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Severe Fixed Airflow Obstruction and Air Trapping Consistent with Probable Obliterative Bronchiolitis in a Patient with Remote Bleomycin Pulmonary Toxicity and Systemic Vasculitis: A Case Report

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Abstract

Obliterative bronchiolitis is an uncommon small-airway disorder that is characterized by inflammatory and fibrotic narrowing of the membranous and respiratory bronchioles. Its diagnosis can be challenging because the symptoms are nonspecific, the radiological abnormalities may be subtle, and the pulmonary function abnormalities may be disproportionate to the findings on inspiratory computed tomography. Previous drug exposure, autoimmune disease, transplantation, infection, and toxic inhalational injury are recognized associations. The discordance between the relatively limited interstitial computed tomography abnormalities and the profound obstructive physiology with marked air trapping suggested that small-airway disease, rather than restrictive pulmonary fibrosis, was the dominant physiological process. This case illustrates the importance of integrating pulmonary physiology, lung volumes, and thoracic imaging in patients with complex historical pulmonary insults and cautions against attributing late respiratory impairment solely to remote bleomycin exposure.

Keywords: Obliterative bronchiolitis; Constrictive bronchiolitis; Bleomycin pulmonary toxicity; Small-airway disease; Air trapping; Vasculitis

Introduction

Obliterative bronchiolitis, which is often used clinically interchangeably with constrictive bronchiolitis, is a rare disorder that is characterized by inflammation and concentric fibrosis of the small conducting airways, and this process results in progressive luminal narrowing or complete obliteration. Histologically, the process predominantly involves the membranous and respiratory bronchioles, and it differs fundamentally from the intraluminal fibroproliferative process that was historically termed bronchiolitis obliterans organizing pneumonia, which is now designated as cryptogenic organizing pneumonia when it occurs in an idiopathic form. Obliterative bronchiolitis is most firmly recognized following lung or hematopoietic stem-cell transplantation, although it has also been associated with autoimmune disorders, respiratory infections, toxic inhalational exposures, and selected drugs. Sporadic disease that occurs outside the transplant setting remains uncommon and diagnostically challenging. Progressive exertional dyspnoea and cough are the typical manifestations of this condition. Pulmonary function testing frequently demonstrates persistent airflow obstruction and air trapping, while the diffusing capacity may be normal or reduced. High-resolution computed tomography may demonstrate mosaic attenuation, expiratory air trapping, bronchial wall thickening, bronchiectasis, or bronchiolectasis. Importantly, small-airway disease can produce severe physiological

impairment despite relatively modest abnormalities that are visible on inspiratory computed tomography.

Bleomycin is well recognized for its potential to cause pulmonary toxicity. The classical manifestations are pneumonitis and subsequent pulmonary fibrosis, although a spectrum of histopathological patterns has been described. Consequently, the development of severe obstructive physiology many years after documented bleomycin pulmonary toxicity presents an important diagnostic challenge, particularly when other potential causes of airway disease coexist. We describe a patient with remote bleomycin-associated pulmonary toxicity and systemic vasculitis who subsequently presented with hypoxemic respiratory failure and was found to have profound fixed airflow obstruction and air trapping, with radiological features that raised the possibility of obliterative bronchiolitis [1-16].

