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Case report: Endobronchial mass in a young male with AA amyloidosis secondary to Crohn's disease

Abstract

AA amyloidosis, also known as secondary amyloidosis, is a systemic disorder resulting from chronic inflammatory conditions and characterized by extracellular deposition of Serum Amyloid A (SAA)-derived fibrils. Although renal involvement is most common, pulmonary manifestations are rare and may present diagnostic challenges, particularly when involving the endobronchial tree. Endobronchial amyloidosis is an uncommon presentation that can mimic malignancy and lead to significant respiratory morbidity.

We report the case of a 30-year-old male with a long-standing history of Crohn's disease who presented with progressive cough, hemoptysis, dyspnea, weight loss, and fatigue. Imaging studies, including chest X-ray and computed tomography, revealed a mass in the right main bronchus causing partial airway obstruction and distal lung consolidation. Bronchoscopic evaluation identified a polypoid endobronchial lesion. Histopathological examination of biopsy samples demonstrated amorphous eosinophilic deposits, and Congo red staining confirmed amyloid deposition by showing apple-green birefringence under polarized light. Immunohistochemistry identified serum amyloid A protein, establishing the diagnosis of AA amyloidosis secondary to chronic inflammation.

The patient was managed with supportive respiratory care and intensified treatment of the underlying Crohn's disease using immunosuppressive and biologic therapies. Over a three-month period, he showed marked clinical improvement, with resolution of respiratory symptoms and significant reduction in the size of the endobronchial lesion on follow-up imaging. Pulmonary function tests also demonstrated improvement.

This case highlights the importance of considering AA amyloidosis in the differential diagnosis of endobronchial masses, particularly in patients with chronic inflammatory diseases. Early recognition and histological confirmation are essential for appropriate management. Controlling the underlying inflammatory process remains the cornerstone of treatment and can lead to significant clinical improvement. Continuous monitoring is crucial due to the potential for recurrence or disease progression.

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Introduction

AA amyloidosis, also known as secondary amyloidosis, is a systemic disorder that develops as a consequence of prolonged inflammatory states [1]. It is characterized by the extracellular deposition of insoluble amyloid fibrils derived from Serum Amyloid A (SAA) protein, an acute-phase reactant synthesized by the liver in response to chronic inflammation [2-4]. Conditions such as Crohn's disease, rheumatoid arthritis, chronic infections, and other inflammatory disorders are commonly associated with sustained elevations of SAA levels. Over time, persistent overproduction of SAA leads to its misfolding and aggregation into amyloid fibrils, which deposit in tissues and disrupt normal organ function [5].

Among the various organs affected, the kidneys are most frequently involved, often presenting with proteinuria and progressive renal impairment [5,6]. However, AA amyloidosis can also involve the liver, spleen, gastrointestinal tract, and, less commonly, the respiratory system. Pulmonary manifestations of AA amyloidosis are relatively rare compared to other forms, such as AL amyloidosis, but they can pose significant diagnostic and clinical challenges when they occur. The respiratory involvement may present in several forms, including diffuse interstitial infiltration, parenchymal nodules, or tracheobronchial deposits [7,8].

Endobronchial amyloidosis, in particular, represents an uncommon manifestation of the disease but carries important clinical implications. In this form, amyloid deposits accumulate within the bronchial walls or lumen, potentially leading to airway narrowing or obstruction. Patients may present with nonspecific respiratory symptoms such as persistent cough, dyspnea, wheezing, or hemoptysis [9,10]. Because these symptoms overlap with more common respiratory conditions, including infections and malignancies, diagnosis is often delayed or initially misdirected [10].

Radiologic imaging, such as Computed Tomography (CT), may reveal endobronchial masses or airway irregularities, which can be mistaken for tumors. Bronchoscopic evaluation typically demonstrates nodular or plaque-like lesions within the bronchial tree. Definitive diagnosis relies on histopathological examination of biopsy specimens, where amyloid deposits appear as amorphous eosinophilic material. Confirmation is achieved using Congo red staining, which exhibits the characteristic apple-green birefringence under polarized light [11,12].

The presence of endobronchial involvement in AA amyloidosis underscores the systemic nature of the disease and highlights the importance of considering it in patients with chronic inflammatory conditions such as Crohn's disease [13]. Early recognition is crucial, as management strategies focus primarily on controlling the underlying inflammatory disorder to reduce SAA production and prevent further amyloid deposition [14,15]. In cases with significant airway obstruction, interventional bronchoscopic procedures may be necessary to alleviate symptoms and restore airway patency.

