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Dyspnea is a Conundrum in the COVID-19 Era - A Case Report

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Abstract

Background: In the COVID-19 era, a simple dyspnea can raise diagnostic uncertainties especially when presented with patients with underlying interstitial lung disease or congestive cardiac failure which can both present clinically and radiologically similar to COVID-19 pneumonitis. Moreover, such co-morbidities have an increasingly poorer outcome when associated with SARS-CoV-2 infection.

Case Report: An 85-year-old lady presented with dyspnea a week after being treating for chest infection by general practitioner. Chest X-rays and CTPA showed bilateral airspace opacifications with organizing pattern and SARS-CoV-2 was not detected on three PCR swabs. She was managed with diuretics, antimicrobials, steroids, oxygen and non-invasive ventilation.

Conclusion: An acute exacerbation of ILD, decompensated CCF and CT findings of an organizing pneumonia pattern, raises the possibility of previous SARS-CoV-2 infection being the trigger for the patient's presentation. Nasal swabs only detect viral shedding, but not the pending cytokine storm thereby posing diagnostical difficulties.

Keywords: Dyspnea; SARS-CoV-2; Interstitial lung disease; CCF; COVID pneumonia

Abbreviations

COVID-19: Coronavirus disease; RT-PCR: Reverse Transcription-Polymerase Chain Reaction; ILD: Interstitial Lung Disease; CCF: Congestive Cardiac Failure; RA: Rheumatoid Arthritis; ED: Emergency Department; CT: Computed Tomography; O_2: Oxygen; CXR: Chest X-Ray; ARDS: Acute Respiratory Distress Syndrome; SARS-CoV-2: Severe Acute Respiratory Syndrome Coronavirus 2; CRP: C-Reactive Protein; LDH: Lactate Dehydrogenase; CTPA: CT Pulmonary Angiogram; IV: Intravenous; PJP: Pneumocystis Jiroveci Pneumonia; FiO2: Fractional inspired Oxygen; BAL: Bronchoalveolar Lavage

Introduction

The Coronavirus Disease (COVID-19) global pandemic has become a major threat to public health and has completely transformed the clinical approach to patients presenting with dyspnea and other respiratory symptoms.

As clinicians, it is now crucial to include COVID-19 infection and sequela in our differential when presented with all cases of new or increasing shortness of breath. This is also true in cases with negative nasopharyngeal COVID-19 Reverse Transcription – Polymerase Chain Reaction (RT-PCR) tests in light of the sensitivity widely acknowledged as approximately 70% [1].

The diagnostic uncertainty is increased further when presented with patients with underlying lung or cardiac pathologies such as Interstitial Lung Disease (ILD) or Congestive Cardiac Failure (CCF) which can both present clinically and radiologically similarly to COVID-19 pneumonitis [2].

With up to 40% of patients hospitalized with COVID-19 having a background of cardiovascular disease this is an exceedingly common diagnostic dilemma encountered by physicians [3]. Furthermore, patients with COVID-19 infection and CCF are particularly vulnerable to hemodynamic decompensation, ensuing additional diagnostic ambiguity.

Moreover, in those with ILD and CCF, it is imperative to recognize that COVID-19 infection has worse outcomes, especially in elderly population [4-5].

In this article, we report a case of an 85 years old lady who presented with dyspnea and features

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suggestive of COVID-19 infection on a background history of Rheumatoid Arthritis (RA) associated ILD, asbestosis and congestive cardiac failure to highlight the above-mentioned diagnostic difficulties and the importance of multimodality imaging in patients with dyspnea of unclear or potentially multifactorial origin.

Case Presentation

An 85 years old lady presented to Emergency Department (ED) with dyspnea on exertion, bipedal edema and facial puffiness for 2 weeks. Few weeks prior to her dyspnea, she had some dry cough and was treated for lower respiratory tract infection with oral doxycycline. Her worsening dyspnea prompted her visit to ED. She had a background history of seropositive rheumatoid arthritis associated ILD, hypertension, atrial fibrillation, heart failure with preserved ejection fraction of 50% to 55% and possible asbestosis as per recent Computed Tomography (CT) imaging 4 months ago (Figure 1).

