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Case Report

Simultaneous Occurrence of Pneumomediastinum, Pneumopericardium and Surgical Emphysema in a Patient of COVID-19: A Case Report

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I N F O

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A B S T R A C T

As we enter the third year of the COVID pandemic, the SARS-CoV-2 virus keeps on surprising clinicians globally with atypical manifestations. Spontaneous pneumomediastinum is the accumulation of free air within the mediastinum without any preceding trauma or invasive procedure. Pneumomediastinum has been recently reported in the literature, often with associated mortality and morbidity in COVID-19 patients exposed to positive pressure ventilation due to barotrauma. We report the case of a 47-year-old man admitted for management of infection by the SARS-CoV-2. After one week of in-patient treatment, the patient still had worsening dyspnea. Repeat chest X-ray revealed surgical emphysema in neck and NCCT chest revealed pneumomediastinum and pneumopericardium. The spontaneous pneumomediastinum and pneumopericardium with surgical emphysema resorbed over 22 days with a favourable clinical outcome without any invasive intervention. Patient was discharged subsequently with vitals maintaining on room air. A keen clinical observation and timely imaging study can help in detecting these complications early and direct the management accordingly.

Keywords: COVID-19, SARS CoV-2, Pneumomediastinum, Pneumopericardium, Surgical Emphysema, ARDS

Introduction

Pneumomediastinum is defined as the presence of free air in the mediastinum with an incidence of 1 in every 25000 cases in ages between 5 and 34 years, predominantly in males.[1] Many parenchymal and extra-parenchymal abnormalities due to the novel coronavirus Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) have been described on computed tomography (CT) of chest. Parenchymal lesions are both alveolar and interstitial. The presentation on CT depends on the evolution in time of the pathology. Indeed,

the most frequent and early manifestation is parenchymal ground glass opacities.[2] The occurrence of spontaneous pneumomediastinum is an uncommon presentation and spontaneous pneumopericardium with surgical emphysema is even rarer. We are reporting a case of a 47-year-old man admitted for management of infection by the SARS-CoV-2 complicated by pneumomediastinum, pneumopericardium, and surgical emphysema who responded to conservative management.

Case Report

We report a case of 47-year-old gentleman who was hypertensive, hypothyroid, and a recipient of a renal transplant (on immunosuppressive therapy), presenting to the emergency department with complaints of dry cough and shortness of breath for 2 days and was tested positive for SARS-CoV-2 by reverse transcriptase polymerase chain reaction (RT-PCR) on nasopharyngeal swab. The cough and shortness of breath progressively increased and were present both at rest and on minimal exertion. He denied any history of smoking, tobacco, alcohol, or drug use. On physical examination, the patient was conscious and oriented with a blood pressure of 114/68 mmHg, pulse rate of 90 beats per minute, respiratory rate of 24 per minute with the use of accessory muscles of respiration at presentation. He was hypoxic with oxygen saturation (SpO_2) of 85% on room air that improved to 97% on 10 L/min via a non-rebreather mask (NRM). His lung examination revealed bilateral crepitations and the rest of his physical examination was within normal limits.

The patient showed type 1 respiratory failure on arterial blood gas analysis. His haematological profile was unremarkable with total leukocyte count of 12,100 (Normal: 4000-11,000) with neutrophils of 85%. His biochemical profile showed normal liver function teat (LFT), blood urea of 64 mg/dl (19-43), and serum creatinine of 1.27 mg/dl (0.66-1.25) with raised inflammatory markers. Among the inflammatory markers, LDH was 312 U/L (normal:120-246 U/L), high sensitive C-Reactive Protein (hs-CRP) was 47.62 mg/L (Normal:0-5 mg/L), D-dimer was 852 ng/ml (Normal:<500ng/ml), IL-6 was 41.3 pg/ml (Normal:0-7pg/ml), and ferritin was 906 ng/ml (Normal:30-400ng/ml). His chest X-ray (CXR) showed bilateral infiltrates. Hence, a diagnosis of SARS-CoV-2 related severe acute respiratory illness (SARI) with hypoxemic respiratory failure was made in this patient. Patient was started on injectable Ceftriaxone, Azithromycin, Methylprednisolone, Enoxaparin sodium, and injectable Remdesivir was given for 5 days with daily LFT and KFT monitoring. Tacrolimus and Mycophenolate mofetil were continued. He maintained normal vitals on non-invasive supplemental oxygen, requiring 10 litres/minute via NRM but he was still symptomatic after 1 week of supportive management.

