

Uncommon Presentation of Undiagnosed B-Cell Lymphoproliferative Disorder as Nodular Pulmonary Amyloidosis

Harsh Patel^{a, *}, Aaiyat Sheikh^b, Gnana Deepthi Medarametla^c, Sri Abirami Selvam^d,
Syed Nazeer Mahmood^e, Gurleen Johal^f, Janani Arunachalam^g,
HariPriya Radhakrishnan^h, Viray Shahⁱ, Aditya Lal Vallath^j, Digantkumar Patel^k,
Saketh Palasamudram Shekar^l, Urvish Patel^m, Nisarg Changawalaⁿ

Abstract

B-cell lymphoproliferative disorders are characterized by the accumulation of mature B lymphocytes in the bone marrow, lymphoid tissues, and/or peripheral blood. They can cause amyloid deposits in the lungs. In rare cases, lung nodules can be the first sign of this disorder. We present the case of an 89-year-old woman with stable shortness of breath and lung nodules on imaging. A positron emission tomography-computed tomography (PET-CT) scan showed the most intense hypermetabolic nodule in the patient's lung, which was 1.5 ×

1.4 cm. A biopsy of this nodule showed amyloid material with trapped plasma cell infiltrate on microscopy. Congo red stain under polarizing microscopy showed apple-green birefringence, which is diagnostic for amyloidosis. Immunohistochemistry showed a mixture of kappa-positive and lambda-positive cells. B-cell gene rearrangement-clonal gene rearrangements were detected in the immunoglobulin heavy chain (IgH) gene and the kappa light chain (IGK). These findings suggest a B-cell lymphoproliferative disorder, such as a plasmacytoma or a marginal cell lymphoma with plasma cell differentiation. The patient was diagnosed with a B-cell lymphoproliferative disorder and pulmonary amyloidosis. Isolated amyloidosis in the lungs usually has a good prognosis, but it can be a sign of autoimmune diseases or B-cell lymphoproliferative disorders, as in this case. Early diagnosis of B-cell lymphoproliferative disorder can lead to successful treatment and prevents complications.

Manuscript submitted November 21, 2022, accepted January 11, 2023
Published online January 18, 2023

^aDepartment of Family Medicine, Central Jersey Urgent Care, Green Brook, NJ 08812, USA

^bEra's Lucknow Medical College, Lucknow, Uttar Pradesh 226003, India

^cNRI Medical College and General Hospital, Guntur, Andhra Pradesh 522503, India

^dDepartment of Internal Medicine, St Mary Medical Center, Langhorne, PA 19047, USA

^eDepartment of Medicine, Section of Pulmonary/Critical Care, MedStar Washington Hospital Center, Washington, DC 20770, USA

^fDepartment of Medicine, Hackensack Meridian Palisades Medical Center, North Bergen, NJ 07047, USA

^gDepartment of Biomedical Engineering, University of Houston, Houston, TX 77021, USA

^hShimoga Institute of Medical Sciences, Shimoga, Karnataka 577201, India

ⁱDepartment of Hospital Medicine, Medstar Good Samaritan Hospital, Baltimore, MD 21239, USA

^jDepartment of Emergency Medicine, Peerless Hospital and BK Roy Research Center, Kolkata, West Bengal 700094, India

^kSpringfield Memorial Hospital, Springfield, IL 62781, USA

^lInterventional Pulmonology, Department of Pulmonary and Critical care Medicine, Pulmonary and Sleep Associates of Huntsville, Huntsville Hospital, Huntsville, AL 35801, USA

^mDepartment of Public Health and Neurology, Icahn School of Medicine at Mount Sinai, New York, NY 10029, USA

ⁿThe Lung Center of Nevada, Las Vegas, NV 89138, USA

*Corresponding Author: Harsh Patel, Department of Family Medicine, Central Jersey Urgent Care, Green Brook, NJ 08812, USA.
Email: drharshp25@gmail.com

Keywords: Nodular pulmonary amyloidosis; Pulmonary amyloidosis; B-cell lymphoproliferative disorder; Pulmonology; Oncology; Evidence based medicine

Introduction

Amyloidosis is a rare disorder caused by amyloid material building up in organs or tissues and causing organ dysfunction or death. It can affect multiple organs (systemic) or just one organ (localized). Localized amyloidosis usually affects the skin, bladder, or lungs. There are three types of amyloidosis in the lungs: diffuse alveolo-septal, nodular, and tracheobronchial. Most cases of amyloidosis in the lungs do not cause symptoms and are found by accident. Localized amyloidosis has a good prognosis, with a 90.6% survival rate of 5 years [1]. The incidence of systemic amyloidosis is about 14 cases per 1,000,000 people per year. However, localized amyloidosis is rare, and its estimated incidence is unknown [2].

