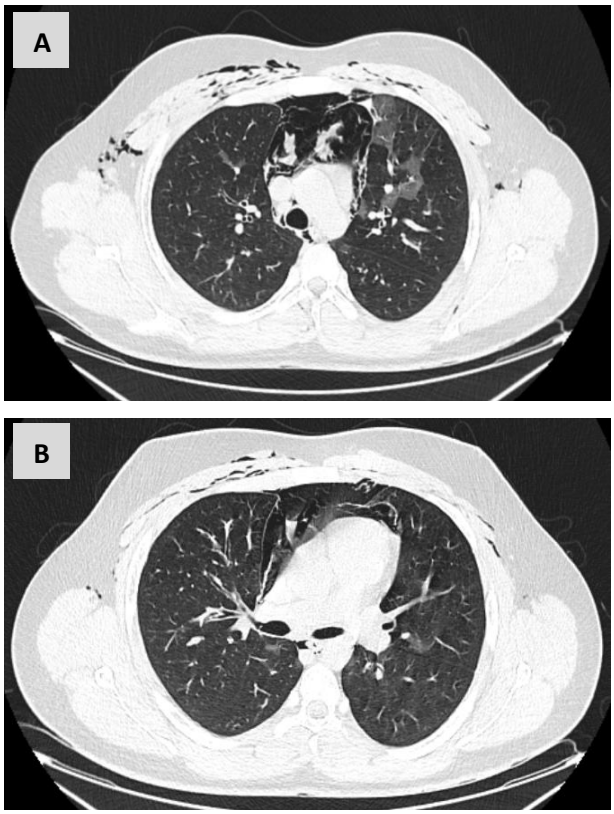

Figure 1. Chest computed tomography (CT) scan. It is observed in **A**: pneumomediastinum associated with subcutaneous emphysema, which compromises the anterior chest wall and dissects muscular planes. Centriacinar opacities with ground glass attenuation and septal thickening, predominantly in both upper lobes. **B**: Scarce continuous air in the minor fissure, which could be due to the Macklin Effect.

Case 2

A 37-year-old male, high-performance athlete. He is an asthmatic patient under treatment with two (2) daily doses of inhaled budesonide/formoterol 160 mcg/4.5 mcg; fully vaccinated against COVID-19. He consulted the emergency service of our center due to a 5-day history of headache and fever of 38.6°C, associated with asthenia and dyspnea mMRC I/II that appeared in the last 24 hours. As relevant background, he mentioned that he went mountain running at an altitude of 2000 meters at the beginning of the month, and at the time of the consultation, he presented as well with a 20-day history of diarrhea and vomiting. Due to the fever and dyspnea, a nasopharyngeal PCR test for COVID-19 was performed, along with a chest CT. The PCR results were not detectable, but the CT certified the presence of pneumomediastinum (Figure 2). Hospitalization was decided based on the CT finding and the symptoms. Two blood culture samples were later requested.

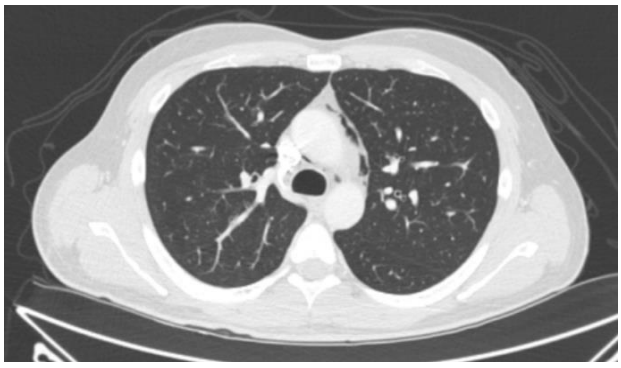

Figure 2. Chest computed tomography. Pneumomediastinum bubbles are observed. There is no evidence of pleuro-parenchymal alterations or enlarged lymph nodes.

On admission, the patient was normotensive, tachycardic (110 beats/minute), and had a temperature of

37°C and a SpO_2 level of 97% on room air. During the physical examination, normal respiratory mechanics were observed and lung sounds were clear in all lobes bilaterally; no added sounds were auscultated. The abdomen was soft to the touch in all quadrants, with no masses or pain. Venous blood samples were taken for routine laboratory tests, but the obtained results showed no significant findings. Salmonella ssp. was isolated in the blood cultures (positive results 2/2), so a 7-day treatment with intravenous ceftriaxone was started due to antibiotic sensitivity. In addition, oxygen therapy and budesonide/formoterol were indicated according to the asthma treatment.

The case was interpreted as PM secondary to an asthma exacerbation, combined with Salmonella bacteremia in the context of traveler's diarrhea. However, vomiting or altitude could not be ruled out as possible causes of PM.

Seven days later, a new chest CT was performed for control no mediastinal ni pleuro-pulmonary alterations were observed. Except for the initial dyspnea, he had no other asthma symptoms during hospitalization. He presented a satisfactory evolution of symptoms, thus hospital discharge was indicated. He is currently being followed up by the pulmonology team.

