

Anthracofibrosis Presenting as Middle Lobe Syndrome in a Patient With Difficult-to-Treat Asthma: A Case Report

Antracofibrosis como síndrome del lóbulo medio en paciente con asma de difícil control: reporte de caso

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ABSTRACT

Background: Middle lobe syndrome (MLS) is characterized by recurrent or persistent collapse of the right middle lobe, secondary to bronchial obstruction. Anthracofibrosis (AF), which can cause bronchial stenosis through anthracotic pigment deposition and fibrosis, may produce this obstruction and mimic refractory asthma.

Objective: To describe a case of MLS secondary to AF in a patient with difficult-to-treat asthma.

Case report: A 69-year-old man who had never smoked and lived in a rural area; with a history of poorly controlled asthma, multiple exacerbations, and persistent middle lobe (ML) atelectasis. The CT showed atelectasis and traction bronchiectasis. Flexible bronchoscopy (FBC) revealed marked stenosis of the ML bronchus with architectural distortion. Due to a poor response to medical management, a video-assisted thoracoscopic surgery (VATS) middle lobectomy was performed. Pathologic findings revealed nodal anthracosis.

Conclusion: AF should be included in the differential diagnosis of MLS and difficult-to-treat asthma in patients with chronic biomass smoke exposure or those residing in rural areas.

Key word: middle lobe syndrome anthracosis bronchial Stenosis asthma

RESUMEN

Introducción: El síndrome del lóbulo medio consiste en el colapso recurrente o persistente del lóbulo medio por obstrucción bronquial. La antracofibrosis, al generar estenosis por depósito antracótico y fibrosis, puede producir esta obstrucción y simular un cuadro de asma refractaria.

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Objetivo: Describir un caso clínico de síndrome del lóbulo medio secundario a antracofibrosis en un paciente con asma de difícil control.

Caso clínico: Paciente varón de 69 años, no tabaquista, residente en una zona rural, con antecedente de asma de mal control, múltiples exacerbaciones y atelectasia persistente del lóbulo medio. La tomografía evidenció atelectasia y bronquiectasias por tracción. La fibrobroncoscopia mostró estenosis marcada del bronquio del lóbulo medio con alteración de la arquitectura. Ante la mala respuesta al tratamiento médico, se realizó una lobectomía media por videotoracosopia. La anatomía patológica informó antracosis ganglionar.

Conclusión: La antracofibrosis debe incluirse en el diagnóstico diferencial del síndrome del lóbulo medio y del asma de difícil control en pacientes con exposición crónica a biomasa o residentes en áreas rurales.

Palabras clave: síndrome del lóbulo medio antracosis estenosis bronquial asma

INTRODUCTION

Middle lobe syndrome (MLS), historically referred to as Lady Windermere syndrome, is characterized by recurrent or persistent atelectasis of the right middle lobe. It can result from extrinsic or intrinsic bronchial obstruction or from functional and anatomical alterations leading to impaired drainage and collapse.¹⁻⁶

Exposure to particulate matter from biofuel smoke (BSPM) and environmental pollutants (EPs) is a direct cause of health problems. A systematic analysis from the Global Burden of Disease Study (1990–2010) identified pollution as one of the top 10 risk factors for impaired health in developing countries.⁷⁻⁹

Anthracosis is not a disease in itself: first described by Cohen in 1951, the term describes black pigmentation of the tracheobronchial tree involving the mucosal, submucosal, and parenchymal layers. When anthracosis is associated with mucosal proliferation and fibrosis that narrow and distort the bronchial lumen –potentially leading to atelectasis of the corresponding parenchyma– it is termed bronchial anthracofibrosis (AF), an entity first described by Chung et al in 1998.^{10,11}

In recent years, interest in recognizing its clinical relevance has increased, as anthracosis is frequently an incidental finding during bronchoscopic evaluation of patients with nonspecific symptoms such as cough, dyspnea, sputum production, and wheezing. Although more frequently

described in Asian countries, its recognition is increasing in other regions.