In summary, AA amyloidosis is a serious complication of chronic inflammatory diseases, driven by prolonged elevation of serum amyloid A protein. Although pulmonary involvement is rare, endobronchial manifestations can lead to considerable morbidity and mimic more common respiratory pathologies. Awareness of this potential presentation is essential for timely diagnosis and appropriate management, particularly in patients with known inflammatory conditions. This case report describes a young male patient with a history of Crohn's disease who presented with an endobronchial mass subsequently diagnosed as amyloidosis.

Case presentation

Patient information

A 30-year-old male with a known history of Crohn's disease for over ten years presented to the emergency department with complaints of progressive cough, hemoptysis, and dyspnea over the preceding month. He reported weight loss, night sweats, and fatigue but denied any significant respiratory symptoms prior to the recent exacerbation. He had previously been managed with corticosteroids and immunosuppressive therapy.

Clinical examination

On physical examination, the patient appeared moderately ill, with vital signs showing mild tachypnea (respiratory rate of 24 breaths per minute) and slight hypoxia (SpO_2 of 92% on room air). Auscultation revealed wheezing and decreased breath sounds in the right lung field. There was no lymphadenopathy or clubbing noted.

Diagnostic workup

Laboratory tests revealed mild anemia, elevated inflammatory markers (CRP and ESR), and normal renal function. A chest X-ray demonstrated a right-sided parenchymal opacity suggestive of an obstructive lesion. A subsequent CT scan of the chest showed a circumferential mass in the right main bronchus, causing partial obstruction and consolidation in the right lower lobe.

Bronchoscopy was performed, revealing an endobronchial mass obstructing the right main bronchus. The mass exhibited a smooth, polypoid appearance, measuring approximately 3 cm in diameter. Biopsy samples were obtained, and histopathological analysis was conducted.

The biopsy specimens displayed amorphous eosinophilic deposits and Congo red staining demonstrated characteristic apple-green birefringence under polarized light, confirming the diagnosis of amyloidosis. Immunohistochemical studies revealed the presence of serum amyloid A protein within the deposits, substantiating the diagnosis of AA amyloidosis secondary to chronic inflammation associated with Crohn's disease.

Management

The patient was started on supportive care, including bronchodilators and corticosteroids to alleviate respiratory symptoms. Additionally, treatment focused on managing underlying Crohn's disease with intensified immunosuppressive therapy, including azathioprine and biological agents targeting TNF-alpha.After three months of treatment, the patient showed marked clinical improvement, with resolution of cough and hemoptysis. A follow-up CT scan indicated significant reduction in the size of the endobronchial mass and improvement in lung function. Spirometry demonstrated normalizing patterns with improved Forced Expiratory Volume (FEV1) and Forced Vital Capacity (FVC).

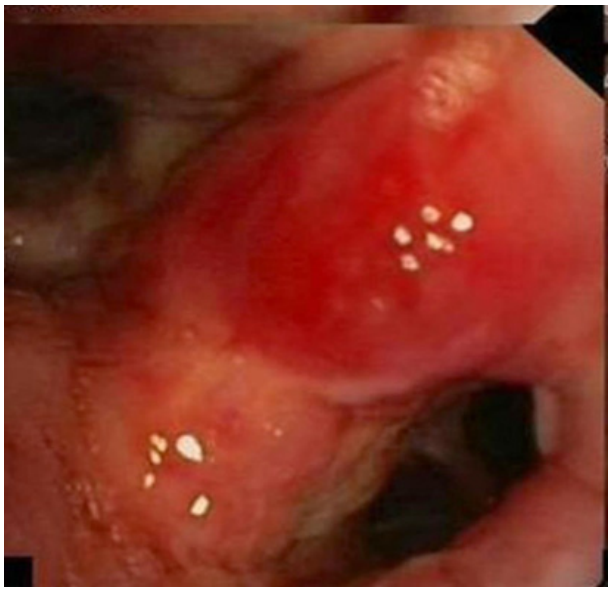

Figure 1: Endobroncial mass.

