

Beyond the Ground-Glass Opacity: Diagnostic Mimics of COVID-19 Pneumonia—A Three-Case Series

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Received: October 21, 2026; **Published:** October 28, 2026

Abstract

Coronavirus disease 2019 (COVID-19) commonly produces non-specific respiratory symptoms and chest computed tomography (CT) abnormalities that can overlap with non-infectious disorders. We describe three patients whose clinical and radiological presentations initially suggested COVID-19 pneumonia but whose severe acute respiratory syndrome coronavirus 2 testing was negative. A 53-year-old woman with rheumatoid arthritis had multifocal bilateral consolidation and ground-glass change; bronchoscopic cryo biopsy showed organising pneumonia, and the abnormalities resolved with prednisolone. A 19-year-old man had bilateral ground-glass opacities, marked peripheral eosinophilia, and bronchoscopic lavage with 50% eosinophils, supporting idiopathic eosinophilic pneumonia; he improved after prednisolone. A 68-year-old man with fever and haemoptysis had bilateral nodular and ground-glass changes; echocardiography revealed severe mitral valve prolapse with chordal rupture and significant mitral regurgitation. These cases demonstrate that COVID-19-like imaging is not specific and that persistent test negativity should prompt careful evaluation for alternative inflammatory, eosinophilic, and cardiogenic causes.

Keywords: COVID-19 Pneumonia; COVID-19 Mimics; Pulmonary Eosinophilia; Cryptogenic Organising Pneumonia; Mitral Valve Regurgitation; Cryo Lung Biopsy

Introduction

The COVID-19 pandemic complicated the diagnosis of respiratory illness because common symptoms and imaging findings overlap with many infectious and non-infectious conditions. Patients commonly present with cough, fever, myalgia, headache, and dyspnoea; however, fever is not universal, and one New York series reported a temperature above 38°C in 31% at admission [1-4]. Chest computed tomography (CT) often shows bilateral peripheral ground-glass opacities and patchy consolidation [5]. Similar appearances occur in non-COVID-19 conditions. This case series describes three patients in whom COVID-19 was initially suspected but alternative inflammatory, eosinophilic, and cardiogenic diagnoses were established after further investigation.

Case Reports

Case 1

A 53-year-old Asian woman with a history of seronegative rheumatoid arthritis, hypertension, hyperlipidaemia, and hypothyroidism was admitted with mild cough and abnormal findings on chest X-ray and CT scans. Her regular medications included hydroxychloroquine, sitagliptin, metformin, rosuvastatin, and thyroxine.

Citation: Trilok Chand and Samah Beltagy. "Beyond the Ground-Glass Opacity: Diagnostic Mimics of COVID-19 Pneumonia—A Three-Case Series". *EC Pulmonology and Respiratory Medicine* 15.11 (2026): 01-07.

On examination, she was afebrile and normoxic, and chest auscultation revealed a few basal crackles. A chest X-ray revealed multifocal, ill-defined patchy opacities in the left mid-zone and bilateral lower zones (Figure 1). High-resolution computed tomography (HRCT) of the chest showed multifocal patchy and confluent areas of consolidation, with ground-glass attenuation and interlobular septal involvement in both lungs (Figure 2 and 3). Blood tests were unremarkable, and severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) reverse transcription polymerase chain reaction (RT-PCR) was negative.



Figure 1: Chest X-ray in case 1 showing multifocal ill-defined patchy opacities in the left mid-zone and bilateral lower zones.