The etiology remains unknown. However, several hypotheses suggest that prolonged exposure to biomass fuel emissions, genetic predisposition, chronic inflammatory responses, outdoor and household air pollution, and chronic infections such as tuberculosis (TB) play a role in the pathogenesis of this condition.^{4,5,7,12-18}

This case demonstrates how AF can mimic refractory asthma and cause MLS, leading to delayed diagnosis and prolonged, ineffective treatment.¹⁹⁻²¹

CASE REPORT

A 69-year-old man, non-smoker, presented with a history of difficult-to-treat asthma, chronic pansinusitis, Barrett esophagus, dyslipidemia, and obstructive sleep apnea, treated with continuous positive airway pressure (CPAP), formoterol/budesonide/glycopyrronium, nasal fluticasone, montelukast, esomeprazole, simethicone, and rosuvastatin. His medical history was also notable for posterior subcapsular cataract and open-angle glaucoma with keratitis, treated with topical ophthalmic medications, as well as surgery for retinal detachment of the left eye. He resides in a rural area with potential biomass smoke exposure.

The patient had frequent consultations for bronchospasm and acute asthma exacerbations requiring multiple courses of oral and intravenous corticosteroids. He was referred by Ophthalmolo-

gy for bilateral chronic open-angle glaucoma and posterior subcapsular cataract, which could not be surgically managed because of persistent cough.

Physical examination: weight, 91 kg; height, 176 cm. Body mass index, 29.4 kg/m². Pulse oximetry, 98%. Generalized hypoventilation with bilateral rhonchi and wheezing, accompanied by persistent cough with mucoid sputum. Asthma Control Test (ACT) score, 7 points, consistent with very poorly controlled asthma.

Previous studies: craniofacial CT: pansinusitis.

Chest CT scan: consolidations with air bronchograms. Tree-in-bud opacities in the medial segment of the middle lobe (ML). Adjacent ground-glass opacity in the posterior basal segment of the right lower lobe (RLL). Bronchial wall thickening. The mediastinum is midline, with prominent lymphadenopathy at station 4R, the subcarinal station, and the right hilum.

Esophagogastroduodenoscopy (EGD): Barrett esophagus. Treated with esomeprazole, 40 mg/day, and an aluminum hydroxide/simethicone preparation. Repeat EGD 3 years later: Barrett esophagus, erosive antritis, and a gastric fundic polyp. *Helicobacter pylori* was not detected. Polypectomy: hyperplastic polyp.

Chest CT angiography: no evidence of pulmonary embolism. ML atelectasis.

Obstructive sleep apnea: nocturnal positive airway pressure therapy for the past 4 years, with good adherence, at a pressure of 8.5 cm H₂O. Apnea-hypopnea index, 1.8 events/h.

Diagnostic workup

Chest radiograph: middle lobe atelectasis and right lower lobe opacity. (Fig. 1).

• Chest CT: middle lobe atelectasis with traction bronchiectasis and centroacinar opacities. Slightly enlarged prevascular and subcarinal lymph nodes. (Fig.2).

• Complete pulmonary function test (PFT): FVC, 87%; FEV1, 83%; FEF (forced expiratory flow), 81%; FEV1/FVC 73%; SVC (slow vital capacity), 88%; DLCO (diffusing capacity for carbon monoxide), 21.97 (85% of predicted), within normal limits; no significant bronchodilator response. (Fig. 3). Alternative diagnoses to the previously reported diagnosis of severe asthma were considered.

• Laboratory findings: elevated IgE and initial eosinophilia. Cultures for common pathogens, mycobacteria, and fungi were negative. Relevant laboratory results were as follows: hematocrit, 39%; hemoglobin, 12.1 g/dL; white blood cell count, 7.8 × 10⁹/L (10.5% eosinophils); total IgE, 2,000 IU/mL; C-reactive protein (CRP), 15.3 mg/L; *Aspergillus fumigatus*-specific IgE, 0.41IU/mL (class 1, low); erythrocyte sedimentation rate, 19 mm/h; CRP, 30.26 mg/L. Arterial blood gas analysis showed pH of 7.56, PaO₂ of 104 mm Hg, PaCO₂ of 27 mm Hg, and oxygen saturation of 98%; all other values were within reference ranges. Viral serologies and rheumatologic testing were negative. Sputum cultures: Fungal cultures negative; mycobacterial cultures negative; acid-fast bacilli (AFB) smear negative; upper airway flora isolated.

Flexible bronchoscopy: larynx without lesions; intermediate bronchus showed reduced caliber with architectural distortion; scope could not be advanced further. Bronchoalveolar lavage (BAL) yielded purulent fluid. Biopsy revealed mucoid material; alveolar macrophages, 30%; lymphocytes, 70%.

