CASE REPORT

Nodular pulmonary amyloidosis: a complex disease with malignancy association

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SUMMARY

Pulmonary amyloidosis is a rare disease that incorporates deposition of amyloid microfibril material in the lung parenchyma. The condition generally presents as an indolent subacute-to-chronic pulmonary disease and requires tissue biopsy to establish the diagnosis. Nodular pulmonary amyloidosis, a subtype of pulmonary amyloidosis, is characterised by special radiographic and pathological features. While the disease can be associated with inflammatory conditions; its association with mucosal-associated lymphoid tissue (MALT lymphoma) is unusual and carries management challenges. Herein, we illustrate a case study of nodular pulmonary amyloidosis associated with underlying MALT lymphoma in a patient with known systemic lupus erythematosus. The aim of this article is to share the management experience of this complex condition with the medical community and to conduct an up-to-date literature review on nodular pulmonary amyloidosis.

BACKGROUND

Amyloidosis is a heterogeneous disease that is characterised by misfolded protein of amyloid microfibril deposition in various tissue and organs in the human body.¹ Subsequently, the disease can result in different clinical presentations depending mainly on protein subtype, degree of deposition and organ type involvement.^{1,2} The disease is either localised or systemic and can involve one or more organs simultaneously. Epidemiologically, the disease is classified as 'rare' with estimated incidence of 10 cases per 1 million persons-years.^{3,4} Pulmonary amyloidosis can be a product of chronic inflammatory process such as systemic lupus erythematosus (SLE) or rheumatoid arthritis.^{4,5} Pulmonary manifestations of the disease include interstitial lung infiltrates, tracheobronchial infiltration, parenchymal nodules and cystic lung disease.⁶ Pleural effusion, although common in systemic and cardiac amyloidosis, has unknown incidence in pulmonary amyloidosis and can be seen in some cases (like our case).^{4,7}

Nodular pulmonary amyloidosis (NPA), a subtype of pulmonary amyloidosis, is thought to be associated with inflammatory and malignant conditions.^{3,4,8} NPA has been reported to mask underlying malignancies in a few case reports including haematological malignancies and, rarely, mucosal-associated lymphoid tissue (MALT) lymphoma.⁹ Often, NPA can be completely asymptomatic and may be diagnosed as an incidentaloma on chest imaging.

In other instances, the disease can be manifested as a significant pulmonary syndrome with obstructive and/or restrictive pulmonary burden.⁶ In this article, we discuss a case of NPA complicated by MALT lymphoma in a patient with history of SLE and recently treated Hodgkin's lymphoma.

CASE PRESENTATION

A 69-year-old female with a complex medical history including SLE, an unspecified, interstitial lung disease and Hodgkin's lymphoma presented to the emergency room with progressive dyspnoea and hypoxaemia. At baseline, she used 2 L of oxygen via nasal cannula, which had increased to 5 L in the past few days. She complained about dry cough and significant weight loss (12 kg) over the past 3 months prior to the presentation. She denied any other significant symptoms including orthopnoea, paroxysmal nocturnal dyspnoea, fever, night sweat, haemoptysis or any gastrointestinal symptoms.

The patient had a long-standing remitting SLE, previously treated with hydroxychloroquine. She had a radiographic evidence of fibrotic interstitial lung disease, which was managed with home oxygen therapy. Two years prior to this visit, the patient was diagnosed with Hodgkin's lymphoma. She underwent six cycles of rituximab, doxorubicin, vincristine and prednisone with complete remission. The patient was a lifetime non-smoker and denied alcohol use. She had no significant bird or hot-tub exposure and denied recent travels outside her home town in southeast USA. She had no family history of autoimmune disease, lung disease or malignancy.

Physical examination at presentation showed blood pressure of 154/88 mm Hg, heart rate of 105 beats-per-minute and respiratory rate of 32 breaths-per-minute with oxygen saturation of 91% on 5 L nasal cannula. There was audible wheezing during speech, with diffuse bilateral crackles and mixed inspiratory–expiratory wheezes auscultated diffusely but most pronounced in the midlower posterior lung fields. She demonstrated respiratory distress with nasal flaring and accessory muscle use. Cardiovascular examination was non-remarkable, and no palpable lymphadenopathy was present.

