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REVIEW ARTICLE

Infectious Disease

Pneumomediastinum in COVID-19 disease: Clinical review with emphasis on emergency management

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Abstract

Pneumomediastinum can be primary (spontaneous) or secondary to iatrogenic, traumatic, and non-traumatic causes. The incidence of spontaneous and secondary pneumomediastinum is higher in patients with coronavirus disease 2019 (COVID-19) compared to the general population. So, pneumomediastinum should be considered in the differential diagnosis of any patient with COVID-19 presenting with chest pain and breathlessness. A high level of suspicion is required to diagnose this condition promptly. Unlike in other disease conditions, pneumomediastinum in COVID-19 has a complicated course with higher mortality in intubated patients. No guidelines exist for managing pneumomediastinum patients with COVID-19. Therefore, emergency physicians should be aware of the various treatment modalities besides conservative management for pneumomediastinum and life-saving interventions for tension pneumomediastinum.

1 | INTRODUCTION

The World Health Organization announced the coronavirus disease 2019 (COVID-19) pandemic on March 11, 2020.[1] COVID-19 is caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2).[2] The most common symptoms of this disease are fever and cough; however, a wide range of symptoms have been reported.[3] Pneumomediastinum is a well-described, life-threatening complication related to the use of mechanical ventilation, especially in patients with acute respiratory distress syndrome (ARDS).[4–6] The incidence of pneumomediastinum in patients with COVID-19 is higher (1:5498) than in the general population (1:7000 to 1:45,000).[7,8] Patients with pneumomediastinum had evidence of increased disease severity at admission.[9] A meta-analysis by Shrestha et al. in 2022 revealed a linear relationship between the incidence of pneumomediastinum and disease severity.[10] Pneumomediastinum in COVID-19 has a complicated course with higher mortality in intubated patients, unlike other conditions in which it is usually benign and self-limiting.[11] A retrospective analysis by Kangas-Dick et al. reported a higher prevalence of pneumomediastinum and mortality in patients with COVID-19 compared to the historical patients with ARDS.[12]

Spontaneous pneumomediastinum (SPM) is an uncommon complication of viral pneumonia, and SARS-CoV-2 is a new addition to the etiology of SPM. The UK POETIC survey reports the incidence of SPM in individuals with COVID-19 as 0.13%, which is nearly 6000 times higher than in the general population (0.00002%).[13,14]

2 | PATHOPHYSIOLOGY

Pneumomediastinum is classified into primary (spontaneous) and secondary, with the latter being subdivided into iatrogenic, traumatic,

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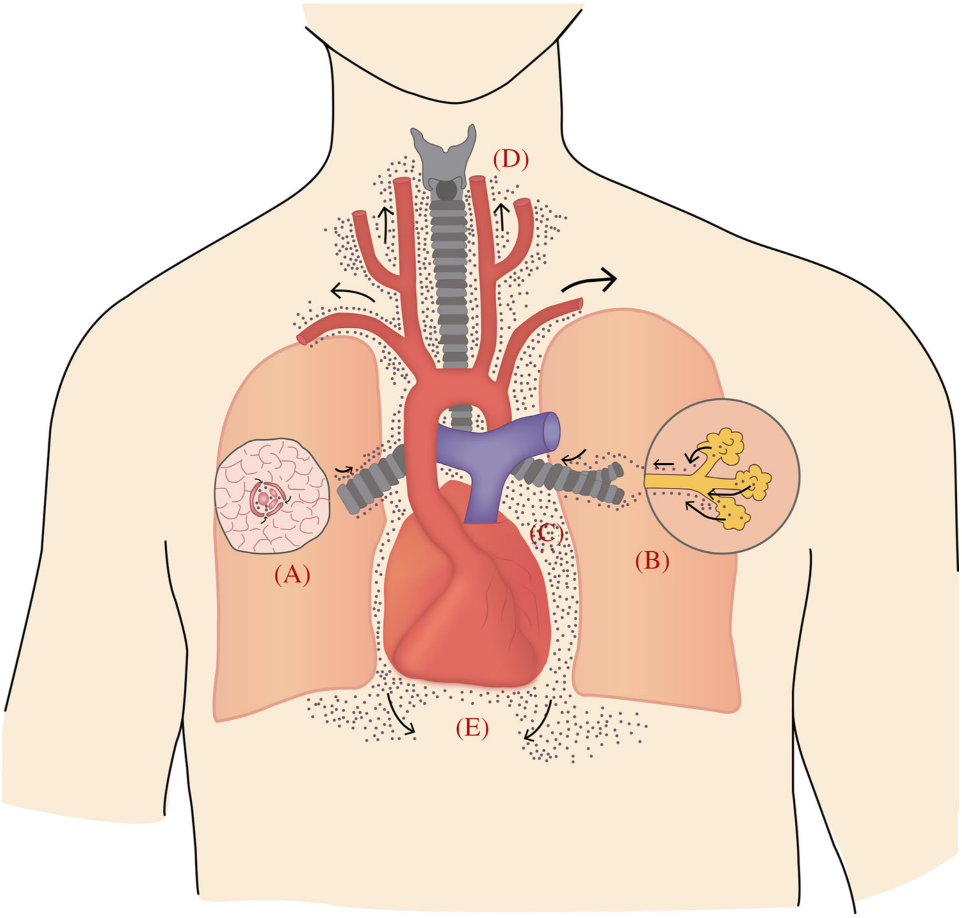