Figure 1. Posterior-anterior and right lateral chest radiograph

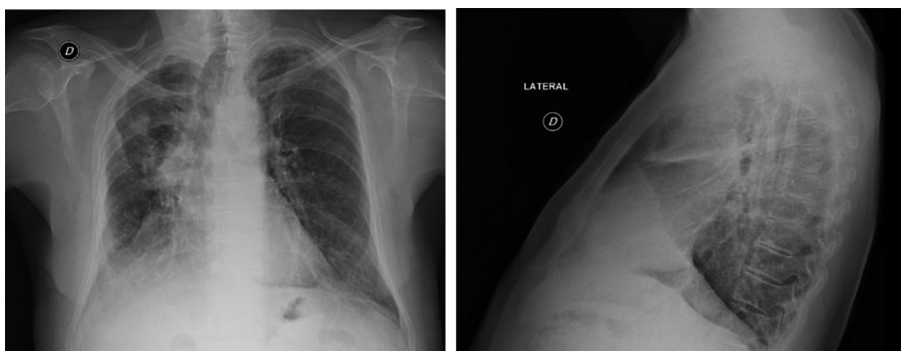
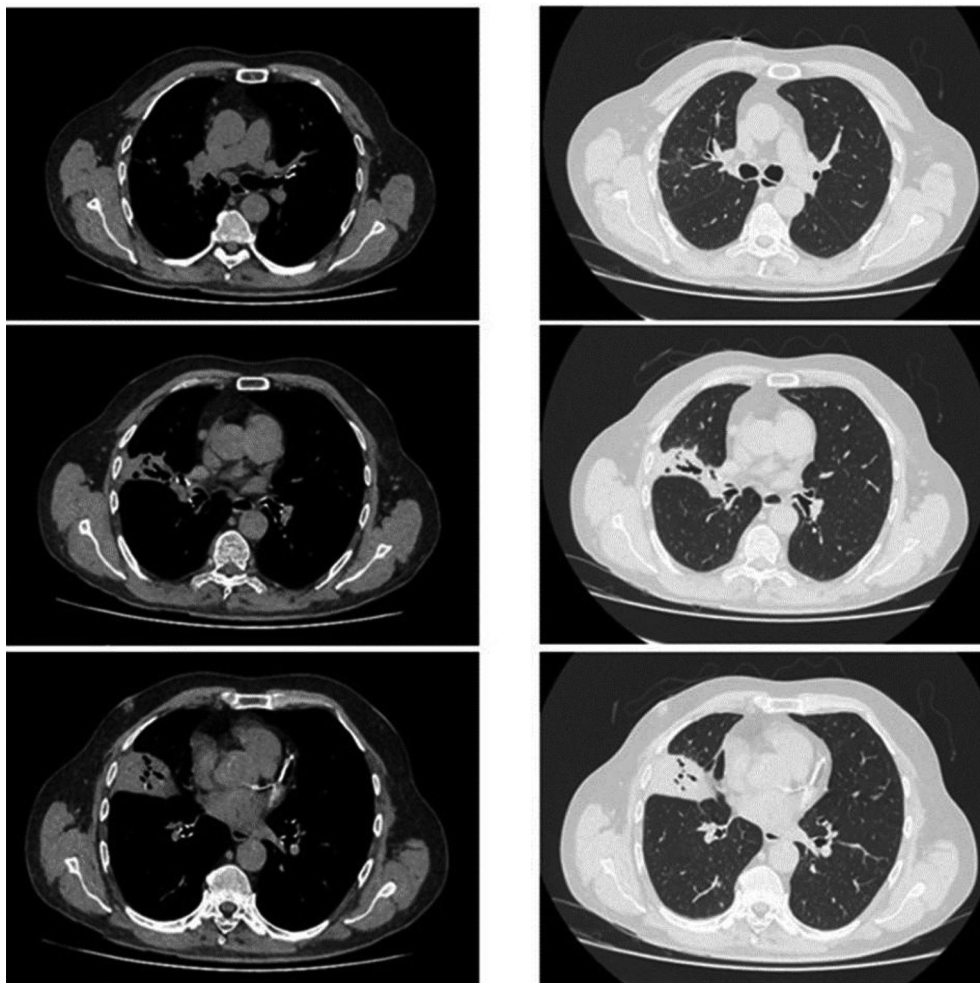


Figure 2. Chest CT: middle lobe atelectasis associated with traction bronchiectasis. Centroacinar opacities are present in the posterior basal segment of the right lower lobe, representing small-airway obstruction. The trachea and main bronchi are patent. Prevascular and subcarinal lymph nodes measure 10 mm and 11 mm, respectively.