Case Presentation

A 53-year-old woman presented to the emergency department on 31 July 2026 with a five-day history of progressively worsening shortness of breath. Her symptoms were predominantly exertional in nature and were associated with a dry cough, occasional white sputum production, and wheezing. Cyanosis of the lips had been observed by her family members. She denied having any chest pain. She had also experienced swelling of the hands in the context of her known systemic vasculitic disorder and reported having had two episodes of haemoptysis during the broader clinical course, although none had occurred during the immediate presenting illness. Her medical history was notable for Hodgkin lymphoma that had been diagnosed at approximately 21 years of age, and this had been treated with a bleomycin-containing chemotherapy regimen. The historical medical record documented bleomycin-associated pulmonary toxicity during her oncological treatment, and this had been characterized by respiratory symptoms and radiographic abnormalities that had responded to corticosteroid therapy. Her Hodgkin lymphoma subsequently entered long-term remission and remained in remission at the time of presentation. Her additional medical history included biopsy-proven leukocytoclastic vasculitis or polyarteritis nodosa, which had been diagnosed in her forties following a skin biopsy that demonstrated leukocytoclastic vasculitis. The clinical features had included cutaneous manifestations, arthralgias, and peripheral neuropathy, with systemic involvement that was consistent with polyarteritis nodosa. She had also had a previous right breast carcinoma that had been treated with a right mastectomy, and there was no current evidence of recurrence. She had chronic hepatitis B that was managed with appropriate antiviral therapy, type 2 diabetes mellitus that was well-controlled with oral hypoglycemic agents, and radiological emphysema that had been documented on prior thoracic imaging. She had also experienced a large pleural effusion during early adulthood that was estimated at approximately 1.5 L and had required therapeutic thoracentesis, although the etiology remained unclear and the condition had resolved without sequelae.

She had a substantial cigarette-smoking history, having commenced smoking at a young age, with an estimated 30 pack-year history. She had ceased smoking approximately three to four years before the current presentation. There was no history of occupational or environmental exposures to known respiratory toxicants. On presentation, her vital signs demonstrated sinus tachycardia at 112 beats per minute, a blood pressure of 138/82 mmHg, a respiratory rate of 22 breaths per minute, and an oxygen saturation of 88% on room air, which improved to 94% with supplemental oxygen at 2 L per minute via nasal cannula. Her temperature was 37.1°C. The initial laboratory investigations demonstrated mildly elevated inflammatory markers, with a C-reactive protein of 24 mg/L where the reference value is less than 5 mg/L, and an erythrocyte sedimentation rate of 38 mm/h where the reference value is less than 20 mm/h. The high-sensitivity troponin I was elevated at approximately 510 ng/L where the reference value is less than 34 ng/L. The complete blood count showed a hemoglobin of 12.8 g/dL, a white blood cell count of 9.4 × 10^9/L, and a platelet count of 312 × 10^9/L. The renal function tests showed a creatinine of 82 μmol/L and a urea of 5.6 mmol/L. The liver function tests were within normal limits, and the serum electrolytes were also within normal limits. Electrocardiography demonstrated sinus tachycardia without ischemic changes. Nasopharyngeal testing for SARS-CoV-2, influenza A and B, and respiratory syncytial virus was negative. The patient was admitted with acute hypoxemia for further respiratory and cardiovascular evaluation.

Diagnostic Assessment

Thoracic Imaging

The notable findings included subtle interstitial septal thickening and traction bronchiectasis that involved the medial aspects of both upper lobes and the superior segments of both lower lobes. There was also bilateral bronchial wall thickening and mucus plugging within the segmental and subsegmental bronchi. Minor plate-like atelectatic changes were present within the medial basal segments of the lower lobes. There was no pulmonary mass, pleural effusion, or significant supraclavicular, axillary, mediastinal, or hilar lymphadenopathy. The overall radiological appearances were reported as being suggestive of obliterative bronchiolitis, with the combination of bronchial wall abnormalities, mucus plugging, and bronchiectatic change raising the possibility of a chronic small-airway process that was superimposed on limited interstitial or fibrotic abnormalities. It is important to note that the available study was a computed

tomography pulmonary angiogram protocol rather than a dedicated inspiratory-expiratory high-resolution computed tomography protocol. Consequently, expiratory imaging was not obtained, and this limited the assessment of dynamic air trapping.