DISCUSSION

Two cases of PM secondary to asthma exacerbation are presented. The first PM reports date back to 1819 by Laennec, who described this condition as secondary to trauma. Since then, various classifications have been postulated, with the division into *primary* (spontaneous, without apparent cause) or *secondary* PM being the most currently accepted. In turn, the latter is subdivided into traumatic or non-traumatic. Frequent non-traumatic causes include the use of mechanical ventilation (iatrogenic NM), or the presence of underlying lung diseases, such as asthma.

Asthma exacerbations can cause a complex clinical picture; in certain cases, patients present with dyspnea and hypoxemia as a consequence of ALS (Gomella, 2004). The pathophysiological process of this entity is explained by alveolar overinflation followed by alveolar rupture, due to the airflow obstruction typically seen in asthma. Coughing and other unintentional Valsalva maneuvers, such as force exerted during vomiting or defecation, worsen the condition by indirectly increasing intrapulmonary pressure.

The Macklin Effect (Wintermark and Schnyder, 2001) describes the cascade of events that lead to the development of PM: initially, the rupture of the marginal alveoli causes an air leak into the interstitium, also known as *pulmonary interstitial emphysema*. From this site, the air flows contiguously to the bronchovascular tree, through the perivascular and peribronchial sheaths, reaching the hilum and finally the mediastinal cavity. The latter communicates directly with the retropharyngeal and submandibular spaces, so finding *subcutaneous emphysema* in the cervical region should raise the suspicion that the patient has an associated PM (Ochoa-Ariza et al., 2020). In turn, the pressure exerted by free air in the cavity can lead to rupture of the mediastinal pleura and invasion of air into the pleural space, causing a *pneumothorax*. On the other hand, from the mediastinum, the air dissects the muscular and adipose layers, which can result in the appearance of a *pneumopericardium* or *pneumorrachis*. The downward extension of air at the periaortic or periesophageal level can cause a *pneumoperitoneum* or *pneumoretroperitoneum*. All the air leaks mentioned here are known as air-leak syndromes (ALS), with PM being one of the most frequently seen.

In this way, asthma exacerbation may contribute to the genesis of PM. Case 1 exposes a typical exacerbation of patients with poor control of their underlying disease. The absence of anti-inflammatory therapy was the triggering factor for the onset of the crisis, which later caused the PM (due to the pathophysiological process previously described). On the other hand, Case 2 describes a patient with good control of his asthma, whose exacerbation is probably related to mountain sports and being suddenly exposed to cold temperatures. It is also postulated that altitude could have contributed to the development of the PM. Although the association is not yet clear, there have been reports of non-asthmatic patients with no predisposing factors who develop spontaneous PM at high altitudes (Kalafat, et al., 2018). Finally, it must also be taken into account that the patient presented with a medical history of vomiting and diarrhea: these cannot be ruled out as other potential causes or aggravators of the PM.

For both patients, the diagnosis of PM was reached with a chest CT image, which is considered the gold standard. Chest X-rays are useful for general medical assessment but to a lesser extent, since pulmonary interstitial emphysema, as well as about a third of PM cases, are not always evident in these images (Arcos, 1966; Caceres et al., 2008).

Patients with PM are frequently asymptomatic; however, certain findings in the medical background or physical examination point to the diagnosis of PM: dyspnea or chest pain of varying intensity, hypoxemia refractory to oxygen treatment, or palpation of subcutaneous emphysema as seen in Case 1. Dysphagia and dysphonia have been reported in patients who, due to compression of mediastinal structures, suffer a displacement of the esophagus and trachea, respectively.

The pathognomonic sign which could identify patients with PM is *Hamman's sign* (Álvarez et al., 2009). That is the auscultation on the precordial area of crepitus

concomitant with cardiac systole. It is usually assessed with the patient placed in the left lateral decubitus position or sitting. This sign occurs as a consequence of the accumulation of air between the pericardium and the anterior chest wall.

Regarding the management of PM, it will depend on the clinical status of each patient, but they usually resolve spontaneously. Treatment is based on the immediate correction of the underlying cause, oxygen therapy, rest, and hospital monitoring. The latter is necessary to prevent or anticipate the appearance of complications associated with PM, such as *pneumothorax*, *pneumopericardium* or *tension PM* that would compromise blood flow. Barotrauma should be taken into consideration as a possible complication in patients with an indication of mechanical ventilation. Analgesics can be administered if required, and both Valsalva maneuvers and risk factors for the development of PM after hospital discharge should be avoided. In this sense, education and follow-up of asthmatic patients are essential to prevent asthma attacks and eventually PM or other ALS.

CONCLUSIONS

Currently there is no consensus to determine the real incidence of PM associated with asthma exacerbation since only isolated cases have been reported in the medical literature. The absence of specific signs and symptoms is likely to make it an underdiagnosed and underestimated entity.

Although its course is generally benign and self-limited, a few severe cases (Karakaya et al., 2012) have been described. Clinical suspicion of PM is essential while evaluating asthma exacerbations. It must be taken into account for a prompt diagnosis since a PM in the context of an asthma crisis is considered an indication for the hospital admission. These patients should be closely monitored to anticipate the development of other ALS and serious complications.

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