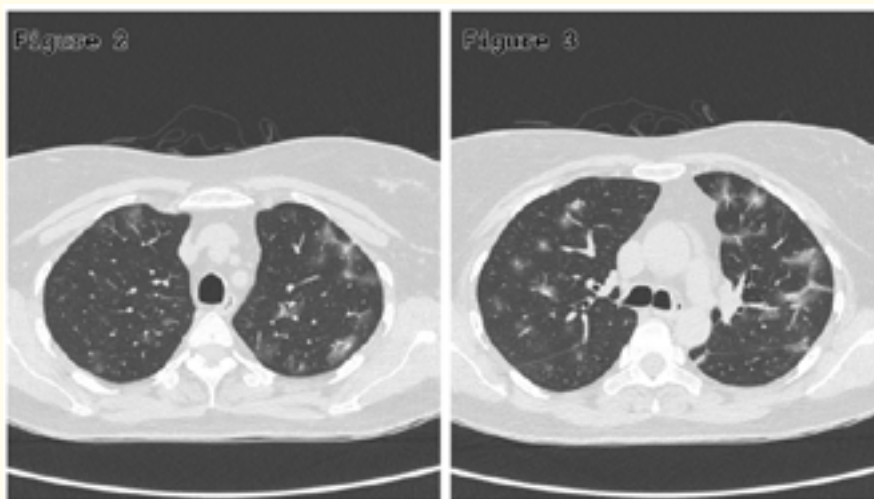


Figure 2 and 3: High-resolution chest CT in case 1 showing multifocal patchy and confluent consolidation with bilateral ground-glass attenuation and interlobular septal involvement.

Because the lung shadows persisted, a bronchoscopic cryo lung biopsy was performed and showed focal organising pneumonia with mild chronic interstitial inflammation. The patient was started on prednisolone therapy, and after six months of treatment, follow-up chest CT showed complete resolution of the abnormalities (Figure 4 and 5).

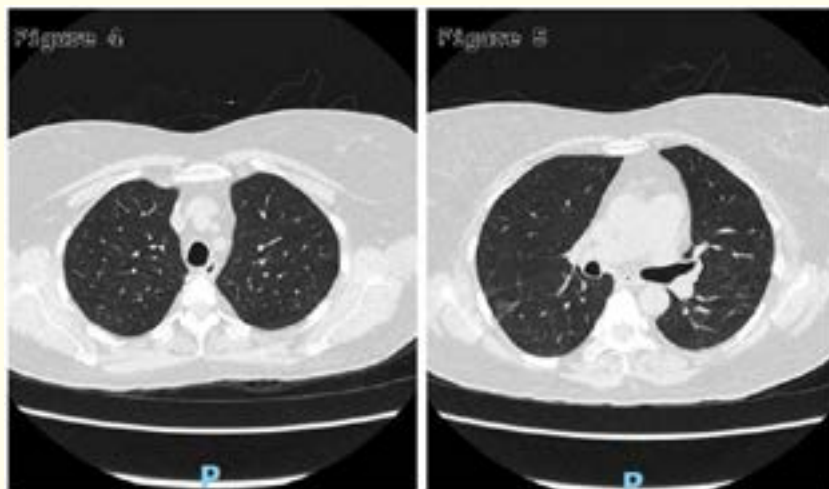


Figure 4 and 5: Follow-up CT chest imaging in case 1 after six months of prednisolone showing complete resolution of the previous abnormalities.

Case 2

A 19-year-old Arab man, a smoker, presented with dry cough, fever, and occasional dyspnoea accompanied by wheezing for three days. He reported a history of mild cough lasting two months. On examination, he was febrile and normoxic, with normal bilateral breath sounds. A chest X-ray showed ill-defined, inhomogeneous soft-tissue opacities in the right lower lung zone, raising suspicion of an infectious aetiology (Figure 6).

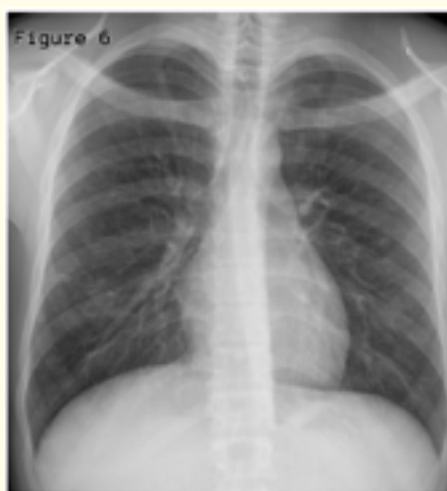


Figure 6: Chest X-ray in case 2 showing ill-defined inhomogeneous soft-tissue opacity in the right lower lung zone.

HRCT chest showed scattered patchy areas of alveolar opacification with bilateral ground-glass appearances. Most lesions were in the central zones, with ground-glass halos surrounding peribronchial opacities (Figure 7 and 8). Blood tests showed elevated C-reactive protein (29.50 mg/L), ferritin (436.20 mcg/L), white blood cell count ($23.13 \times 10^3/\mu\text{L}$), and eosinophil count ($18.86 \times 10^3/\mu\text{L}$). SARS-CoV-2 RT-PCR was negative, as were antinuclear antibody (ANA) and antineutrophil cytoplasmic antibody (ANCA) profiles. Stool tests for ova and cysts were also negative.