Pulmonary Function Testing

Comprehensive pulmonary function testing was performed on 5 August 2026 and demonstrated a striking obstructive physiological phenotype. The pre-bronchodilator spirometry showed an FEV_1 of 0.87 L, which was 33.6% of the predicted value, and an FVC of 2.07 L, which was 63.9% of the predicted value, producing an FEV_1/FVC ratio of 0.42. The mid-expiratory flow was profoundly reduced, with an $FEF_{25–75}$ of 0.23 L per second, which was 9.4% of the predicted value. Following the administration of 400 µg of inhaled salbutamol, the FEV_1 increased to 1.02 L, which was 39.6% of the predicted value, and the FVC increased to 2.27 L, which was 70.2% of the predicted value, with an FEV_1/FVC ratio of 0.45. The mid-expiratory flow increased to 0.30 L per second, which was 12.2% of the predicted value. The increase in FEV_1 was 150 mL, and when this was expressed relative to the predicted FEV_1, it represented approximately 5.8%. This did not satisfy the contemporary ERS/ATS criterion for a significant bronchodilator response, which requires a change of more than 10% of the predicted value. The static lung volumes were particularly informative. The total lung capacity was 4.58 L, which was 91.8% of the predicted value, and this indicated preserved total lung capacity and argued against a dominant restrictive ventilatory defect. In marked contrast, the residual volume was 3.27 L, which was 174.1% of the predicted value, and the RV/TLC ratio was 58.2%, which demonstrated severe air trapping. The functional residual capacity was also increased at 125% of the predicted value. The gas transfer was moderately impaired, with a DLCO of 9.38 mL per minute per mmHg, which was 47.4% of the predicted value, and a KCO of 68.1% of the predicted value. Parameter Pre-Bronchodilator Post-Bronchodilator % Predicted / Interpretation (Table 1).

Table 1: *Comprehensive Pulmonary Function Findings.*

Parameter	Pre-bronchodilator	Predicted	Post-bronchodilator	Predicted	Interpretation
FEV1	0.87L	33.6	1.02L	39.6%	
FVC	2.07L	63.9%	2.27L	70.2%	
FEV_1/FVC	0.42		0.45		Severe obstruction
$FEF_{25–75}$	0.23L	9.4%	0.30L	12.2%	
TLC	4.58L	91.8%			
RV	3.27L	174.1%			
RV/TLC		58.2%			Markedly elevated
DLCO	9.38L	47.4%			
KCO	2.94L	68.1%			
Abbreviations: BD, bronchodilator; FEV_1, forced expiratory volume in 1 second; FVC, forced vital capacity; $FEF_{25–75}$, forced expiratory flow at 25-75% of vital capacity; TLC, total lung capacity; RV, residual volume; FRC, functional residual capacity; DLCO, diffusing capacity of the lung for carbon monoxide; KCO, carbon monoxide transfer coefficient.					

Taken together, these findings demonstrated severe airflow obstruction with profound air trapping, preserved total lung capacity, and moderately reduced gas transfer, and this pattern was consistent with significant small-airway disease.

Cardiovascular Assessment

Transthoracic echocardiography demonstrated normal left ventricular dimensions and preserved systolic function, with a Simpson biplane left ventricular ejection fraction of approximately 62%. The right ventricular size was normal, although the systolic indices were mildly reduced. There was no hemodynamically significant valvular abnormality. The inferior vena cava was normal in size with inspiratory collapse, and the estimated right atrial pressure was approximately 8 mmHg. The preserved left ventricular systolic function and the absence of major structural cardiac disease made cardiac failure an unlikely explanation for the degree of respiratory physiological impairment. The elevated troponin in the setting of acute hypoxemia and tachycardia was considered to be compatible with acute myocardial injury or demand ischemia; however, a primary acute coronary syndrome could not be entirely excluded based on the available data.