Computed tomography (CT) scans can show different types of amyloidosis in the lungs: tracheobronchial, diffuse alveolar septal, and nodular parenchymal [3]. Positron emission tomography (PET) scans usually show positive fluorode-

doi: <https://doi.org/10.14740/jmc4026>

oxyglucose (FDG) uptake in nodular pulmonary amyloidosis [4]. Compared to transbronchial needle aspiration, radiology-guided fine-needle aspiration biopsy of a peripheral pulmonary nodule has a higher yield for pathologic diagnosis [5]. Localized amyloidosis can be managed without treatment, while systemic amyloidosis can be treated with chemotherapy and anti-inflammatory drugs [6].

B-cell lymphoproliferative disorders are characterized by the accumulation of mature B lymphocytes in the bone marrow, lymphoid tissues, and/or peripheral blood. B-cell lymphoproliferative disorders are diagnosed with flow cytometric immunophenotyping that shows clonal light-chain (kappa or lambda) restricted population showing B-cell markers in the blood or bone marrow [7]. This case illustrates a B-cell lymphoproliferative disorder associated with nodular pulmonary amyloidosis, presenting as pulmonary nodules. The patient had no other symptoms or signs of this disorder. A lung nodule biopsy showed both amyloidosis and a B-cell lymphoproliferative disorder.

Case Report

Investigations

An 89-year-old woman with a past medical history of pulmonary arterial hypertension, atrial fibrillation, and ischemic stroke presented to the office to establish care for chronic stable exertional dyspnea for many years. The patient had no associated cough, wheezing, chest pain, or palpitations. The patient did not have a history of smoking cigarettes, alcohol, or illicit drugs. Her family history was significant for stroke and uterine cancer in her mother, multiple myeloma in two of her brothers, and renal cell cancer in her sister. Further investigation into history led to the discovery of a CT chest performed 4 years prior that showed multiple bilateral calcified and noncal-

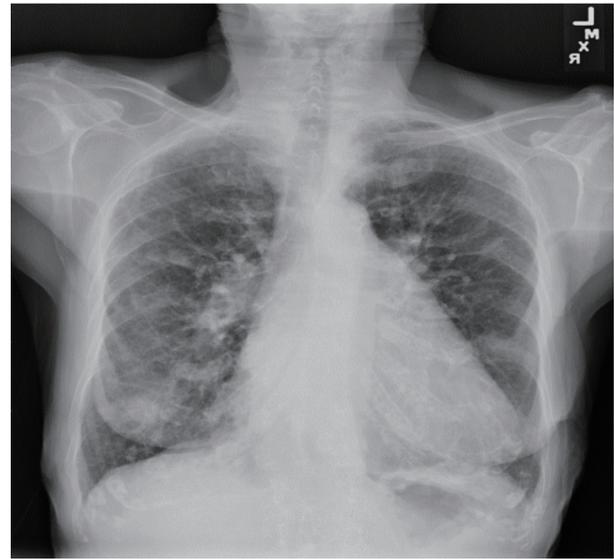


Figure 1. Chest X-ray showed extensive pulmonary nodulo-reticular shadowing at the peripheries signifying extensive fibrotic changes with scattered nodules.

cified lung nodules. The patient did not have any other further workup at that time.

Diagnosis

A plain chest X-ray performed at the presentation showed extensive pulmonary nodulo-reticular shadowing at the peripheries signifying extensive fibrotic changes with scattered nodules (Fig. 1).

Subsequently, a CT chest performed in March 2022 (Fig. 2) showed several nodules bilaterally, demonstrating an inter-



Figure 2. CT chest showed several nodules bilaterally: a calcified nodule (1.4 cm) in the apical segment of the left lower lobe and a 2-cm calcified nodule along the posterior mediastinal pleural surface of the left lung (blue arrow), above the level of the aortic knob. CT: computed tomography.

