

CASE REPORT

The Diagnostic Value of Auscultation in a Post-COVID Patient with Lobar Pneumonia

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ABSTRACT

Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2), the virus responsible for COVID-19, often progresses to multisystemic disease. Persistent pulmonary symptoms are common due to ongoing inflammation, endothelial injury, and parenchymal damage, which increases susceptibility to secondary infections. A comprehensive physical examination, with careful attention to pulmonary auscultation, is a critical component in the early detection of such complications.

We report the case of a 73-year-old woman with a history of hypertension and chronic rhinitis who presented for follow-up for COVID-19. She had tested for COVID-19 approximately 3.5 weeks earlier, and mild congestion and cough had persisted. She was a non-smoker and reported occasional social alcohol consumption. On physical examination, her vital signs were stable, and pulmonary examination revealed coarse crackles in the right lower lobe. Chest radiograph showed hyperinflated lungs with right middle and lower lobe opacities and atelectasis; overall findings consistent with lobar pneumonia. She was treated with azithromycin and benzonatate and scheduled for close clinical follow-up.

This case emphasizes the importance of careful physical exam maneuvers on post-COVID patients to monitor complications. Auscultatory abnormalities can serve as an early indicator of post-infectious pulmonary complications, guiding timely imaging, diagnosis, and treatment to reduce morbidity in the post-COVID period.

KEYWORDS

Post-COVID syndrome; Complications of COVID-19; Lobar pneumonia; Atelectasis

INTRODUCTION

Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2), the causative agent of COVID-19, has infected millions of individuals worldwide and continues to pose significant challenges not only to healthcare systems but also to the global economy [1]. Although initially considered an acute respiratory illness, COVID-19 is now recognized as a multisystem disease [2]. The clinical presentation of acute infection typically includes fever, fatigue, chills, cough, and upper respiratory symptoms. However, progressive disease may involve multiple organ systems, including the cardiovascular, neurologic, and gastrointestinal systems. Although pulmonary involvement remains the most common and significantly affected manifestation of COVID-19, complications such as pneumonia, thromboembolic events, and myocardial injury highlight the broad pathophysiological impact of SARS-CoV-2 infection [1,2].

As the pandemic has evolved, increasing attention has been directed toward characterizing patients with persistent symptoms beyond the acute infectious phase. According to the definitions provided in the National Institute for Health and Care Excellence (NICE) guidelines, COVID-19 can be categorized into three distinct phases: acute COVID-19 (symptoms lasting up to 4 weeks), ongoing symptomatic COVID-19 (symptoms persisting from 4 to 12 weeks), and post-COVID-19 syndrome (symptoms persisting beyond 12 weeks without an alternative diagnosis) [3].

The pathogenesis of post-COVID-19 syndrome is multifactorial and is associated with a wide range of clinical manifestations. Persistent immune activation and dysregulation of inflammatory pathways play important roles. Elevated levels of proinflammatory cytokines, including interleukin-6 (IL-6), interleukin-1 (IL-1), and tumor necrosis factor-alpha (TNF- α), may contribute to sustained inflammation and tissue injury. Moreover, endothelial dysfunction and abnormalities in coagulation pathways may result in microvascular thrombosis, impaired tissue perfusion, and further tissue injury [4]. Within the respiratory system, these processes may contribute to parenchymal injury, atelectasis, and persistent airway inflammation, potentially increasing susceptibility to secondary infections, including bacterial pneumonia.

Despite significant advances in the prevention and management of COVID-19, post-COVID-19 complications remain clinically important, particularly those affecting pulmonary function. Respiratory symptoms such as cough, dyspnea, and wheezing are commonly reported among patients with post-COVID-19 syndrome, including individuals who initially experienced mild disease [1,4]. In outpatient settings, where access to radiographic imaging may be limited, physical examination remains an essential component of clinical assessment. Careful pulmonary auscultation may reveal abnormalities such as fine or coarse crackles and asymmetric breath sounds, which can prompt further diagnostic evaluation and influence clinical management [3,4].

This case highlights the importance of performing a thorough physical examination in patients with persistent respiratory symptoms following COVID-19 infection to facilitate the early identification of potentially serious pulmonary complications.