INVESTIGATIONS

Laboratory tests revealed haemoglobin of 10.1 mg/dL (reference value in female 12–15.5 mg/dL) and creatinine level of 0.9 mg/dL (79.56 µmol/L) (reference value 0.8–1.2 mg/dL). Other laboratory tests

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Figure 1 (A) Chest X-ray showing nodular opacities and infiltrates in both middle and lower lung fields. (B) Chest CT showing bilateral, prominent, ill-defined nodular opacities occupying both lungs in the middle-to-lower fields causing narrowing of the pulmonary arteries (arrows). Although those findings are non-specific, they may suggest inflammatory or neoplastic pulmonary process.

including procalcitonin, antinuclear antibody, anti-Ro (SSA) or anti-La (SSB) antibodies, complement levels, C reactive protein and sedimentation rate returned within normal limits. Kappa free light chain, lambda free-light chain and kappa/lambda ratio were 2.04 mg/dL (reference range 0.33–1.94 mg/dL), 1.28 mg/dL (reference range 0.57–2.63 mg/dL) and 1.59 (reference range 0.26–1.65), respectively. IgA, IgG and IgM were within normal limits. Respiratory and blood cultures were negative. Chest X-ray showed an interval increase in nodular opacities since the previous study, (figure 1). Chest CT revealed bilateral, prominent, ill-defined, perihilar nodular opacities with bilateral consolidations, more noticeable in the left lung with large (~5 cm) hilar lymphadenopathy causing narrowing of both pulmonary arteries. As laboratory data did not suggest any abnormal autoimmune activity and clinical presentation did not point towards an infectious aetiology, thus findings were most concerning for an inflammatory or neoplastic process (figure 1). Fibreoptic bronchoscopy was performed and showed minimal focal narrowing of the left main and subsegmental bronchi.

Figure 2 (A) Pathology image of the right lower lobe wedge resection revealed multiple foci of nodular amyloidosis within the alveolar space (arrows), associated with stromal fibrosis, patchy perivascular and septal amyloid deposition, H&E×4. Amyloid phenotyping by mass spectrometry analysis confirmed the diagnosis of amyloidosis. (B) Congo-red stain showing apple-green birefringence confirming the diagnosis of pulmonary amyloidosis. (C) Negative CD20 staining of the sample, indicating amyloidosis with no evidence of lymphoma or lymphoproliferative disease.

Endobronchial ultrasound guided fine-needle aspiration of the mediastinal lymph nodes was performed; however, pathology analysis was inconclusive.

A wedge resection of the right lower lobe was performed using video-assisted thoracoscopic surgery. Pathology of the wedge resection revealed foci of nodular amyloidosis within the alveolar spaces, associated with stromal fibrosis, patchy perivascular and septal amyloid deposition. Amyloid phenotyping by mass spectrometry analysis confirmed the diagnosis of amyloidosis, Amyloid Light chain2 (AL) (kappa)-type, (figure 2).

ECG revealed sinus tachycardia with normal QTc-interval and voltage activity. A transthoracic echocardiogram showed normal left ventricular ejection fraction, wall thickness and no signs of pericardial effusion or restrictive pattern to suggest infiltrative cardiac disease. Serum amyloid P component scintigraphy was not performed, as there was neither suspicion for systemic multi-organ involvement nor is the necessary testing equipment available at our centre.

DIFFERENTIAL DIAGNOSIS

Due to the patient's history of SLE and Hodgkin's lymphoma, the differential diagnosis for the new pulmonary nodular infiltrates should include malignant, inflammatory or infectious processes. Lymphoma relapse and other solid lung malignancy (both primary and metastatic) should be considered in such presentation. Inflammatory conditions like SLE relapse and rheumatoid arthritis are possibilities. Infections must be excluded in those circumstances. The pathology findings revealed the final diagnosis of AL (kappa)-type pulmonary amyloidosis which, in the setting of pulmonary nodules on imaging, promotes the diagnosis of NPA.

TREATMENT

A multidisciplinary management approach was adopted including internal medicine, pulmonology and haematology/oncology teams. After shared decision making with the patient and her family, the treatment plan was tailored to target the NPA with cyclophosphamide, bortezomib and dexamethasone (CyBORd) for seven cycles.¹⁰ Patient responded to supportive treatment and was discharged to rehabilitation facility after 10 days of hospitalisation.