In the second week, the patient complained of persistent cough with increased dyspnea with no history of fever, chest pain, orthopnea, pedal edema, decreased urine output, bluish discoloration of nails or altered behaviour. On examination, vitals were normal with SpO_2 of 80% on room air with swelling and crepitus noted on the left side of the neck and crepitations on chest auscultation. Investigations revealed normal electrocardiography, biochemical profile, and CPK-T. and MB with elevated inflammatory markers. The patient required increased oxygen flow of 15 litres per minute via NRM to maintain saturation >94%. A repeat CXR was done, which showed bilateral infiltrates with surgical emphysema on left side of the neck. Thereafter, the same day, a non-contrast CT (NCCT) chest was done which revealed multifocal areas of consolidation noted in bilateral lower lobes with ground glass opacities in bilateral upper lobe and medial segment of the right middle lobe and evidence of air was noted tracking along the mediastinal vessels, trachea, esophagus extending into the deep infrahyoid neck spaces suggestive of pneumomediastinum with surgical emphysema on the left side of the neck [Figure 1(a), 1(b)]. It also revealed air in the pericardial space suggestive of pneumopericardium without any fluid infiltration and with no pneumothorax [Figure 1(c) and 1(d)]. Two-dimensional echocardiography was also done, which revealed normal left ventricular function with an ejection fraction of 60%. The increased dyspnea was attributed to the development of pneumomediastinum and pneumopericardium with surgical emphysema. The patient was kept on intensive observation and was shifted to a higher intensive care unit (ICU) where oxygen therapy was continued with NRM. The patient was continued on conservative management with close monitoring of vitals and oxygen saturation.

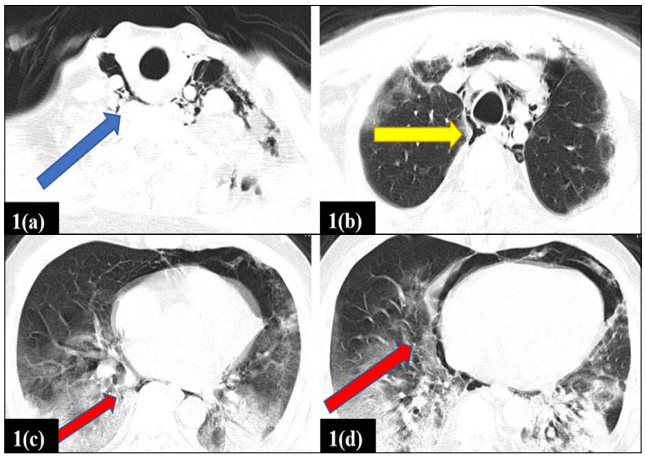

Figure 1.Axial Lung Window of Neck and Chest Show 1(A)-Subcutaneous Emphysema (Blue Arrow);1(B)-Pneumomediastinum (Yellow Arrow), 1(C) and 1(D)-Pneumopericardium (Redarrow), and with Typical CT Pattern of COVID

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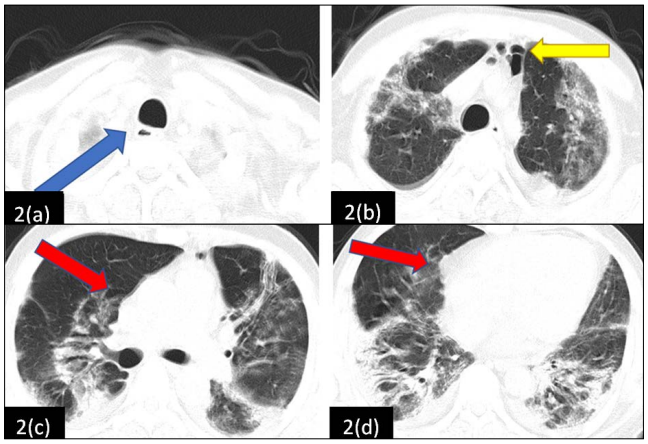

Figure 2.Axial Lung Window of the Chest Reveals 2(a)-Resolution of the Subcutaneous Emphysema (Blue Arrows);2(c) and 2(d)-Resolution of Pneumopericardium (Red Arrow); 2(b)-Pneumomediastinum (Yellow Arrow) Can Still be Seen with Typical Pattern of COVID in Lung