CASE PRESENTATION

We report the case of a 73-year-old woman with a medical history of well-controlled hypertension, chronic rhinitis, and osteoporosis. She presented to the clinic for her annual visit with an additional complaint of persistent nasal congestion following COVID-19 infection. She tested positive for COVID-19 three and a half weeks prior to presentation on an at-home test and initially experienced sore throat, nasal congestion, postnasal drip, loss of

taste, and decreased appetite. Her cough was described as mostly dry and non-productive, though she also reported some sputum production. Although her symptoms initially improved with conservative management using acetaminophen and cetirizine, she continued to experience residual congestion and cough interfering with her daily activities. She denied any known sick contacts.

Her current outpatient medications included lisinopril 10 mg daily, propranolol 60 mg daily, cholecalciferol 25 mg daily, mometasone nasal spray once daily, and omeprazole 40 mg daily.

Family history was notable for breast and colon cancer in a paternal aunt, and coronary heart disease and hypertension in both parents. Social history was negative for smoking and illicit drug history, but positive for occasional alcohol consumption, averaging approximately 10 drinks per week.

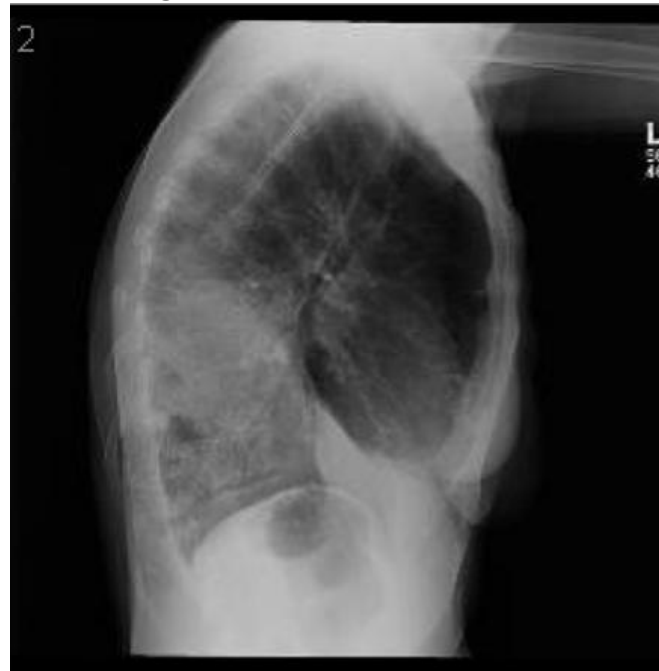
On physical examination, her vital signs were stable with a blood pressure of 104/70 mmHg, heart rate of 79 beats per minute, and temperature of 98°F. Pulmonary examination revealed normal breath sounds overall, with coarse crackles noted in the right lower lobe. Cardiovascular examination demonstrated a regular rate and rhythm. The patient appeared comfortable and in no acute distress.

Upon assessment, the patient's presentation was consistent with post-COVID respiratory symptoms, potentially related to residual upper and lower airway inflammation superimposed on chronic rhinitis. She was prescribed benzonatate 100 mg three times daily for cough, and a chest X-ray was ordered for further evaluation. Chest X-ray demonstrated hyperinflated lungs with sparing of the broncho-vascular bundles, a dense opacity in the right lower and middle lobes, and a triangular density in the right middle lobe suggestive of atelectasis (Figure 1 and Figure 2). Additional findings included an enlarged cardiomeastinal silhouette, minor aortic calcification, and a prominent pulmonary artery. The overall radiographic impression was lobar pneumonia with associated atelectasis on a background of emphysematous changes. The patient was instructed to continue symptomatic management, monitor her respiratory symptoms. She was subsequently started on azithromycin 250 mg with two tablets for one day and one tablet per day for the next four days.

Figure 1: Chest X-Ray (anteroposterior view).



Figure 2: Chest X-Ray (lateral view).



DISCUSSION

COVID-19 remains a significant contributor to global morbidity, acutely presenting an upper respiratory illness with the capacity to affect a multitude of systems in the body. While most patients recover from the acute phase, literature highlights the persistence of symptoms and the risk of delayed complications termed as post-COVID-19 [4].

As described previously, post-COVID-19 syndrome most commonly leads to symptoms such as persistent cough and shortness of breath but may lead to more lasting damage in the form of pulmonary hypertension or fibrosis. Non-respiratory symptoms may include chronic fatigue and a perceived decrease in cognitive function usually referred to as brain fog. It is due to the possibility of these symptoms developing that patients should continue to follow up for several weeks after the initial diagnosis of acute COVID-19. Elderly patients with risk factors for increased disease severity, such as the patient presented in this case, who was 73-year-old with a history of chronic rhinitis, are recommended to follow up within 3 weeks of initial diagnosis [5].