Nine months later, the patient's hypoxaemic respiratory failure had deteriorated again, mandating hospitalisation and subsequently intubation and mechanical ventilation. Infectious processes were unlikely as blood and bronchial alveolar lavage cultures were negative. Chest X-ray showed worsening atelectasis and consolidations bilaterally, with more prominence on the left side, and chest CT scan showed worsening infiltrates in both lung fields (figure 3). Fibreoptic bronchoscopy showed focal narrowing of the left main and ostium of subsegmental bronchi concerning for extramural compression. Endobronchial mucosa was also friable and oedematous with difficulty to further pass the bronchoscope through the distal segments (figure 4).

Forceps biopsies of endobronchial mucosa were obtained with pathology analysis showed findings consistent with MALT lymphoma. The H&E stain of the bronchial biopsy obtained from main stem of left lung revealed dense lymphocytic proliferation underneath the benign bronchial mucosa. Immunostaining of CD20 showed predominant small to medium B lymphocytes. Additional immunostaining of CD138, kappa and lambda revealed scattered intermixed large atypical plasma cells with kappa restriction. The overall findings were compatible with extranodal marginal zone

Figure 3 (A) Chest X-ray showing worsening opacities of both lung fields, which is more prominent on the left side with almost complete lung involvement as compared with chest X-ray after treatment with rituximab and high dose glucocorticoids as shown in panel B. (C) CT showing worsening interstitial opacities in both lung fields with nodular infiltrates with bilateral moderate pleural effusions (arrows). Image is compared with CT after treatment as shown in panel D.

B cell lymphoma of MALT type. Special stain of Congo red was negative for amyloid. No evidence of classic Hodgkin's lymphoma was present (figure 5).

Treatment for MALT lymphoma was initiated with high-dose glucocorticoids and rituximab.^{11–14} A pulse-dose of 1000 mg of methylprednisolone was administered for three consecutive days, with a second round started 7 days later. Rituximab was administered every week for 4 weeks with a dose of 375 mg/m². Strict supportive management including a low tidal volume mechanical ventilation, and critical care support was continued.

Figure 4 Fiberoptic bronchoscopy image of the left main bronchus showing the bifurcation of the left upper and middle segmental bronchi. Mucosa appeared to be friable and oedematous (arrows).

Figure 5 (A) Pathology image of the bronchial biopsy obtained from main stem of left lung revealed dense lymphocytic proliferation underneath the benign bronchial mucosa, H&E $\times 10$ original magnification. (B) Immunostaining of CD20 showed predominant small to medium B lymphocytes that confirms the diagnosis of extranodal marginal zone B cell lymphoma, MALT type, $\times 10$ original magnification.

OUTCOME AND FOLLOW-UP

Nine days after the initiation of high-dose glucocorticoids and rituximab, the patient condition improved gradually. A follow-up chest X-ray and chest CT scan showed dramatic decrease in size of infiltrates/consolidations with re-expansion of the left upper lobe (figure 3). Bilateral pleural effusions were resolved after sequential drainage by thoracentesis. Pleural fluid analysis showed transudate with no infectious or malignant findings. She was subsequently liberated from mechanical ventilation and transferred to a regular medical floor with persistent clinical improvement. Follow-up after discharge from the hospital was arranged with pulmonary medicine and haematology/oncology where she was found to be in stable clinical condition using baseline oxygen requirement. Patient was to receive maintenance rituximab every 2 months at 375 mg/m² with monitoring of disease resolution on CT scan. In total, she received five doses of rituximab and was found to have hypogammaglobulinaemia on the sixth cycle, mandating treatment with intravenous immunoglobulin.

Over the course of the next year, the patient condition improved in first 6 months; however, she deteriorated again in the later course with another episode of acute on chronic respiratory failure. Ibrutinib was offered as a single agent to treat MALT lymphoma based on a recent multicentre, open label study.¹⁵ Also, rituximab and lenalidomide were considered as a treatment for MALT lymphoma based on a phase 2 multicentre study, however, the patient and her family declined any further treatment after a thorough multidisciplinary discussion on prognosis.¹⁶ The patient received palliative measures after withdrawal of all invasive interventions. She subsequently died soon after under hospice care.

DISCUSSION

The term 'amyloid' refers to an abnormal, misfolded, fibrillar protein deposition that can involve various organs in the human body. Classically, the disease can be diagnosed by Congo red stain with apple-green birefringence under polarised light microscope. Amyloidosis can be classified to 15 known subtypes according to its precursor protein derivative. Four subtypes have been recognised to represent the majority of the diagnosed cases worldwide (table 1).⁴

Pulmonary amyloidosis is a rare disease that involves different pathological patterns:⁴

- Diffuse alveolar septal amyloidosis, which involves alveolar deposition of amyloid material leading to defected gas exchange process. This type is usually associated with poor prognosis.