FIGURE 1 Schematic picture depicting the Macklin effect. (A) Alveolar rupture and dissection of air into the bronchovascular sheath. (B) Air passes along the bronchovascular sheath toward the pulmonary hilum. Air reaches the mediastinum (C), the subcutaneous space (D), and the retroperitoneum (E).

and non-traumatic. Most cases of secondary pneumomediastinum reported in COVID-19 were due to positive pressure ventilation (PPV).[10] SPM occurs most commonly in young males. It is defined by the presence of air in the mediastinum without any preceding trauma, surgical or medical procedure (including mechanical ventilation), hollow viscus perforation, or gas-producing infection.[15] There are certain predisposing and precipitating factors for the development of SPM. The predisposing factors include current or past smoking, recent respiratory infection, and substance abuse. The common precipitating factors include cough and vomiting.[16]

The pathophysiology of pneumomediastinum was explained by Macklin and Macklin in 1944, now popularly known as the Macklin effect.[17] According to the Macklin effect, the presence of a transalveolar pressure gradient (the difference between the pressure in the alveoli and the pressure in the perialveolar interstitial space) leads to alveolar rupture resulting in the accumulation of air in the interstitium. From the interstitium, air dissects centripetally along the bronchovascular sheaths toward the pulmonary hila, eventually reaching the mediastinum (Figure 1). The pressure gradient develops because of an increase in intra-alveolar pressure or a decrease in perialveolar interstitial pressure. Valsalva maneuver, coughing, sneezing, defecation, labor, nausea, and vomiting can cause an increase in intra-alveolar pressure. Extreme respiratory effort, marijuana smoking, diabetic ketosis, and rapid reduction in atmospheric pressure can cause a decrease in perialveolar interstitial pressure.[18] Young individuals are more likely to develop spontaneous pneumomediastinum, owing to their loose and flaccid mediastinal tissues, as opposed to the elderly, who have fibrosed planes and sheaths, making air movement more difficult.[15]

Several respiratory infections have alveolocapillary membrane abnormalities, which make the alveoli more susceptible to rupture. Although uncommon, SPM has been reported in various cases of viral pneumonia, including H1N1 influenza.[19] In fact, SPM was also reported in the SARS pandemic in 2003 caused by the SARS coronavirus 1 (SARS-CoV-1).[20] To date, there have been very few case reports of pneumomediastinum without invasive or non-invasive ventilation in patients infected with SARS coronavirus 2 (SARS-CoV-2). COVID-19 renders the alveoli more prone to rupture by cytokine storm-induced diffuse alveolar injury and direct viral infection of type I and type II pneumocytes culminating in pneumomediastinum by the Macklin effect.[11] The most common precipitating factor for SPM is cough, and as explained earlier, cough causes a sudden increase in pressure of these susceptible alveoli leading to overdistension and rupture.[16,21]