Nocturnal Oximetry

Overnight pulse oximetry subsequently demonstrated a mean SpO_2 of 93.1%, with approximately 104.1 minutes, which represented 17.6% of the valid recording time, being spent below an SpO_2 of 90%. The recorded minimum SpO_2 was 29.8%,

although given the extreme value and the nature of unattended pulse oximetry, this isolated nadir was considered to be potentially artifactual and was not interpreted in isolation. Nevertheless, the prolonged cumulative time below 90% supported clinically relevant nocturnal oxygen desaturation. Formal sleep evaluation was subsequently arranged for the possible initiation of nocturnal supplemental oxygen therapy.

Differential Diagnosis

The principal diagnostic challenge was determining whether the patient's respiratory impairment represented late fibrotic sequelae of bleomycin toxicity, smoking-related COPD or emphysema, bronchiolar disease related to systemic inflammation, or a combination of these processes. The computed tomography abnormalities initially raised concern regarding a fibrotic process because of the presence of interstitial septal thickening and traction bronchiectasis. However, the pulmonary physiology demonstrated preserved total lung capacity with a disproportionately elevated residual volume and RV/TLC ratio, together with severe airflow obstruction. This pattern was difficult to reconcile with pulmonary fibrosis as the dominant physiological process, as a fibrotic process would typically reduce the total lung capacity and produce a restrictive ventilatory defect. The patient had a substantial smoking history of approximately 30 pack-years and documented emphysema, and smoking-related COPD could clearly contribute to the severe obstruction, air trapping, and reduced DLCO. Furthermore, air trapping is not specific for obliterative bronchiolitis, as asthma, COPD, bronchiectasis, and other small-airway diseases may produce similar physiological and radiological abnormalities. However, the radiologist independently identified bronchial wall thickening and mucus plugging as being suggestive of obliterative bronchiolitis, while the severe air trapping and relatively fixed obstruction provided physiological corroboration of substantial small-airway dysfunction that extended beyond typical COPD.

The patient's biopsy-proven polyarteritis nodosa or leukocytoclastic vasculitis provided another potential contributor. Although clinically overt pulmonary involvement is considered to be uncommon in polyarteritis nodosa, pathological studies have demonstrated pulmonary vascular abnormalities more frequently than had been previously appreciated. In one autopsy series of ten patients with polyarteritis nodosa, bronchial arteritis was found in seven patients, and diffuse alveolar damage and interstitial fibrosis were also described. Given the available evidence, we formulated a working diagnosis of probable obliterative bronchiolitis that was superimposed on smoking-related emphysema, with possible contributions from remote bleomycin pulmonary toxicity and systemic vasculitis. The term "probable" was intentional, as it acknowledged the absence of histopathological confirmation.

Discussion

The principal value of this case lies not simply in the presence of severe airflow obstruction, but in the discordance between the apparent radiological interstitial abnormalities and the physiological phenotype. This discordance highlights the importance of comprehensive pulmonary function testing in patients with complex respiratory histories and underscores the diagnostic challenges that are inherent in identifying small-airway disease.

Obliterative Bronchiolitis as a Small-Airway Disorder

Constrictive or obliterative bronchiolitis is characterized pathologically by peribronchiolar inflammation and concentric fibrosis that results in the narrowing or obliteration of the bronchiolar lumina. Because the disease primarily affects airways that are less than approximately 2 mm in diameter, abnormalities can be difficult to identify directly on conventional computed tomography. Indirect signs therefore become critically important. Physiologically, obliterative bronchiolitis typically produces airflow obstruction and air trapping. Bronchiectasis and bronchiolectasis may occur as later manifestations, and the DLCO can be either preserved or reduced, depending on the presence of coexisting parenchymal involvement. The current patient demonstrated several of these features, including an FEV_1 of only 33.6% of the predicted value, an FEV_1/FVC ratio of 0.42, a profoundly reduced mid-expiratory flow with an $FEF_{25–75}$ of 9.4% of the predicted value, and a striking elevation of the residual volume at 174% of the predicted value and an RV/TLC ratio of 58.2%. The most informative observation was arguably the preserved total lung capacity of 91.8% of the predicted value despite a residual volume of 174% of the predicted value. This provided objective physiological evidence that gas trapping, rather than loss of total lung volume, was a major determinant of the reduced FVC.