The patient was treated with meprednisone and itraconazole. Due to the absence of microbiological isolation in cultures and an ophthalmology contraindication, oral corticosteroids were discontinued, and omalizumab was initiated.

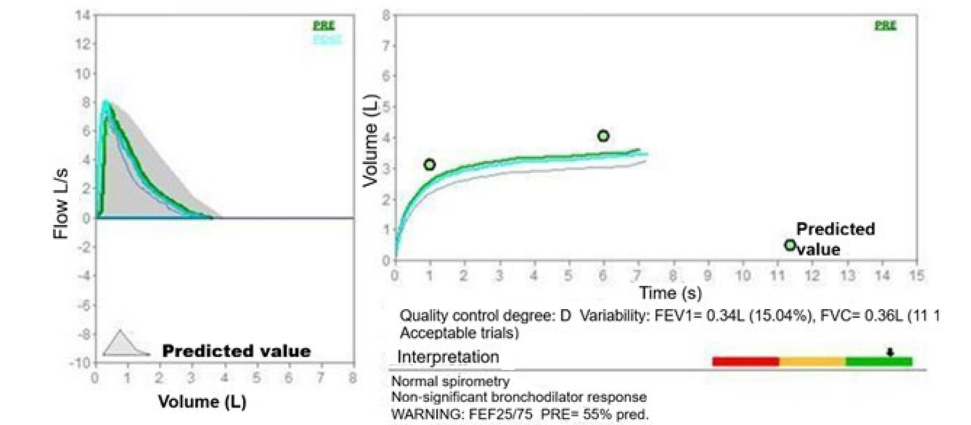
The patient experienced a torpid clinical course characterized by recurrent chronic cough, episodes of bronchospasm, and mild hemoptysis, with persistent atelectasis. Repeat flexible bronchoscopy revealed distorted airway architecture with cartilaginous excrescences resembling tracheobronchopathia osteochondroplastica. In the ML, there was cicatricial stenosis of the ostium with purulent secretions. Repeat biopsy and BAL were negative.

Due to lack of improvement and evidence of structural obstruction, a middle lobectomy

was performed via video-assisted thoracoscopic surgery (VATS). The postoperative course was uneventful, with no anesthetic complications and adequate pain control.

Pathologic findings: Gross examination: Two brown fragments measuring 0.7×0.3 cm in total. The pleura was smooth and glistening. On sectioning, there was increased parenchymal density with areas of bronchial dilation containing mucoid material. Microscopic examination: histologic sections showed lymph node fragments with anthracosis. Lung parenchymal fragments revealed dilated bronchi containing mucoid material and a dense inflammatory infiltrate involving the bronchial wall and peribronchial tissues, with areas of fibrosis and abscess formation. Microscopic examination: Lung, group 10, histopathologic

Figure 3. Spirometry with bronchodilator testing. Flow-volume and volume-time curves. The following parameters were measured: forced vital capacity (FVC), forced expiratory volume in the first second (FEV₁), FEV₁/FVC ratio, peak expiratory flow (PEF), and forced expiratory flow at 25% to 75% of FVC (FEF₂₅₋₇₅).



Test date PRE-bronchodilator 05/14/2024 13:33:41					POST- bronchodilator with salbutamol – 13:54:40							
Parameters		LLN	Predicted value	Best	% predicted	Z-score	PRE #1	PRE #2	PRE #3	POST	% predicted	% change
FVC	L	3.03	4.03	3.63*	90	-0.65	3.27	3.63		3.49*	87	-4
FEV1	L	2.27	3.11	2.60*	84	-0.99	2.26	2.60		2.46*	79	-5
FEV1/FVC	%	63.2	75.0	71.6*	96	-0.47	69.1	71.6		70.5*	94	-2
PEF	L/s	6.04	8.03	7.94*	99	-0.08	7.68	7.94		8.07*	100	2
ELA	years		68	85	125		97	85		90	132	6
FEF25-75	L/s	1.48	3.19	1.77	55	-1.36	1.35	1.77		1.60	50	-10
FET	s		6.00	7.03	117		7.23	7.03		7.27	121	3
FIVC	L	3.03	4.03									
FEV1/VC	%	63.2	75.0									

* Best values of all the curves – BTPS 1.097 24 °C (75.2 °F) – ERS (ECCS)/Knudson