This case is clinically significant as it showcases the development of lobar pneumonia with associated atelectasis in a patient with initially mild COVID-19 symptoms and stable vital signs, highlighting a potentially underrecognized progression of disease in an outpatient setting. Although post-viral bacterial pneumonia is a known complication of respiratory infections, its occurrence in the early post-COVID period without clear clinical deterioration is less evident in current literature [6].

The pathophysiology of this patient's lobar pneumonia is likely to be multifactorial. Post-viral dysregulation may disrupt the host's immune system, increasing susceptibility to infection. In addition, SARS-CoV-2 mediated damage of the ciliated respiratory epithelium, and mucociliary clearance could have contributed to atelectasis due to mucus retention and airway obstruction [7]. Persistent low-grade inflammation and endothelial dysfunction can further exacerbate localized pulmonary injury, even in the absence of signs such as fever. Overall, these mechanisms can provide an explanation for the discordance seen in this patient's benign clinical appearance and significant radiographic findings.

The physical exam played a pivotal role in determining the cause of the patient's symptoms in the post-COVID-19 period. The findings of normal vital signs with coarse crackles on auscultation indicated residual pulmonary infiltration, which is a cause of persistent symptoms in COVID-19. The auscultatory findings led to further workup with chest radiograph imaging, which indicated lobar pneumonia with atelectasis. The initial performance of the auscultation was an essential step in indicating the need for further workup to determine the cause of the symptoms. This is especially relevant in resource-limited areas where imaging is not routinely performed [1].

CONCLUSION

Overall, this case illustrates the value of thorough physical examination, particularly pulmonary auscultation, in patients recovering from COVID-19. Importantly, it highlights that clinically significant pathology may be present even in stable patients with mild symptoms. Clinicians should maintain a low threshold for performing thorough pulmonary examinations and should consider further diagnostic evaluation in patients with persistent symptoms following COVID-19. Subtle findings can serve as the only indication requiring further work up. Follow-up is highly recommended for patients in the weeks after the acute infection, and timely recognition through careful examination can help to identify the cause of any potential symptoms, as it did in the case presented here.

AUTHOR CONTRIBUTIONS

SE: Conceptualization, Original Draft, Review and Editing AG: Original Draft, Review and Editing NK: Original Draft, Review and Editing BK: Conceptualization, Supervision, Review and Editing.

CONSENT FOR PUBLICATION

Written informed consent was obtained from the patient for the publication of this case, including clinical details and laboratory values. Patient confidentiality was preserved with the omission of all identifiable details, and all data was accessed via encrypted and protected applications.

CONFLICTS OF INTERESTS

The authors declared no conflicts of interest between co-authors.

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REFERENCES

1. Siddiqi HK, Mehra MR (2020) COVID-19 illness in native and immunosuppressed states: A clinical-therapeutic staging proposal. *The Journal of Heart and Lung Transplantation* 39(5): 405-407.
2. Perumal R, Shunmugam L, Naidoo K (2023) Long COVID: An approach to clinical assessment and management in primary care. *South African Family Practice* 65(1): e1-e10.
3. Silva Andrade B, Siqueira S, de Assis Soares WR, et al. (2021) Long-COVID and post-COVID health complications: An up-to-date review on clinical conditions and their possible molecular mechanisms. *Viruses* 13(4): 700.
4. Esendağlı D, Yilmaz A, Akçay Ş, et al. (2021) Post-COVID syndrome: Pulmonary complications. *Turkish Journal of Medical Sciences* 51(Suppl 1): 3359-3371.
5. Chudzik M, Babicki M, Kapusta J, et al. (2022) Long-COVID clinical features and risk factors: A retrospective analysis of patients from the STOP-COVID Registry of the PoLoCOV study. *Viruses* 14(8): 1755.

6. Trifonova I, Korsun N, Levterova V, et al. (2025) Respiratory infections in the post-COVID-19 era: Impact, prevalence, and clinical characteristics of bacterial and viral co-infections. *Frontiers in Medicine* 12: 1597782.
7. Robinot R, Hubert M, de Melo GD, et al. (2021) SARS-CoV-2 infection induces the dedifferentiation of multiciliated cells and impairs mucociliary clearance. *Nature Communications* 12(1): 4354.