On examination, she was afebrile with a heart rate of 54 beats per minute, respiratory rate of 20 breaths per minute and oxygen saturation of 75% to 80% on room air. She was dyspneic while conversing, had noticeable facial puffiness, digital clubbing and bilateral pedal edema. On auscultation, bilateral widespread coarse crackles were heard. Her oxygenation improved on 8 Littre's of Oxygen (O2) via high-flow nasal cannula, saturating 96%.

An arterial blood gas on room air revealed hypoxemic respiratory alkalosis. A Chest X-Ray (CXR) (Figure 2) shows bilateral patchy

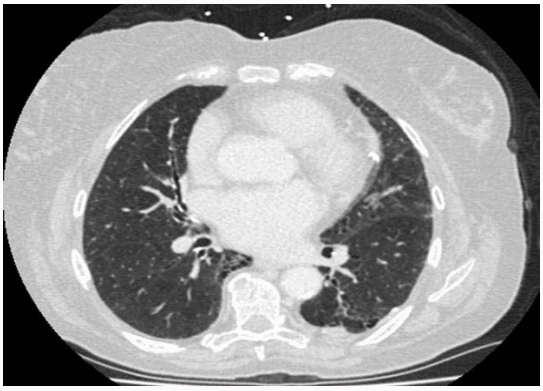

Figure 1: CT Thorax axial image showing multiple calcified processes in keeping with prior asbestos exposure and subpleural areas of fibrosis noted in the lower lungs bilaterally and left upper lobe, in keeping with asbestosis.

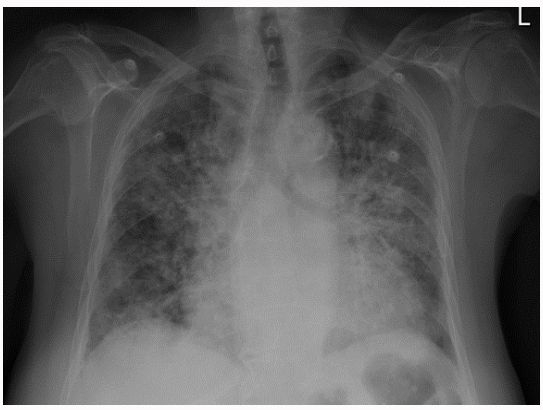

Figure 2: CXR on day 1 showing bilateral air space opacities with Kerley B lines.

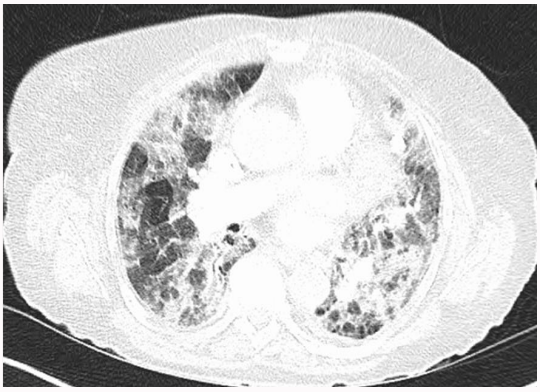

Figure 3: CTPA axial view showing new findings of extensive bilateral peribronchovascular ground glass and patchy consolidation with associated architectural distortion, slightly more marked in the upper lobes with no evidence of pulmonary embolus.