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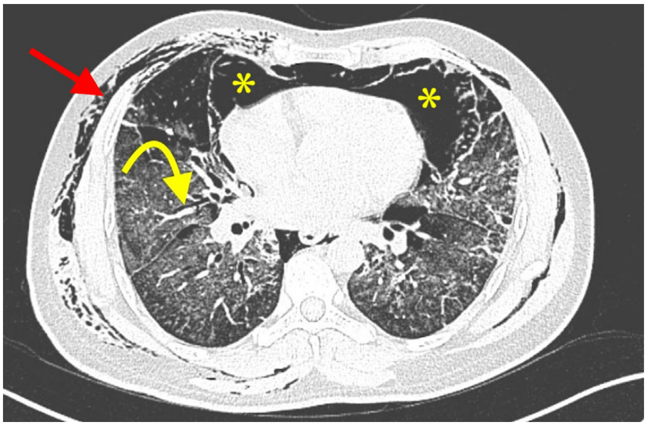

FIGURE 2 Axial computed tomography (CT) chest image of a 34-year-old male patient with COVID-19 demonstrating extensive ground-glass opacities of the lung parenchyma, Macklin effect (curved yellow arrow), pneumomediastinum (asterisks), and subcutaneous emphysema (straight red arrow).

3 | CLINICAL FEATURES AND DIAGNOSIS

The classical triad of pneumomediastinum includes dyspnea, subcutaneous emphysema, and chest pain. Chest pain is usually retrosternal and pleuritic. Cough, neck pain, dysphagia, and odynophagia are the other symptoms. Although chest x-ray may demonstrate continuous diaphragm sign, the gold standard for diagnosis is computed tomography (CT) of the chest.[6,14,22–26] The Macklin effect on CT preceding the development of SPM has also been demonstrated by various radiologists since 1964.[27–30] The Macklin effect could be detected on CT as linear collections of air contiguous to the bronchovascular sheaths in patients with SPM (Figure 2). Recently, in 2021, Belletti et al. did an observational study on COVID-19 patients and found that 95% of them who developed pneumomediastinum demonstrated the Macklin effect on their baseline CT of the thorax. Hence, the Macklin effect on baseline CT scans is a strong predictor of subsequent development of pneumomediastinum.[31] The grading of pneumomediastinum based on chest x-ray and CT is shown in Figure 3. CT of the chest of a patient with grade C pneumomediastinum is depicted in Figure 4.

4 | TREATMENT OF SECONDARY PNEUMOMEDIASTINUM

Pneumomediastinum is usually a benign, self-limiting condition that does not require any specific intervention. In the UK POETIC survey, after the diagnosis of pneumomediastinum, patients who remained on continuous positive airway pressure (CPAP) were retrospectively matched by age, maximum FiO2 and maximum positive end-expiratory pressure with those who were switched immediately to oxygen or high-flow nasal canula. They found no difference in mortality between these 2 subgroups.[13] Thus, there is no evidence to date to recommend taking patients off CPAP when pneumomediastinum develops. Hence, reducing airway pressures to limit the potential for a pressure gradient is the key in cases where pneumomediastinum develops secondary to positive pressure ventilation (non-invasive or invasive). Pneumomediastinum in COVID-19 was associated with higher rates of mechanical ventilation. The presence of pneumomediastinum was also associated with an increased likelihood of ICU admission and greater length of stay.[9] Intubated COVID-19 patients with pneumomediastinum have a complicated course and poor prognosis with increased mortality.[11,13] There are not enough data to date to conclude that mortality is directly related to pneumomediastinum, and additional randomized controlled trials are required to prove this causation. Nevertheless, the increased disease severity per se in patients with pneumomediastinum could have contributed to the mortality.[13]