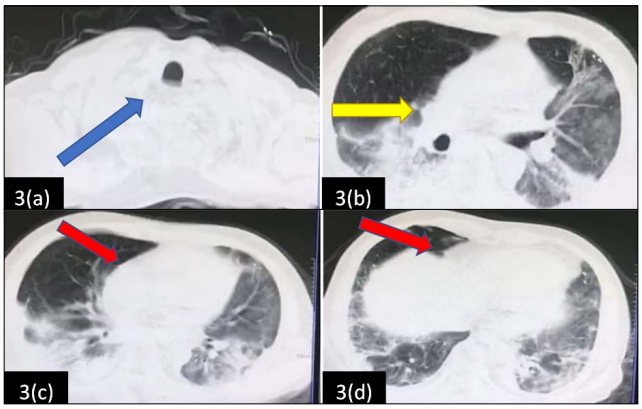

Figure 3.Axial Non Contrast Chest CT in Lung Window Reveals 3(a)-Resolution of Subcutaneous Emphysema (Blue Arrows); 3(b)-Resolution of Pneumomediastinum (Yellow Arrow) and 3(c) and 3(d)-Resolution of Pneumopericardium (Red Arrow) with Persistence of Typical Changes of COVID in the Lung

After 2 weeks, the patient started to improve clinically and was maintaining SpO_2 of 97% on room air at rest, with desaturation of up to 92% on mild exertion. Repeat nasopharyngeal swab was sent for RTPCR for SARS-CoV-2 which came negative.

A repeat NCCT chest was done on day 25 of illness, which showed an improvement and showed few foci of air attenuation in pre-vascular space suggestive of persistent pneumomediastinum but pneumopericardium and surgical emphysema resolved (Figure 2). Eight days later in the fifth week, a third NCCT chest was done which showed resolution of pneumomediastinum as well with typical changes of Coronavirus disease (COVID) in the lung without being drained with a tube (Figure 3).

The patient recovered, remained asymptomatic for further observation period of 3 days and was then discharged, based on existing guidelines. In the absence of a history of chest trauma, positive pressure ventilation, and forceful vomiting with no history of dysphagia or odynophagia, the development of pneumomediastinum and pneumopericardium was attributed to COVID-19 infection.

Discussion

Pneumomediastinum, a condition defined by the presence of air in the mediastinum, was first reported in 1819 by Laennec.[3] In 2015, Kouritas et al. classified pneumomediastinum into spontaneous (primary) and secondary due to iatrogenic; or traumatic; or non-traumatic causes.[4] Spontaneous pneumomediastinum was described by Louis Hamman in 1939, which is why it is also called Hamman's syndrome.[5] Pneumomediastinum has been identified as the most common barotrauma-related event in patients on mechanical ventilation for COVID-19 related acute respiratory distress syndrome (ARDS).[6] Clinical and imaging data of patients seen between March 1, 2020, and April 6, 2020, who tested positive for COVID-19 and experienced barotrauma associated with invasive mechanical ventilation, were compared with patients without COVID-19 infection during the same period. Historical comparison was made to barotrauma rates of patients with acute respiratory distress syndrome from February 1, 2016, to February 1, 2020, at the authors' institution. Comparison of patient groups was performed using categoric or continuous statistical testing as appropriate, with multivariable regression analysis. Patient survival was assessed using Kaplan-Meier curves analysis. Results A total of 601 patients with COVID-19 infection underwent invasive mechanical ventilation (mean age, 63 years ± 15 (standard deviation); 71% men Among patients with COVID-19 pneumonia, 42% could develop (ARDS) with a median time to intubation of 8.5 days from the onset of symptoms.[7,8] The cause of ARDS in COVID-19 is damage to alveolar epithelial cells resulting in hyaline membrane formation in the initial stages, followed by interstitial edema and fibroblast proliferation.[9,10] This causes alveolar rupture, with the leaking of air through the interstitium into the peribronchial and perivascular sheaths backtracking into the hilum and eventually into the mediastinum, a phenomenon which has been described as the Macklin effect, thus leading to pneumomediastinum.[4,11] Pneumomediastinum can be produced, in general, by three different mechanisms:[5] (1) by gas-producing microorganisms present in an infection of the mediastinum or adjacent areas; (2) rupture (whether traumatic or not) of the cutaneous or mucosal barriers-especially perforation of the esophagus or tracheobronchial tree, allowing air to enter the mediastinum from the neck, retroperitoneum or chest wall and (3) the presence of a decreasing pressure gradient between the alveoli and the lung interstitium which may result in alveolar rupture.