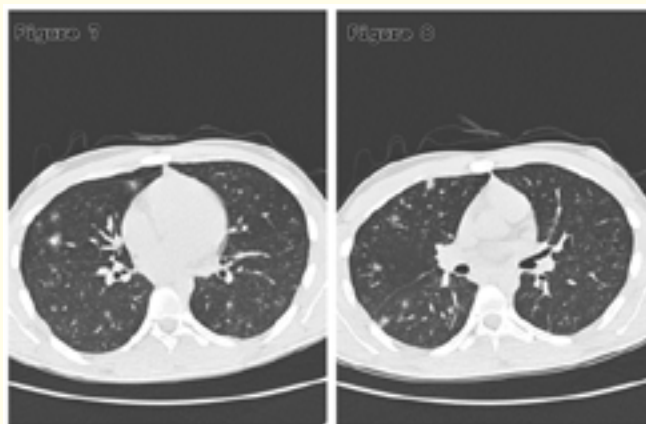


Figure 7 and 8: High-resolution chest CT in case 2 showing scattered bilateral patchy ground-glass alveolar opacification with peribronchial opacities and surrounding ground-glass halos.

Bronchoscopic cryo lung biopsy and lavage revealed 50% eosinophils, with eosinophilic infiltration in the interstitium and alveoli. Macrophages and focal eosinophilic abscesses were noted, but there was no evidence of definitive fibrosis or vasculitis. The findings were consistent with idiopathic eosinophilic pneumonia. The patient was started on prednisolone therapy, and after few weeks of treatment, his eosinophil levels normalized but he was continued on tapering dose of prednisolone for three months. Subsequent CT chest showed complete resolution of the opacities (Figure 9 and 10).

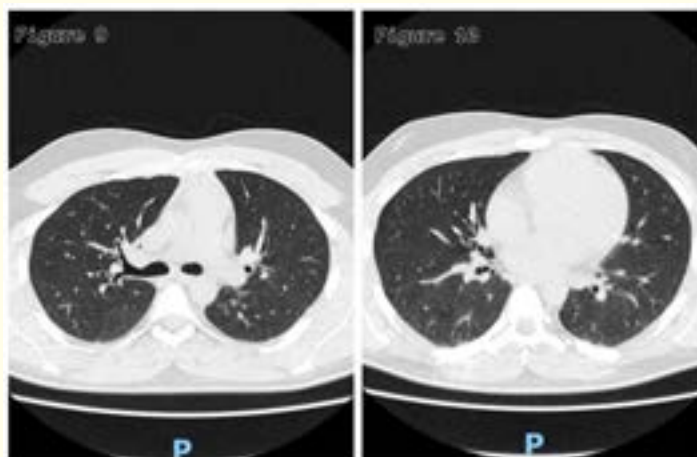


Figure 9 and 10: Follow-up high-resolution chest CT in Case 2 after three months of prednisolone showing complete resolution of the opacities.

Case 3

A 68-year-old European man who had never smoked was admitted with a two-day history of fever and haemoptysis. He had a history of hypertension. On physical examination, his temperature was 37.4°C and oxygen saturation was 94% on room air. Chest auscultation revealed right basal crackles. Laboratory investigations showed haemoglobin 13.6 g/dL, white blood cell count $12.3 \times 10^9/\text{L}$, platelet count $216 \times 10^9/\text{L}$, and C-reactive protein (CRP) 18.41 mg/L. D-dimer was 0.60, serum ferritin was 255.50 mcg/L, and lactate dehydrogenase (LDH) was 279 U/L. SARS-CoV-2 RT-PCR was negative. A chest X-ray revealed patchy consolidation in the right perihilar region and lower lung zones (Figure 11).



Figure 11: Chest X-ray in case 3 showing patchy consolidation in the right perihilar region and lower lung zones.

CT pulmonary angiography ruled out pulmonary thromboembolism but demonstrated multiple, discrete and confluent tiny nodular and tree-in-bud opacities surrounded by ground-glass attenuation. These findings were scattered throughout both lungs, predominantly in the perihilar regions and anterior segments of the upper lobes. Mediastinal and hilar non-necrotic lymphadenopathy were also present. The findings suggested an infectious aetiology (Figure 12 and 13).