<table>
<tr><th>Chest X-ray grading</th><th>Chest CT grading *</th></tr>
<tr><td>Grade 0 = no abnormal findings</td><td rowspan="2">Grade A = free air confined to the superior portion of the mediastinum only</td></tr>
<tr><td>Grade 1 = air space present only in the mediastinum</td></tr>
<tr><td>Grade 2 = air space in the mediastinum plus mild subcutaneous emphysema, which was found by careful observation</td><td>Grade B = free air extending from the superior portion of the mediastinum to the middle portion of the mediastinum</td></tr>
<tr><td>Grade 3 = subcutaneous emphysema clearly revealed</td><td rowspan="2">Grade C = free air extending from the superior to the inferior portion of the mediastinum</td></tr>
<tr><td>Grade 4 = marked subcutaneous emphysema</td></tr>
<tr><td colspan="2">* Superior portion of the mediastinum = portion superior to the carina, Middle portion of the mediastinum = from the level of the carina to the orifice of the inferior pulmonary vein, Inferior portion of the mediastinum = portion inferior to the pulmonary vein</td></tr>
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FIGURE 3 Chest x-ray and CT grading of pneumomediastinum.[32] Abbreviation: CT, computed tomography.

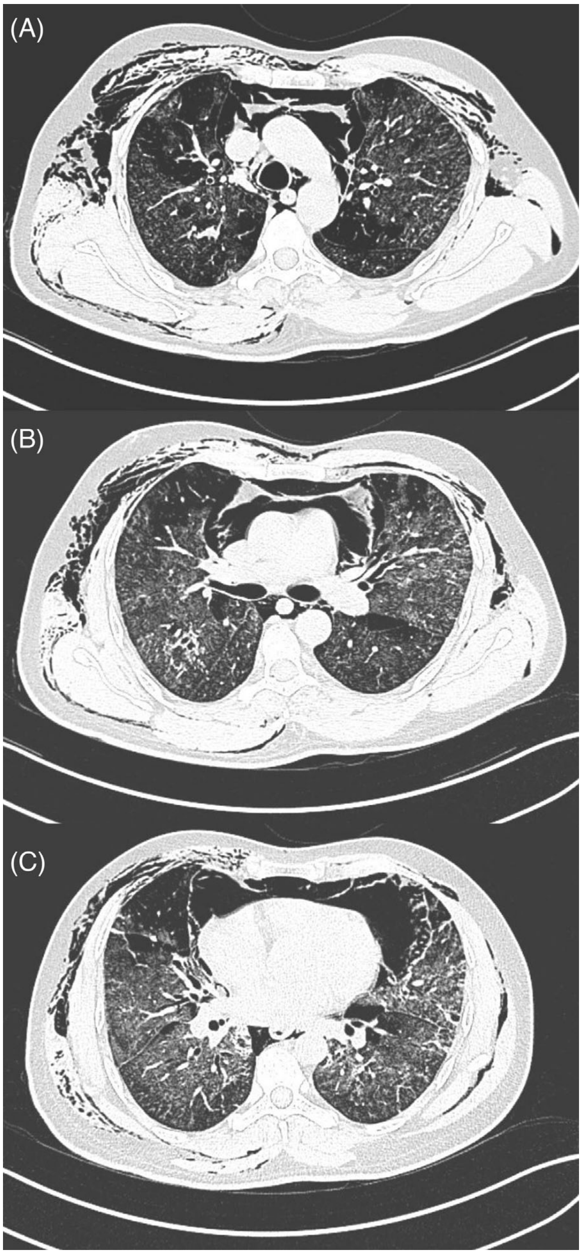

FIGURE 4 Axial CT images of a patient with grade C pneumomediastinum. Free air expands in the superior (A), middle (B), and inferior (C) portions of the mediastinum. Abbreviation: CT, computed tomography.

5 | TREATMENT OF SPONTANEOUS PNEUMOMEDIASTINUM

Supplemental oxygen may be provided to enhance the resorption of interstitial free air. There have been case reports of spontaneous pneumomediastinum being successfully treated with 100% oxygen supplementation.[33] Theoretically, breathing 100% oxygen washes nitrogen out of the blood, thereby increasing the gradient for gas absorption and causing a 4- to 6-fold increase in the gas absorption rate. Though it is a well-known and proven treatment for asymptomatic, small primary spontaneous pneumothorax, there are no randomized controlled trials to support its use in the context of spontaneous pneumomediastinum. Additionally, cough suppressants may reduce the risk of worsening pneumomediastinum.