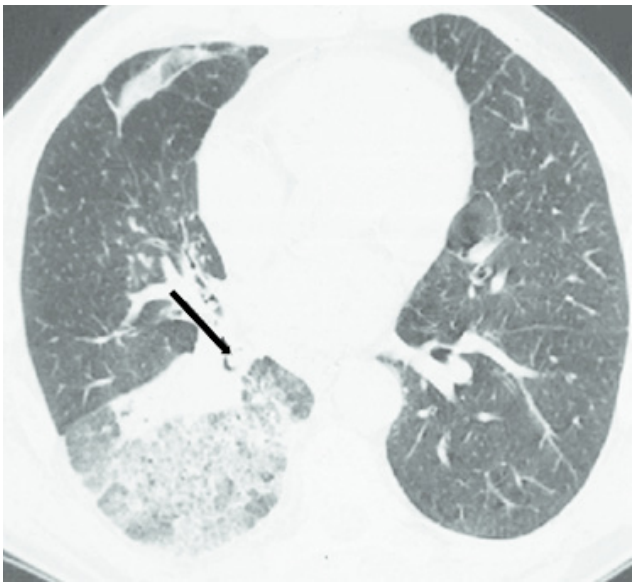

Figure 2: Endobroncial mass in chest CT scan.

Discussion

AA amyloidosis is a rare but serious complication of chronic inflammatory diseases such as Crohn's disease. It arises from prolonged elevation of serum amyloid A protein, an acute-phase reactant produced during sustained inflammation, which eventually deposits as insoluble fibrils in various tissues. While the kidneys are most commonly affected, pulmonary involvement is uncommon and may present unique diagnostic challenges. Among these, endobronchial amyloidosis is a particularly rare manifestation but can result in significant respiratory complications due to airway obstruction.

Endobronchial amyloidosis may present with nonspecific respiratory symptoms, including cough, dyspnea, wheezing, or hemoptysis. These features can easily mimic more common conditions such as infections or malignancies, often leading to delays in diagnosis. Radiological imaging may reveal an endobronchial mass or airway irregularity, further raising suspicion for neoplastic processes. Therefore, a high degree of clinical suspicion is required, particularly in patients with known chronic inflammatory conditions like Crohn's disease.

Definitive diagnosis relies on histological confirmation. Tissue biopsy obtained through bronchoscopy typically demonstrates amorphous eosinophilic material consistent with amyloid deposition. Congo red staining is essential, revealing the characteristic apple-green birefringence under polarized light, which confirms the presence of amyloid. Identifying the subtype as AA amyloidosis is also important, as it directly influences management strategies.

Management primarily focuses on controlling the underlying inflammatory disease to reduce ongoing production of serum amyloid A protein and limit further deposition. In this case, appropriate treatment of the patient's Crohn's disease, combined with targeted management of the endobronchial lesion, resulted in significant improvement in respiratory symptoms and overall lung function. Interventional procedures may be required in some cases to relieve airway obstruction, depending on the severity and extent of involvement.

Continuous monitoring and follow-up are essential, as amyloidosis can recur or progress if the underlying inflammatory condition is not adequately controlled. Regular clinical assessment, imaging, and, when necessary, repeat bronchoscopic evaluation play a key role in long-term management.

This case highlights the importance of considering amyloidosis in the differential diagnosis of pulmonary masses, particularly in patients with chronic inflammatory diseases. Early recognition and accurate diagnosis are critical for guiding appropriate treatment and improving patient outcomes.

Conclusion

AA amyloidosis represents a rare yet significant complication of chronic inflammatory conditions such as Crohn's disease, with endobronchial involvement being an especially uncommon manifestation. Its clinical presentation can closely resemble more prevalent respiratory pathologies, including malignancy and infection, often leading to diagnostic uncertainty and delay. This case highlights the importance of maintaining a high index of suspicion in patients with longstanding inflammatory diseases who present with atypical pulmonary findings.

Histopathological confirmation remains the cornerstone of diagnosis, with Congo red staining playing a critical role in identifying amyloid deposition. Accurate subtyping of amyloidosis is essential, as it directly influences therapeutic decisions and overall management strategy. In this patient, timely diagnosis allowed for appropriate intervention, avoiding unnecessary invasive procedures aimed at presumed malignancy.

The cornerstone of treatment in AA amyloidosis is effective control of the underlying inflammatory disease to reduce serum amyloid A production and prevent further amyloid accumulation. In this case, intensified therapy for Crohn's disease, combined with supportive respiratory management, resulted in significant clinical, functional, and radiological improvement. This favorable outcome underscores the potential reversibility of symptoms when the underlying cause is adequately addressed.

Long-term follow-up is essential, as recurrence or progression of amyloidosis may occur if inflammation is not well controlled. Regular monitoring through clinical evaluation, imaging, and pulmonary function testing is crucial to ensure sustained disease control and early detection of complications.

Overall, this case emphasizes the need to consider amyloidosis in the differential diagnosis of endobronchial masses, particularly in patients with chronic inflammatory disorders. Early recognition, accurate diagnosis, and a multidisciplinary management approach are key to improving patient outcomes and minimizing morbidity associated with this rare condition.

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