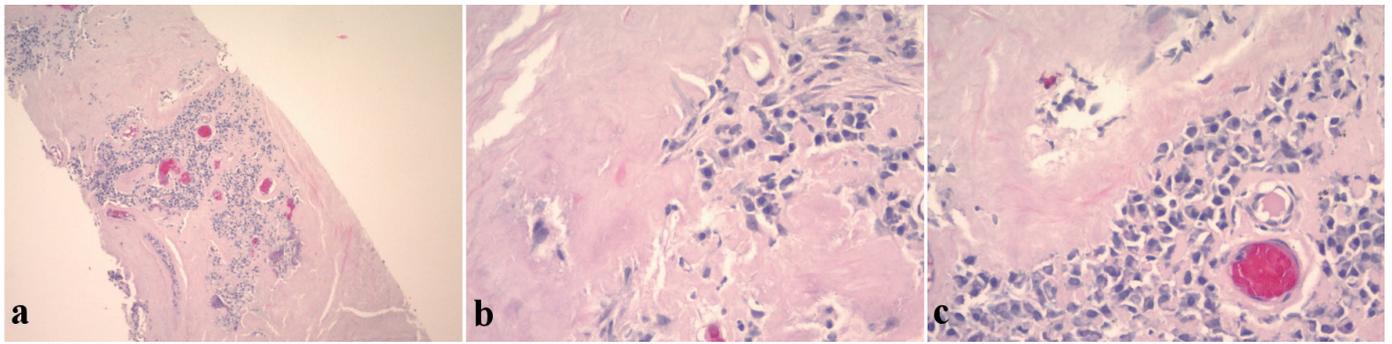


Figure 3. (a) Low magnification H&E stain. (b) High magnification H&E stain showing homogenous acellular amorphous material (amyloid) with plasma cells. (c) High magnification H&E stain showing homogenous acellular amorphous material (amyloid) with plasma cells. H&E: hematoxylin and eosin.

val increase in size compared to the prior study. A calcified nodule in the apical segment of the left lower lobe demonstrated increased soft tissue component measuring at 1.4 cm, compared to 1.2 cm in the prior study. Along the posterior mediastinal pleural surface of the left lung, above the level of the aortic knob, a 2-cm calcified nodule was spotted, unchanged since the prior study. An interval increase in the size of the nodular density in the right apex, as well as the development

of several new nodules was also noted.

Coccidioides antibody level, complement fixation (CF) was within normal limit (WNL) at < 1:2. A PET-CT scan was performed to direct appropriate lung nodule biopsy. It showed multiple bilateral mildly hypermetabolic pulmonary nodules. The most intense was in the posterior right upper lobe measuring 1.5 × 1.4 cm. Another representative area is a nodule in the lateral right middle lobe of the lungs measuring 2.4 × 2.0 cm with a max standardized uptake value (SUV) of 1.9.

A CT-guided needle biopsy of FDG avid right upper lobe (RUL) lung nodule was performed. Pathology showed homogenous acellular amorphous material with an entrapped plasma cell infiltrate on microscopy (Fig. 3a-c). Congo red stain using polarizing microscopy confirmed the presence of apple-green birefringence (Fig. 4a, b). Hematoxylin and eosin (H&E) and Congo red findings were diagnostic of amyloidosis. Immunohistochemistry showed a mixture of kappa-positive (Fig. 5) and lambda-positive (Fig. 6) cells but the predominance of kappa-positive cells. These findings suggest an atypical plasma cell population, and a tissue block was sent for B-cell rearrangement studies to diagnose plasma cell neoplasm. B-cell gene rearrangement-clonal gene rearrangements were detected in the immunoglobulin heavy chain gene (IgH) and the kappa light chain gene (IGK).