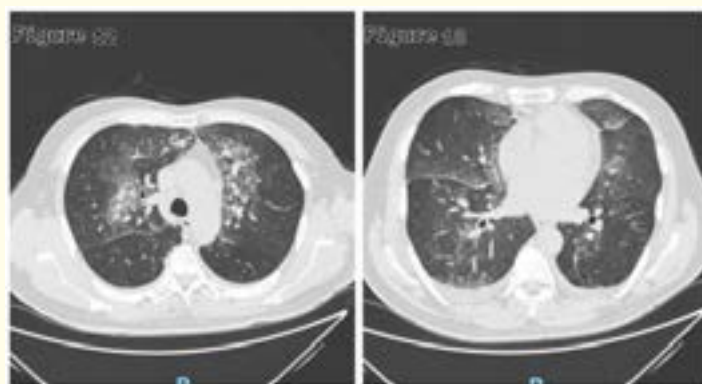


Figure 12 and 13: CT pulmonary angiography in case 3 showing tiny nodular and tree-in-bud opacities with surrounding ground-glass attenuation, with mediastinal and hilar non-necrotic lymphadenopathy.

An autoimmune profile was negative in the evaluation for alveolar haemorrhage. However, a two-dimensional echocardiogram revealed severe mitral valve prolapse with chordal rupture, resulting in significant mitral regurgitation. Pulmonary artery systolic pressure (PASP) was elevated at 70 mmHg. The patient was started on diuretics and digoxin, which led to slight improvement, but surgical intervention was recommended to repair the ruptured chordae.

Discussion and Conclusion

The COVID-19 pandemic significantly reshaped the diagnostic approach to respiratory illness. Its broad clinical presentation and characteristic radiological patterns created a high index of suspicion for SARS-CoV-2 in patients with fever, cough, and dyspnoea. However, overlap in symptoms and imaging with other pulmonary and cardiopulmonary disorders can increase the risk of misdiagnosis. This case series highlights three patients in whom clinical and radiological features initially suggested COVID-19 but further evaluation established different diagnoses.

Across the three cases, presenting features included fever, cough, dyspnoea, or haemoptysis, and chest CT showed bilateral abnormalities that included ground-glass attenuation. SARS-CoV-2 RT-PCR testing was negative in all three patients. Although false-negative RT-PCR results are recognised, testing should be interpreted alongside the clinical course and alternative diagnoses rather than imaging alone [6]. The first case was diagnosed with organising pneumonia on bronchoscopic lung biopsy. The second case was diagnosed with idiopathic eosinophilic pneumonia on the basis of marked peripheral eosinophilia and bronchoscopic biopsy and lavage findings. The third case was ultimately attributed to severe mitral valve prolapse with chordal rupture and significant mitral regurgitation, confirmed on echocardiography.

Organising pneumonia may present with patchy or confluent consolidation and ground-glass change and can respond to corticosteroid therapy [7]. Eosinophilic lung diseases can produce pulmonary infiltrates with peripheral or bronchoalveolar eosinophilia, making tissue and lavage findings important to diagnosis [8]. Severe mitral regurgitation due to chordal rupture can cause cardiogenic pulmonary oedema that is mistaken for pulmonary infection, and echocardiography can help distinguish the cardiac cause [9].

These cases underscore the importance of maintaining a broad differential diagnosis in patients with respiratory symptoms and imaging consistent with COVID-19. Ground-glass opacities are commonly associated with COVID-19 but are not pathognomonic and can occur in infectious, inflammatory, eosinophilic, and cardiogenic conditions [5]. A comprehensive history, focused laboratory investigation, and consideration of patient-specific risk factors remain essential.

Over-reliance on radiological findings alone can delay appropriate treatment. Clinicians should remain alert to diagnostic anchoring on COVID-19, particularly when the clinical course or investigation results do not support the initial impression [10]. These cases illustrate the continuing need to reassess the differential diagnosis when COVID-19-like respiratory presentations remain unexplained.

Data Availability Statement

The clinical data reported in this case series are presented within the manuscript and accompanying figures.

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Volume 15 Issue 11 November 2026

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