PPV may lead to pulmonary barotrauma because of the elevation of the transalveolar pressure gradient. So, PPV (invasive or non-invasive ventilation) is contraindicated in patients with SPM. However, in many instances, PPV remains the only option for physicians to prevent these patients with acute respiratory distress from respiratory fatigue and arrest. When PPV is inevitable, minimum required airway pressures should be used to limit the potential for a pressure gradient. Different perspectives exist among various authors in managing these patients who require PPV. Wali et al. advocated using bilateral chest and subcutaneous drains connected to a closed system.[34] It is based on the hypothesis that bilateral intercostal drains would decrease the transpulmonary pressure gradient, which in turn would reduce the intra-alveolar pressure and decrease the risk of alveolar rupture. This ultimately would lead to a decrease in the pneumomediastinum.[19] In contrast, Hamad et al. followed a unilateral intrapleural chest drain approach to protect at least 1 side against potential pneumothorax.[35] On the other hand, Volpi et al. emphasized that conservative management is usually all that is required unless the patient develops pneumothorax.[36] Similarly, Kangas-Dick et al. did not find any statistically significant difference in mortality whether or not a chest drain was placed.[12] To conclude, there is no consensus regarding the use of intercostal drains in intubated patients with pneumomediastinum.

The use of high-flow nasal cannula oxygen therapy in treating ARDS is relatively new. The high-flow nasal cannula generally delivers a much lower positive end-expiratory pressure than non-invasive and invasive mechanical ventilation, and some of this pressure escapes through the mouth opening. However, SPM was reported with the use of high-flow nasal cannula in adults and the pediatric population before the COVID-19 pandemic.[37–39] Few patients with COVID-19 on high-flow nasal cannula also developed SPM.[40–42] Because the alveoli of patients with COVID-19 are more prone to rupture due to diffuse alveolar injury caused by SARS-CoV-2, even the lower positive end-expiratory pressure offered by high-flow nasal cannula might have caused SPM. On the contrary, some studies show that high-flow nasal cannula is safe, and 74% of pneumomediastinum resolved after high-flow nasal cannula therapy.[43] Because the development of SPM could not be solely attributed to high-flow nasal cannula, more randomized controlled trials with matched baseline characteristics are needed to investigate the efficacy and safety of high-flow nasal cannula in patients with COVID-19 who have SPM.

6 | TREATMENT OF TENSION PNEUMOMEDIASTINUM

Due to pneumatic compression of the thorax, massive subcutaneous emphysema associated with pneumomediastinum can cause progressive hypoxemia and hypercapnia and jeopardize the life of the patient. The use of subcutaneous drains in such cases is life saving and effective almost immediately.[44] Malignant or tension pneumomediastinum

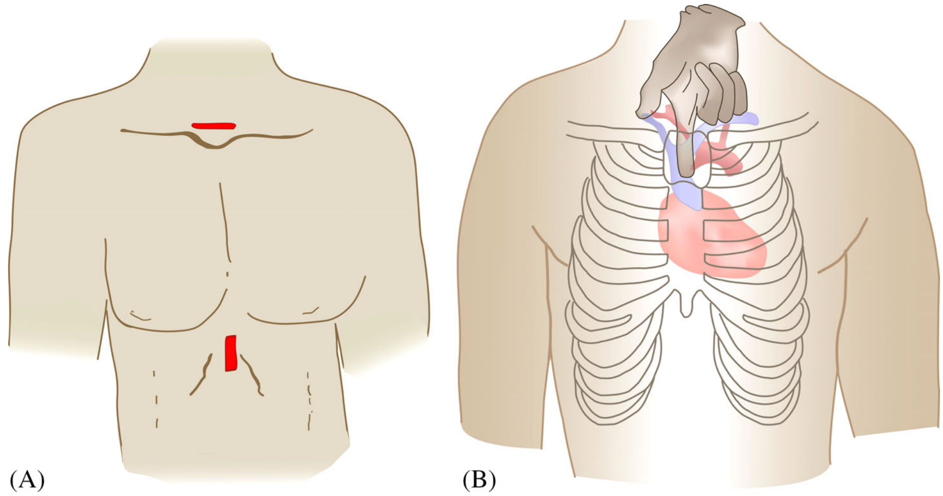

FIGURE 5 (A) Schematic picture depicting the sites for mediastinotomy. (B) Schematic picture depicting mediastinal blunt finger dissection technique via suprasternal approach.