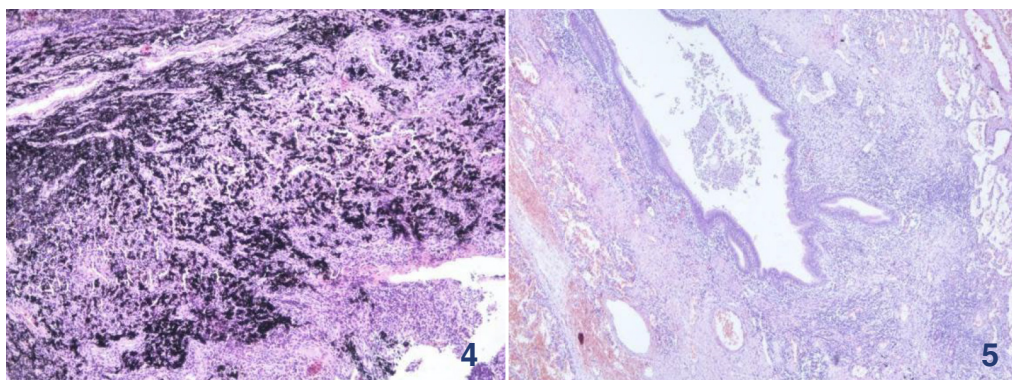
examination. Lymph node with anthracosis. Right middle lobe lung, histopathologic examination. Bronchiectasis with chronic inflammatory process and areas compatible with AF (Figures 4 and 5).

Postoperative non-contrast chest CT: Decreased right lung volume consistent with prior middle lobectomy, with surgical sutures noted at the hilar and fissural levels. There was diffuse bronchial wall thickening associated with a mild increase in centrilobular nodular opacities, some with ground-glass attenuation, likely of inflammatory nature. Biapical pleuroparenchymal scarring was present, sequela in appearance. Mild thickening of the right costal pleura in the upper lobe, involving the fissural plane, consistent with sequelae. Post-surgical changes were noted in the right lateral chest wall. No mediastinal or axillary lymphadenopathy was evident. The largest lymph nodes measured 9 mm in short-axis diameter.

No pleural or pericardial effusion was visualized.

Physical examination: the patient remained clinically stable. Weight: 88 kg; height: 176 cm; body mass index, 29.4 kg/m². Oxygen saturation, 98%. Respiratory examination revealed hypoventilation and isolated rhonchi at the left lung base. A right axillary surgical scar was present, without signs of inflammation and with good healing. Occasional episodes of cough with mucoid secretions persisted. Follow-up sputum analysis: non-representative sample; upper airway flora isolated; direct acid-fast bacilli (AFB) smear and cultures negative. On directed questioning, the patient reported living for more than 17 years in a house in the Paraná Delta region, heated with a wood-burning stove, where he resides and performs remote information technology work. He also reported exposure to smoke from grassland burning, a common practice in rural Delta areas.

Figure 4-5. Pathologic examination revealed a lymph node with anthracosis and lung parenchymal fragments showing dilated bronchi, mucoid material, and a dense inflammatory infiltrate involving the bronchial wall and peribronchial tissues, with areas of fibrosis and abscess formation. Histologic sections showed lymph node fragments free of lesions but with anthracosis (Fig. 4). Lung parenchymal fragments showed bronchiectasis with an inflammatory process, fibrosis, and abscess formation (Fig. 5).



The importance of enhancing biosafety measures and optimizing home ventilation systems was explained.

Follow-up spirometry (2 months postoperatively): FVC, 99%; FEV₁, 93%; FEV₁/FVC, 72%; FEF₂₅₋₇₅, 64%. Interpretation: Normal.

Follow-up echocardiography: systolic function preserved. The E/A ratio was preserved, indicating a normal relaxation pattern. No pathologic gradients were recorded. Mild tricuspid regurgitation allowed estimation of a systolic pulmonary artery pressure (sPAP) of 48 mm Hg.

Follow-up laboratory findings: hematocrit, 42%; hemoglobin, 12.7 g/dL; mean corpuscular volume (MCV), 73 fL; white blood cell count, 7.3×10^9 /L (neutrophils 46%, eosinophils 3%); platelet count, 188×10^9 /L; total IgE, 1,520 IU/mL. Hematology evaluation for anisocytosis, microcytosis, hypochromia, and polychromatophilia showed: reticulocyte count, 1.1%; serum iron, 46 µg/dL; transferrin, 242 mg/dL; ferritin, 78 ng/mL; hemoglobin electrophoresis: adult hemoglobin (HbA) 98%; fetal hemoglobin (HbF) not detectable; HbA₂ 2%; folate, 9.1 ng/mL; vitamin B₁₂ 374 pg/mL. Diagnosis: Alpha-thalassemia.