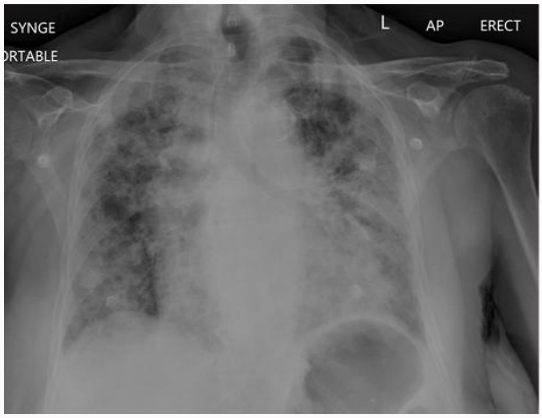

Figure 4: CXR on day 4 with worsening pulmonary opacities.

airspace opacification with Kerley B lines raising the possibility of cardiogenic pulmonary edema and Acute Respiratory Distress Syndrome (ARDS) secondary to Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) was contemplated in view of prior dry cough. Initial bloods revealed raised C-Reactive Protein (CRP) of 37.19, white blood cell count 10.9, neutrophil count 9.1 and a lymphocyte count of 1.2. Her hyperinflammatory markers were mildly elevated, Lactate Dehydrogenase (LDH) -484, D-Dimer 942, IL-6 202.96 and serum Ferritin 138.6.A CT Pulmonary Angiogram (CTPA) was performed (Figure 3) in view of her raised D-dimer revealed multiple differentials.

She was initially treated as a community acquired pneumonia with Intravenous (IV) co-amoxiclav 1.2 g/8h and diuresis for heart failure with IV furosemide 40 mg/12h. Due to risk of prolonged QTC interval, Clarithromycin was withheld. She was isolated pending COVID-19 RT-PCR tests by 3 and all 3 swabs obtained on consecutive days did not detect SARS-CoV-2. On Day 3, her antimicrobial therapy was escalated to piperacillin-tazobactam 4.5 g/6h with oral prednisolone 60 mg.

On Day 4, IV meropenem 1 g/8h, oral doxycycline 100 mg once daily and co-trimoxazole 80/400 mg per oral/12h to cover for Pneumocystis Jiroveci Pneumonia (PJP) were initiated following microbiology and respiratory specialist advice.

Although her volume status improved, her O_2 requirement

increased warranting escalation to High Flow Nasal Oxygen (HFNO) with Fractional inspired O_2 (FiO_2) of 40% to 60% with a flow rate of 30 L/min to 40 L/min. However, she was unable to maintain adequate oxygenation with maximal FiO_2 setting and repeat portable chest X-ray (Figure 4) revealed disease progression. She declined invasive mechanical ventilation and passed away peacefully after 6 days.

Discussion

We report a case where an acute exacerbation of ILD and cardiogenic pulmonary edema could have been triggered by recent SARS-CoV-2 infection. Our patient had 3 negative nasopharyngeal swabs' PCR for COVID-19 during her hospital stay. However, we reckon that her viral load may have been low to be detected by RT-PCR [6]. In keeping with hospital policy, we went forward with only specimen collection from nasopharynx and did not opt for sputum or Bronchoalveolar Lavage (BAL) swabs.

We were unable to do a serum anti- SARS-CoV-2 antibody test due to restricted resources. There have been multiple studies demonstrating the varying degrees of viral shedding on different specimens, especially in elderly patients [7] and organizing pneumonia has also been reported to be a sequala post resolution of COVID pneumonia [8].

The patient's arthritic symptoms were well controlled with Methotrexate 15 mg weekly and Adalimumab 40 mg subcutaneous injection fortnightly. She never had any baseline pulmonary function tests which could have predicted her outcome for any RA-ILD exacerbation [9].

There are many questions that remain unanswered with her complex case. The duration of illness seems very acute to rationalize a single diagnosis based on her CT findings. Diuresis seemed to have a good effect, but the chest X-rays revealed persistent bilateral pulmonary opacities. These opacities could have been secondary to an atypical viral, bacterial, fungal infection or drug induced pneumonitis or exacerbation of ILD.

This case report demonstrates a potentiality that SARS-CoV- 2 could have been a trigger for her acute flare of RA-ILD as she first developed dry cough a week ago and was treated as a chest infection in the community. It remains uncertain in terms of her prognosis, whether treatment with Tocilizumab coupled with mechanical ventilation would have led to a successful outcome. Nevertheless, the importance of ameliorating management of those with ILD especially during the SARS-CoV-2 period awaits more research.

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