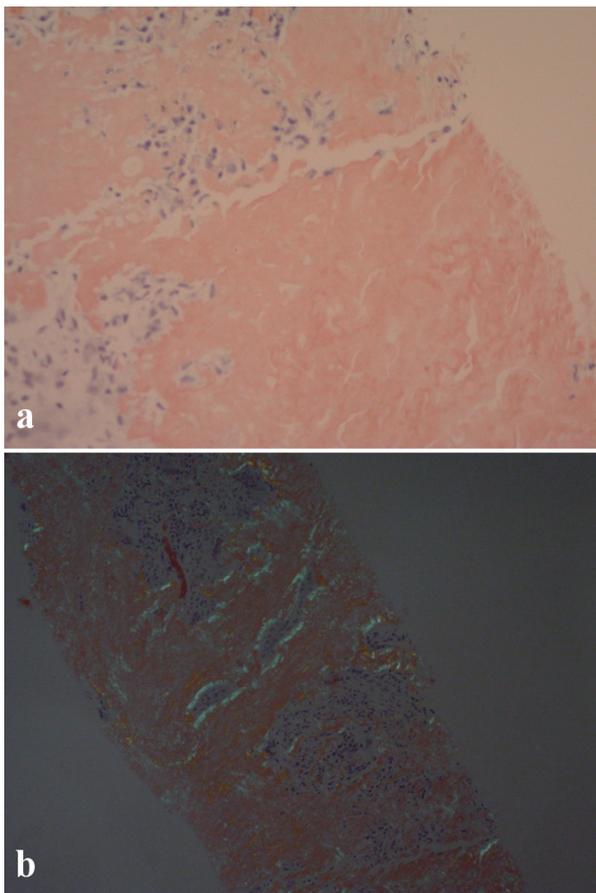


Figure 4. (a) Unpolarized Congo red stain. (b) Polarized Congo red stain showing apple-green birefringence.

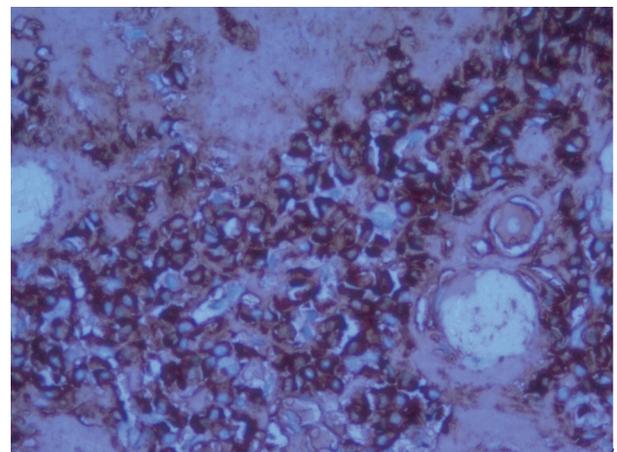


Figure 5. Immunohistochemistry showing kappa light chains.

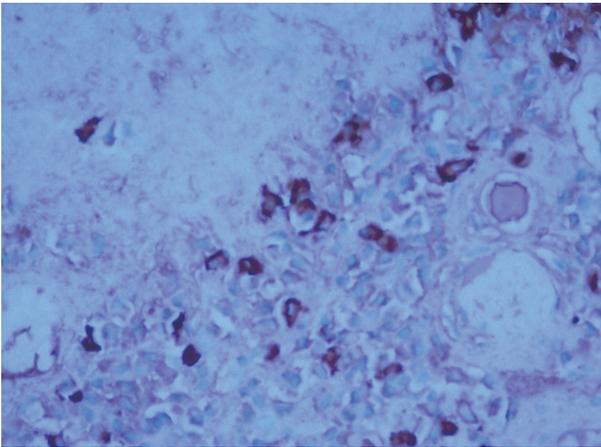


Figure 6. Immunohistochemistry showing lambda light chains.

Treatment

These results were highly suggestive of a B-cell lymphoproliferative disorder, such as a plasmacytoma or a marginal cell lymphoma with plasma cell differentiation. The patient was not prescribed any treatments.

Follow-up and outcomes

The patient was subsequently referred to oncology for further management.