Importance of Expiratory High-Resolution Computed Tomography

Small-airway disorders present a particular imaging challenge. Direct visualization of diseased bronchioles may not be possible because normal small airways are below the spatial resolution of routine computed tomography. Indirect abnormalities therefore become important. Mosaic attenuation on inspiratory imaging and, particularly, air trapping on expiratory high-resolution computed tomography is recognized imaging manifestations of obliterative bronchiolitis. Expiratory computed tomography may reveal substantial small-airway dysfunction even when the inspiratory imaging is relatively unremarkable. In one study, expiratory computed tomography demonstrated air trapping in patients with obliterative bronchiolitis who had normal

inspiratory computed tomography scans. In the present case, the available computed tomography pulmonary angiogram was not a dedicated inspiratory-expiratory small-airway protocol. Consequently, the physiological evidence of severe air trapping is stronger than the available radiological demonstration of expiratory air trapping. This represents both an important limitation and a potential next diagnostic step. A dedicated inspiratory and expiratory high-resolution computed tomography would better characterize the extent and distribution of the small-airway disease (Figure 1).

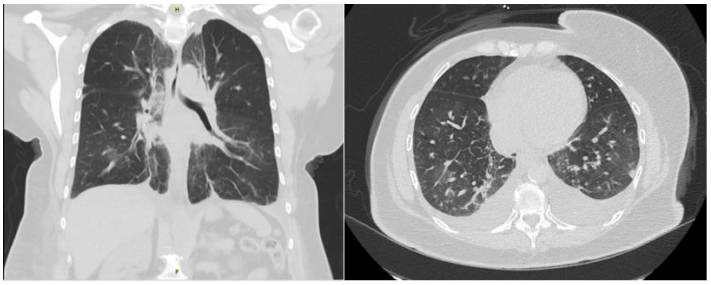


Figure 1*: HRCT (left coronal view and right axial view) demonstrating traction bronchiectasis, diffuse bronchial wall thickening, mosiac interseptal lobular thciekning, small bilateral effusions.*

Could This Simply Represent COPD?

This is an important competing diagnosis. The patient had a substantial previous smoking history of approximately 30 pack-years and documented emphysema, and smoking-related COPD could clearly contribute to the severe obstruction, air trapping, and reduced DLCO. Furthermore, air trapping is not specific for obliterative bronchiolitis. Asthma, COPD, bronchiectasis, and other small-airway diseases may produce similar physiological and radiological abnormalities. In a systematic review of sporadic obliterative bronchiolitis, De and colleagues cautioned against overdiagnosis based solely on nonspecific air trapping because asthma, smoking-related disease, and bronchiectasis can mimic obliterative bronchiolitis. Accordingly, we cannot attribute the entire physiological defect to obliterative bronchiolitis. However, several factors support the diagnosis. The radiologist independently identified bronchial wall thickening and mucus plugging as being suggestive of obliterative bronchiolitis. The severe air trapping and relatively fixed obstruction, with an insignificant bronchodilator response according to the ERS/ATS criteria, provided physiological corroboration of substantial small-airway dysfunction. The presence of multiple potential causes of bronchiolar disease, including bleomycin exposure, systemic vasculitis, and smoking, increased the pretest probability. We therefore consider probable obliterative bronchiolitis that is superimposed on smoking-related emphysema to be a more defensible formulation than either diagnosis alone.