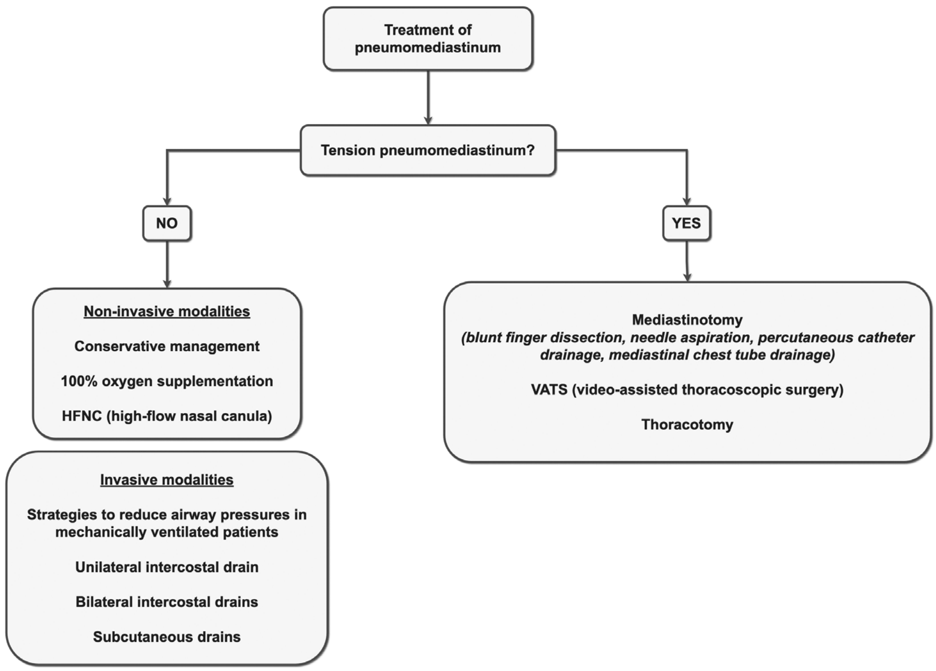

FIGURE 6 Treatment modalities of pneumomediastinum.

is the most severe form in which the trachea and the great vessels of the heart are obstructed, resulting in airway compromise and cardiac tamponade physiology, respectively.[12] The treatment is immediately evacuating the air accumulated in the mediastinum by suprasternal (at the sternal notch) or subxiphoid approach (Figure 5). Mediastinal decompression can be accomplished by various techniques, which include incision followed by blunt finger dissection, needle aspiration, percutaneous drainage catheter followed by continuous suction, and mediastinal chest tube drain placement.[45–47] Because of the risk of aerosolization in patients with COVID-19, physicians should perform blunt finger dissection with appropriate personal protective equipment. The risk of aerosolization can be mitigated in the latter techniques by ensuring it is connected to a closed system. If the tension fails to resolve with mediastinotomy measures, video-assisted thoracoscopic surgery or thoracotomy will be needed for decompression.[15] An approach to the treatment of pneumomediastinum with various modalities of treatment is depicted in Figure 6.

7 | CONCLUSION

Pneumomediastinum requires a high level of suspicion and can be easily missed. Hence, it should be considered in the differential diagnosis of all patients presenting to the emergency department with chest pain and breathlessness in the presence of predisposing and precipitating factors. Pneumomediastinum in COVID-19 is not benign as in other conditions. Hence, it should be promptly recognized. Emergency physicians should be aware of its various treatment modalities, including life-saving interventions for tension pneumomediastinum to manage a wide range of patients presenting during the COVID-19 pandemic.

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