Discussion

Amyloidosis is usually caused by a protein called amyloid precursor protein (APP) being produced in excess or not being broken down properly. It can be inherited or acquired. Inherited amyloidosis is rare and is caused by a gene mutation. Acquired amyloidosis is more common and can be caused by chronic inflammation, multiple myeloma, and certain other blood disorders. Amyloidosis can also be caused by other proteins, such as transthyretin, in rare cases. Transthyretin amyloidosis is usually inherited and can affect the heart. Systemic amyloidosis is caused by changes in proteins and deposits as fibrils in tissues [8, 9]. It is not common for amyloidosis to only affect one area of the body, such as the lungs. In 1877, Lesser was the first who described finding an insoluble fibrillar protein deposit in the lungs during an autopsy [10]. There are three types of amyloidosis in the lungs: tracheobronchial, alveolar septal, and nodular [11].

A study by Mayo Clinic reported a case series of 55 patients with amyloidosis in the lungs from 1980 to 1993. In a study, out of 11 patients with amyloidosis in only their lungs, seven had nodular amyloidosis, and four had tracheobronchial amyloidosis [12]. This means that nodular pulmonary amyloidosis is a slightly more common pulmonary amyloidosis. CT scans of the chest can show multiple or single nodules in the lower part of the lungs and near the surface of the lungs, i.e., a

subpleural region in nodular amyloidosis [13]. The average age of diagnosis is 67, and it is more common in men [14]. Nodular pulmonary amyloidosis is a disease that is often asymptomatic and detected incidentally; however, some people with this disease may experience symptoms such as wheezing, coughing, musical breathing, and recurrent pneumonia. It is important to consider nodular pulmonary amyloidosis in patients with multiple lung nodules [15]. Our patient, in this case, had chronic, stable shortness of breath and multiple pulmonary nodules on earlier imaging tests.

Nodular pulmonary amyloidosis is often misdiagnosed as neoplasm. Other possible causes of multiple lung nodules include infections, tumors, pneumoconiosis, sarcoidosis, rheumatoid arthritis, amyloidosis, and pulmonary alveolar microlithiasis. Tumors such as metastasis or primary lung cancer may cause multiple lung nodules. Infections that may lead to multiple pulmonary nodules include fungal infections like histoplasmosis, coccidioidomycosis, and aspergillosis [16, 17]. Other conditions like tumors and infections usually get worse quickly, but that was not the case for our patient.

A PET-CT with fluorine-18 fluorodeoxyglucose (18F-FDG) can show areas of increased cell activity. It is accurate for finding the cause of lung nodules and guiding a biopsy [18]. In our case, the scan showed the most intense hypermetabolic nodule in the patient's lung, which was 1.5×1.4 cm, guided to a CT-guided needle biopsy of this lung nodule. The gold standard test to detect amyloid material is immunohistochemistry. Amyloid material looks like a homogeneous, acellular, and eosinophilic protein under a microscope. A special Congo red stain, viewed under polarized light, shows a green color [13]. Our patient's pathology showed homogenous acellular amorphous material with an entrapped plasma cell infiltrate on microscopy. Congo red stain using polarizing microscopy confirmed the apple-green birefringence, which is diagnostic of amyloidosis.

Symptoms can guide treatment for amyloid deposits in the lungs. Patients who do not have symptoms usually do not need treatment. Various treatment options include surgical resection, resection by bronchoscopy, Nd:YAG laser therapy, and carbon dioxide laser ablation. Parenchymal amyloid nodules in the lungs usually grow slowly and do not cause symptoms, so they usually do not need treatment. Some drugs are under trial that stabilize amyloid precursor proteins and accelerate the tissue clearance of amyloid fibrils. Our patient was not given any treatment and was referred to a cancer specialist for more testing [11].

A study by Mahmood et al (2015) found that out of 606 patients with localized amyloidosis, including 35 patients with tracheobronchial amyloidosis and 47 patients with nodular pulmonary amyloidosis, only seven of them developed systemic amyloid after an average of 74 months of follow-up. The survival rate for 5 years was 90% and for 10 years was 80%. Localized amyloidosis usually has a good prognosis and does not often turn into systemic amyloidosis [19].

Lymphoproliferative disorders are conditions where certain types of blood cells (e.g., B-cells or T-cells) grow too much. Depending on the specific disorder, they can have different symptoms, pathologic findings, test results, and images. They can be either not cancerous or cancerous, includ-

ing blood cancer-like lymphomas [20]. Flow cytometry can diagnose these disorders by looking at cells from a fine needle aspiration [21].