The Bleomycin Question

The patient's remote bleomycin exposure is particularly intriguing. Bleomycin pulmonary toxicity is a well-established complication of treatment for Hodgkin lymphoma and germ-cell malignancies. Pulmonary injury occurs in approximately 10% of exposed patients in some series, although the incidence varies according to patient characteristics, cumulative exposure, and treatment regimen. Crucially, this patient had not merely received bleomycin; her historical clinical record documented pulmonary toxicity that was attributed to bleomycin, and this had been characterized by respiratory symptoms and radiographic abnormalities that had responded to corticosteroids. This history establishes significant pulmonary susceptibility to bleomycin-related injury. Nevertheless, the present physiology is not characteristic of advanced fibrotic interstitial lung disease as the sole explanation. A dominant fibrotic process would ordinarily be expected to reduce the lung volumes, whereas the total lung capacity was preserved and the residual volume was profoundly elevated. The remote history of bleomycin pulmonary toxicity may explain at least some of the interstitial thickening and traction bronchiectasis. However, the current evidence is insufficient to establish bleomycin as the cause of the probable obliterative bronchiolitis. We therefore consider remote bleomycin-related pulmonary injury to be an important contributor to the patient's complex pulmonary phenotype rather than a proven cause of her current bronchiolar disease.

Potential Contribution of Systemic Vasculitis

The patient's biopsy-proven polyarteritis nodosa or leukocytoclastic vasculitis provides another potential contributor. Classically, clinically overt pulmonary involvement is considered to be uncommon in polyarteritis nodosa. Nevertheless, pathological studies have demonstrated pulmonary vascular abnormalities in polyarteritis nodosa more frequently than had been previously appreciated. In one autopsy series of ten patients with polyarteritis nodosa, Matsumoto and colleagues found bronchial arteritis in seven patients, which represented 70% of the cases, and diffuse alveolar damage and interstitial fibrosis were also described. There is insufficient evidence in this patient to establish vasculitis as the cause of her bronchiolar disease. Nonetheless, the coexistence of systemic inflammatory disease expands the differential diagnosis beyond conventional smoking-related COPD and supports the consideration of immune-mediated airway injury as a contributing factor.

Why Biopsy Was Not Required to Formulate the Working Diagnosis

The term "probable" is intentional. The histological diagnosis of constrictive bronchiolitis can be difficult because the disease distribution may be patchy, and this reduces the sensitivity of transbronchial biopsy. Surgical lung biopsy provides substantially larger tissue samples but carries procedural morbidity, particularly in patients with severe physiological impairment such as this patient, who had an FEV_1 of 33.6% of the predicted value. Conversely, a systematic review of sporadic obliterative bronchiolitis cautioned against overdiagnosis based solely on nonspecific air trapping because asthma, smoking-related disease, and bronchiectasis can mimic obliterative bronchiolitis. For this reason, the available evidence supports probable rather than histologically confirmed obliterative bronchiolitis. The diagnostic approach in this case followed the principle that in patients with severe physiological impairment and multiple competing diagnoses, a working clinical diagnosis that is supported by comprehensive physiological and imaging data may be sufficient to guide management, particularly when invasive diagnostic procedures carry significant risk.

Clinical Implications and Management Considerations

This case has several important clinical implications.

- First, regarding the diagnostic approach, patients with complex respiratory histories require comprehensive pulmonary function testing that includes static lung volumes, not just spirometry. The preserved total lung capacity in the face of severe obstruction and air trapping was the key observation that redirected diagnostic thinking away from predominant fibrosis and toward small-airway disease.
- Second, regarding therapeutic considerations, the fixed nature of the airflow obstruction, as indicated by the insignificant bronchodilator response, suggests limited responsiveness to conventional bronchodilator therapy. Management should therefore focus on supplemental oxygen for nocturnal hypoxemia, avoidance of respiratory irritants, vaccination against influenza and pneumococcus, pulmonary rehabilitation, and monitoring for disease progression with serial pulmonary function tests.
- Third, regarding prognostic implications, obliterative bronchiolitis generally carries a poor prognosis, with many patients experiencing progressive decline in lung function. The presence of multiple potential contributing factors, including bleomycin toxicity, vasculitis, and smoking, may confer an even poorer prognosis.
- Fourth, regarding the need for multidisciplinary management, given the complexity of this patient's presentation, which involved hematologic, rheumatologic, and pulmonary considerations, multidisciplinary management that involves pulmonologists, rheumatologists, and other specialists is essential.