The patient underwent bilateral intraocular lens implantation with good tolerance and recovery.

DISCUSSION

This case highlights an unusual presentation of AF presenting clinically as MLS and initially misdiagnosed as difficult-to-treat asthma. Symptom

overlap represents one of the main diagnostic challenges.

Persistent cough, recurrent bronchospasm, frequent respiratory infections, and poor response to standard asthma therapy prompted investigation into underlying structural etiologies.

Chest CT demonstrated findings consistent with a chronic obstructive process.

Flexible bronchoscopy revealed marked stenosis of the ML bronchus, architectural distortion, and abundant purulent secretions, raising suspicion for an irreversible stenotic process.

Despite treatment with systemic corticosteroids, antifungal therapy, optimized asthma management, and omalizumab, the patient's protracted clinical course underscores that AF represents a mechanical and structural process rather than a purely inflammatory one, which doesn't respond to conventional therapy for asthma or allergic bronchopulmonary aspergillosis.

This phenomenon has been previously described in the literature, where patients initially managed for refractory asthma ultimately underwent multiple endoscopic and radiologic evaluations before receiving a diagnosis of AF.

The most widely accepted etiology involves chronic exposure to biomass smoke or environmental particulate matter, consistent with rural settings. Such exposure can induce chronic inflammation, anthracotic pigment deposition, and progressive fibrosis, leading to bronchial stenosis, atelectasis, and secondary bronchiectasis. The underlying pathophysiology remains a subject of

debate; not all patients present with a recognizable history of exposure, which further complicates the diagnostic process.

Anthracosis has a multifactorial etiology and is associated with an increased risk of cancer and tuberculosis (TB).^{8,13} Negative cultures, the absence of active TB, and the lack of visible endobronchial lesions suggested a noninfectious and non-neoplastic process, narrowing the differential diagnosis to tracheobronchopathia osteochondroplastica, chronic inflammation, or granulomatous diseases. Pathologic findings confirmed a lymph node with anthracosis and bronchiectasis accompanied by severe inflammation and fibrosis, findings consistent with AF.

There is no specific medical treatment to reverse the stenosis; lobectomy remains the definitive therapeutic option, consistent with published literature.

In the present case, chronic exposure to particulate matter from biomass combustion is a recognized risk factor for obstructive airway disease, bronchiectasis, and bronchial remodeling, supporting its potential role in the pathogenesis of AF.

Across published studies, the primary bronchoscopic findings are mucosal anthracotic pigmentation and bronchial narrowing, with no direct correlation between the degree of stenosis and the extent of anthracotic pigmentation. The middle lobe is most frequently affected, followed by the right upper and lower lobes. The FBC demonstrated a hard consistency of the lesion and a marked tendency to bleed following biopsy collection.¹⁸

This highlights the importance of considering AF in the differential diagnosis of MLS, particularly in older, non-smoking patients with poorly controlled asthma and chronic biomass exposure in rural settings.

Furthermore, it reinforces the diagnostic value of FBC and underscores the need to re-evaluate the diagnosis in patients managed for difficult-to-treat asthma who exhibit clinical features such as persistent atelectasis, non-obstructive spirometry, or a lack of response to biologic therapies when a structural component is present.

CONCLUSION

AF represents an uncommon yet clinically significant cause of chronic bronchial stenosis, airway obstruction, and MLS, particularly among rural residents with prolonged exposure to biomass smoke or other inhaled pollutants.

Its clinical presentation can mimic difficult-to-treat asthma or other chronic inflammatory airway diseases, contributing to diagnostic delays and prolonged, ineffective therapies.

This case emphasizes the importance of re-considering alternative diagnoses in patients with persistent respiratory symptoms, poor response to treatment, and structural imaging findings. Flexible bronchoscopy combined with histopathologic evaluation remains essential for the diagnosis.

AF should be included in the differential diagnosis of MLS and refractory asthma, particularly in non-smokers with significant environmental exposure history. Early recognition can prevent diagnostic delays, eliminate unnecessary treatments, and guide appropriate therapeutic strategies.

"As awareness of the respiratory impacts of chronic exposure to biomass increases, AF will likely be diagnosed more frequently in non-traditional patient populations."

Conflict of interest

Authors have no conflicts of interest to declare.

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