When biopsy specimen of the lung nodule was examined under a microscope, it showed certain types of blood cells. We did more testing on the sample and found that it had a mixture of kappa-positive and lambda-positive cells, with more kappa-positive cells. This suggested that the patient had an atypical type of plasma cell. Then, a tissue block was sent for B-cell rearrangement studies to diagnose plasma cell neoplasm. Test results showed changes in specific genes in the patient’s blood cells, which suggested they had B-cell lymphoproliferative disorder. This explained the multiple lung nodules the patient had. They were diagnosed with this disorder and referred to a cancer specialist for further testing and treatment.

Here we are giving a comprehensive review of similar case reports published by other authors (Table 1) [22-30].

In conclusion, a B-cell lymphoproliferative certain kind of blood cell disorder can cause amyloid deposits in the lungs. In rare cases, it can first show as lung nodules. In our case, the patient had lung nodules and trouble breathing for a long time. She was tested following standard procedures, and it was found that her lung nodules were caused by B-cell lymphoproliferative disorder.

Learning points

B-cell lymphoproliferative disorder can present as pulmonary amyloidosis. In rare undiagnosed cases, the first manifestation can be pulmonary nodules. Isolated pulmonary amyloidosis has a good prognosis but should trigger an evaluation for auto-immune diseases and B cell lymphoproliferative disorders, as in this case, with early referral and treatment of the underlying condition. Early diagnosis of B-cell lymphoproliferative disorder can lead to successful treatment and prevents complications.

Acknowledgments

None to declare.

Financial Disclosure

The authors have no financial disclosure.

Conflict of Interest

The authors declare no conflict of interest.

Informed Consent

Informed consent was obtained by the patient for this case report.

Table 1. A Comprehensive Review of Similar Case Reports Published by Other Authors

Study name and year	Age (years)/sex (M or F)	Presenting features	Primary tumor	Immunostaining profile in addition to pathologic diagnosis of amyloidosis	Treatment	Outcome
Core et al, 2017 [22]	69/F	Progressive dyspnea, hypoxemia, dry cough, weight loss, bilateral crackles and wheezes upon auscultation, + respiratory distress	Extranodal marginal zone B cell lymphoma of MALT type with bilateral nodular pulmonary amyloidosis	Lymphocytic cells (CD20 ⁺) with scattered atypical plasma cells (kappa ⁺ , lambda ⁺ , CD138 ⁺)	RLL wedge resection by video-assisted thoracoscopic surgery, bortezomib, cyclophosphamide, dexamethasone, glucocorticoids, rituximab	Improved in the first 6 months; later worsened with acute on chronic respiratory failure
Xiang et al, 2015 [23]	59/M	Asymptomatic	Pulmonary MALT lymphoma of the left lower lobe	Lymphocytes (+ for CD79a, CD20, CD138, bcl-2, lambda light chain, CD43)	Left lower lobectomy with mediastinal lymphadenectomy	No recurrence or metastasis reported 14 months after initial diagnosis
Filippi et al, 2019 [24]	50/M	Hemoptysis	Primary pulmonary MALT lymphoma with pulmonary amyloidosis	B cells (+ for CD79a, CD20, and lambda light chain)	Left upper lobectomy	No local recurrence or metastasis in 2-year follow-up period

Table 1. A Comprehensive Review of Similar Case Reports Published by Other Authors - (continued)