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Air from the pneumomediastinum may decompress into the neck and develop surgical emphysema because visceral layers of the deep cervical fascia are continuous with the mediastinum thus avoiding a pneumothorax and a physiologic tamponade. Pneumopericardium develops in the same way as pneumomediastinum does. It would require an elevated pressure in the deep cervical fascia to redirect air towards other planes which could explain why encountering a pneumopericardium is so infrequent.[11] Mechanical ventilation seldom causes isolated pneumopericardium, it is frequently accompanied by pneumomediastinum.[12] Most of the pneumomediastinum cases previously have been reported among patients on high flow positive pressure ventilation like bilevel positive airway pressure or after invasive mechanical ventilation. Our patient was on a non-rebreathing mask throughout hospital stay with oxygen flow tapered on improvement, and hence barotrauma was ruled out. Also, no other causative factor apart from COVID-19 related lung injury was present in our patient. Spontaneous pneumopericardium is rarer than pneumomediastinum and so far only few cases have been reported in COVID-19[13] but most of a them have been related to barotrauma and had a fatal outcome. To the best of our knowledge, this is the first case of COVID-19 related pneumomediastinum synchronous with pneumopericardium with surgical emphysema unrelated to barotrauma with favourable outcome as shown in comparison to other cases shown in Table 1. In a case series of 12 patients by Juárez-Lloclla et al., four out of twelve patients had pneumomediastinum, pneumopericardium, and surgical emphysema with pneumothorax among two of these four patients. All of these patients succumbed to their illness.[14] In contrast, our patient improved with conservative management without the need of mechanical ventilation and was discharged on room air.

Table 1.Summarising Previous Case Reports of Spontaneous Pneumothorax, Pneumomediastinum, Pneumopericardiumand Surgical Emphysema in COVID-19 Patients

S. No.	Author	Number of patients	Mode of ventilation	IMV	Remdesivir	Steroid	CT Chest Findings				Outcome
							Pneumothorax	Pneumomediastinum	Pneumopericardium	Surgical emphysema	
1.	Hazariwala et al.[16]	2	HFNC	2+	-	+	-	+	+	+	Succumbed
2.	Cut et al.[17]pneumothorax (PT	11	Varied	8+	6	+	8	11	1	7	1 with PP and 7 others succumbed
3.	Juárez-Lloclla et al.[14]	12	Varied	1+	-	+	5	11	11	5	6 succ
In this case series by Juárez-Lloclla et al., 4 out of 5 with SE succumbed and 1 had PM, PP and SE who succumbed											
4.	Rashedi et al.[18]	1	NRM	NA	+	+	+	+	+	+	Succumbed
5.	Sahu et al.[13]	1	BiPAP	+	+	+	-	+ (on chest x-ray)	+ (on chest x-ray)	-	Succumbed
6.	Goldman et al.[19]	1	Low flow	-	NA	NA	-	+	-	-	Discharged
7.	Wang et al.[20]	1	Low flow	-	-	+	+	+	-	+	Discharged
8.	Quincho-Lopez et al.[21]	2	Low flow	-	NA	+	1+	2+	-	-	PT+PM -expired PM-discharged
9.	Singh et al.[22]acute respiratory distress syndrome (ARDS	1	Low flow	-	-	+	-	+	+	-	Discharged
10.	Khan et al.[23]	1	-	-	-	-	-	+	+	-	Discharged

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11.	Pimenta et al.[24]	1	NA	+	NA	NA	+	+	+	+	Post intubation outcome NA
12.	Present case	1	NRM	-	+	+	-	+	+	+	Discharged

PP-pneumopericardium; PM-pneumomediastinum; PT-pneumothorax; SE-subcutaneous emphysema; HFNC-high flow nasal canula; NRM-non-rebreather mask; NA-not available

In severe COVID pneumonia, due to a hyperinflammatory process, diffuse alveolar injury is common which may make the alveoli more prone to ruptue especially in patients with persistent coughing. Although the exact mechanism of spontaneous pneumomediastinum and pneumopericardium with surgical emphysema is unknown, increased alveolar pressure leads to a pressure gradient between the alveoli and the lung interstitium; this pressure difference can lead to alveolar rupture thereby causing escape of air into the interstitium. Once the air is in the lung interstitium it flows towards the hilum and the mediastinum along a pressure gradient between the lung periphery and the mediastinum.[11] Nevertheless, in the context of elevated pressures, because of continuity, spontaneous pneumopericardium can still be observed, most frequently synchronous with pneumomediastinum.

Despite pericardial tightness, support of the pericardial reflections is weak at the venous sheaths and could present as a gateway for air.[15] Further studies are needed to discern the risk factors predisposing to the spontaneous alveolar air leak and to find the measures to prevent these complications which can be fatal.

Conclusion

SARS-CoV-2 is a new addition to the causative list of pneumomediastinum, pneumopericardium and surgical emphysema. It is important to look for complications like surgical emphysema and pneumomediastinum whenever a patient with COVID pneumonia does not improve on appropriate therapy. Our patient responded to a conservative approach with oxygen therapy and had a favourable outcome.

Conflict of Interest: None

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