Study Limitations

Several limitations should be acknowledged. The absence of histopathological confirmation means that the diagnosis of obliterative bronchiolitis is "probable" rather than confirmed by lung biopsy. However, as discussed, the risks of invasive biopsy in this patient with severe obstruction were deemed to outweigh the potential benefits. The incomplete imaging protocol is another limitation, as the available computed tomography was a pulmonary angiography protocol rather than a dedicated inspiratory-expiratory high-resolution computed tomography protocol. Expiratory imaging would have provided additional evidence of air trapping and may have better characterized the extent and distribution of the small-airway disease. The absence of comprehensive bronchoscopic evaluation is also a limitation, as bronchoscopy with bronchoalveolar lavage was not performed, and this might have provided additional information regarding infectious or inflammatory etiologies.

The retrospective nature of this case report is another limitation, and some historical details, particularly regarding the original bleomycin toxicity, could not be fully verified from the available records. Finally, as a single case report, the findings may not be generalizable to other patients with similar presentations. Despite these limitations, this case provides valuable clinical insights regarding the diagnostic approach to complex small-airway disease.

Conclusion

This case illustrates severe chronic small-airway dysfunction in a patient with several potential pulmonary insults, including previous smoking, emphysema, systemic vasculitis, and remote documented bleomycin pulmonary toxicity. Although the computed tomography demonstrated interstitial thickening and traction bronchiectasis, the pulmonary physiology revealed a strikingly different dominant phenotype that consisted of severe relatively fixed airflow obstruction, profound air trapping, and preserved total lung capacity. The integration of imaging and physiological findings supported a working diagnosis of probable obliterative or constrictive bronchiolitis that was superimposed on smoking-related lung disease rather than advanced pulmonary fibrosis alone. The case emphasizes that radiological fibrosis should not automatically be equated with physiologically dominant restrictive lung disease. Careful assessment of the static lung volumes, particularly the residual volume, the RV/TLC ratio, and the total lung capacity, can fundamentally alter the diagnostic interpretation in complex small-airway disease.

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Follow-up Formal sleep assessment was arranged, and ongoing respiratory evaluation was planned.

Learning Points

- Severe small-airway disease may produce profound physiological impairment despite relatively modest abnormalities on inspiratory computed tomography.
- A markedly increased residual volume and RV/TLC ratio with a preserved total lung capacity strongly supports air trapping and can help identify an obstructive rather than predominantly restrictive physiological phenotype.
- Expiratory high-resolution computed tomography is particularly valuable when obliterative bronchiolitis is suspected because expiratory air trapping may be more conspicuous than abnormalities on inspiratory imaging.
- A history of bleomycin pulmonary toxicity should prompt consideration of treatment-related lung disease, but subsequent respiratory abnormalities should not automatically be attributed to bleomycin fibrosis.
- In patients with smoking exposure, systemic inflammatory disease, and previous pneumotoxic therapy, the causality may remain multifactorial, and distinguishing the physiological phenotype from the presumed etiology is essential.
- The diagnosis of "probable" obliterative bronchiolitis may be appropriate when the clinical, physiological, and radiological evidence supports the diagnosis but invasive confirmation carries significant risk.

Declarations

Patient Consent: Written informed consent for the publication of this case report and any accompanying images was obtained from the patient.

Conflict of Interest: The authors declare that they have no conflicts of interest.

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Author Contributions: Pranav Kumar: Conceptualisation of the case report; clinical management of the patient; interpretation of radiological, pathology, and microbiological findings; literature review; manuscript preparation and critical revision.

Ashley Fisser: contributed to interpretation of radiological findings, assisting with clinical management, and writing of the case report. Malindi Wijayasiri, contributed to writing of the manuscript and clinical management of the patient.

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