Study name and year	Age (years)/sex (M or F)	Presenting features	Primary tumor	Immunostaining profile in addition to pathologic diagnosis of amyloidosis	Treatment	Outcome
Moriyama et al, 2000 [25]	63/F	Asymptomatic	MALT lymphoma of the anterior mediastinum (thymus) with nodular pulmonary amyloidosis of the lung	Lobular structures within Hassall's corpuscles (keratin ⁺). Few plasma cells (+ for IgG and kappa light chain) along with medium CCL cells (B cell phenotype + for CD20 and CD79a)	Complete thymus resection by sternotomy, resection of lung nodules by left thoracotomy	No recurrence of lymphoma in 47-month follow-up period
Lantuejoul et al, 2007 [26]	50/F	Asymptomatic with PMH of MALT-type gastric lymphoma due to <i>H. pylori</i> . CXR showed incidental multiple pulmonary nodules.	MALT-type marginal zone lymphoma involving gastrointestinal tract, bone marrow and lungs (stage IV)	Giant cells, crushed plasma cells with thioflavin T staining under UV light, small plasmacytoid lymphocytes, and large lymphocytes (CD20 ⁺)	Surgical resection of pulmonary nodules with adjuvant chemotherapy (rituximab), and bone marrow transplantation	No change in nodules and cysts on radiography during the 17-month follow-up period
Davis et al, 1991 [27]	56/M	Left upper chest pain, hemoptysis, significant smoking history, wheezes over the left upper chest upon auscultation	Small cell non-Hodgkin's left pulmonary lymphoma with bilateral lung nodules (nodular pulmonary amyloidosis)	Lymphocytic foci with plasma cells, and giant cells (+ for leukocyte common antigen). Both the lymphoma and the amyloidomas showed cells with kappa light chains.	Thoracotomy and wedge resection of two left upper lung lobe nodules, prednisone, chlorambucil	Hilar mass and amyloid pulmonary nodules decreased in size on radiography during follow-up
Wiekler et al, 2002 [28]	63/F	Significant PMH of MALT-type lymphoma of right upper eyelid	MALT-type lymphoma (low grade) of the right upper eyelid with AL amyloid deposits	Lymphocytic cell infiltrated with plasma cells (+ for Ki-B3, CD20, CD79a, IgM and h-light chain)	Cyclophosphamide, doxorubicin, hydroxydaunorubicin, oncovin and prednisone	No change in number and size of the pulmonary nodules at 6 months and 18 months follow-up
Kawashima et al, 2005 [29]	75/F	Asymptomatic	Non-Hodgkin's primary pulmonary MALT-type lymphoma of the right lung with nodular pulmonary amyloidosis	B lymphocytes (+ for L26 and CD79)	Partial resection and enucleation of right middle lobe nodules with lobectomy of the right middle and lower lobes	No recurrence of MALT lymphoma at 4-year follow-up
Ihling et al, 1996 [30]	67/M	Asymptomatic with PMH of lymphoplasmacytic immunocytomas of the lung and an abnormal lymphoid infiltrate in the bone marrow	Primary extranodal lymphoplasmacytic immunocytomas of the lung or the bronchus-associated lymphatic tissue with intrafocal tumor-like amyloidosis	Plasma cells between amyloid deposits (+ for lambda light chains), lymphocytes and plasma cells surrounding amyloid tumor (+ for kappa and lambda light chains), amplification of the IgH gene with sharp bands	No treatment as hematopoiesis was not affected	-
Ihling et al, 1996 [30]	66/M	Asymptomatic with tumorous growths in the right lower and left lower lobes on chest X-ray	Primary extranodal lymphoplasmacytic immunocytomas of the lung or the bronchus-associated lymphatic tissue with intrafocal tumor-like amyloidosis	Plasma cells (+ for lambda and kappa light chains in equal frequency), lymphocytes from lung biopsy (CD20-reactive B-lymphocytes and CD3-reactive T-lymphocytes), amplification of the IgH with sharp bands	Removal of right lower lobe and mediastinal lymphadenectomy	-

F: female; M: male; *H. pylori*: *Helicobacter pylori*; +: positive; MALT: mucosa-associated lymphoid tissue; RLL: right lower lobe; PMH: past medical history; CXR: chest X-ray; AL: immunoglobulin light chain amyloidosis; IgH: immunoglobulin heavy chain; UV: ultraviolet; CCL: centrocyte-like.

Author Contributions

Acquisition of data: Nisarg Changawala. Conception or design of the work: Harsh Patel, Janani Arunachalam, Haripriya Radhakrishnan. Drafting of the manuscript: Harsh Patel, Aaiyat Sheikh, Gnana Deepthi Medarametla, Sri Abirami Selvam, Gurleen Johal, Aditya Lal Vallath. Revisions: Harsh Patel, Nisarg Changawala. Supervision: Syed Nazeer Mahmood, Viray Shah, Digantkumar Patel, Saketh Palasamudram Shekar, Urvi Patel, Nisarg Changawala.

Data Availability

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

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