Table 1 Different types of amyloid protein precursors⁴

Type of amyloidosis	Amyloid composition	Organs involved	Comment
AL*	Immunoglobulin-derived light chain	Heart, kidneys, gastrointestinal tract, nervous system and lungs	Associated with plasma cell dyscrasias
ATTRm	Mutant transthyretin	Heart and nervous system	Autosomal dominant
ATTRwt	Wild-type transthyretin	Heart and lungs	Age related, acquired†
AA apolipoprotein	Serum amyloid A	Heart, lungs, liver and kidneys	Secondary to inflammatory conditions

*Can be systemic or localised.

†90% are men >60 years old.

AA, amyloid; ATTR, amyloid transthyretin; AL, amyloid light chain; NPA, nodular pulmonary amyloidosis.

- ▶ Tracheobronchial amyloidosis, which does not usually involve monoclonal proliferation and presents usually with significant pulmonary symptoms secondary to airway stenosis.
- ▶ NPA: this is associated with lymphomas (like in our case) and inflammatory conditions.^{3 4 9} NPA is commonly asymptomatic at the time of diagnosed; however, if presents with symptoms, it carries significant diagnostic and management challenges.⁴

Nodular appearance on lung imaging is usually the initial trigger to suspect NPA. When nodular appearance is localised on chest CT scan, it usually raises the suspicion for primary or metastatic lung malignancy.¹⁷ Although our patient had SLE, it is very rare to see this chronic, inflammatory, autoimmune condition associated with pulmonary amyloidosis. The exact mechanism of this disease and the association has not yet established.^{18 19} The nodular appearance of NPA on imaging has been postulated to represent a pre-existing chronic inflammatory condition.²⁰ Sjögren's disease and lymphoproliferative disorders have been reported to have nodular appearance on chest CT scan mimicking NPA, and the association of these conditions with NPA has been described in only few case reports. Our patient did not have clinical manifestations of Sjögren's disease nor positive anti-Ro (SSA) or anti-La (SSB) antibodies which makes the diagnosis of this disease in this case unlikely.

MALT lymphoma has been reported in the literature to be associated with NPA in rare case reports.^{20 21} Therefore, it is important to highlight that when both conditions (NPA and MALT lymphoma) present simultaneously, the diagnosis can be challenging and more difficult if the conditions present individually. As NPA presentation is usually asymptomatic and the disease natural course is not known, MALT lymphoma usually carries significant morbidity and mortality.^{11 22} It should be noted that our patient received cyclophosphamide, bortezomib and dexamethasone for the NPA months before the diagnosis of MALT-lymphoma. It is unknown what impact the therapy had on the progression and/or occurrence of the lymphoma, and to encounter both conditions simultaneously, the choice to treating them individually or simultaneously can be individually tailored on case-to-case basis as it has not been established or delineated clearly in the literature.^{21 22}

Bortezomib targets the NF-κB-dependent activated B cell subtype of diffuse large B cell lymphoma and has been shown to be partially effective (in about 50% of cases) of

MALT lymphoma.²³ Therefore, if MALT lymphoma was present at time of NPA diagnosis, bortezomib was not effective against the possible underlying malignancy.

The differential diagnosis for nodular appearance on lung imaging is broad, and the exact diagnosis cannot be established by chest imaging alone. Therefore, obtaining a tissue biopsy is crucial to establish the diagnosis of those two, rarely associated diseases. Despite the fundamental importance of obtaining a tissue diagnosis, bronchoscope and endobronchial ultrasound guided biopsies may have limited yield, particularly subcentral lesions, mostly due to technical challenges that are a known limitation of bronchoscopic biopsy.^{24–26} Surgical tissue biopsy may be the ultimate method to achieve the diagnosis in these cases due to its higher yield and sufficient sample size to allow special staining for MALT lymphoma.^{9 11}

It is unknown whether NPA caused the MALT-type lymphoma or vice versa, and whether their coexistence is a correlation or merely a coincidence. While intubated and mechanically ventilated in the critical care unit, our patient underwent a surgical open lung biopsy procedure to yield an adequate sample and firmly establish the diagnosis. The mass spectroscopy, which takes weeks to obtain its results, yielded kappa light chains in the amyloidosis biopsy, and the stain yielded a kappa restriction within the plasma cells of the MALT lymphoma biopsy. Also, the characteristics of light chain deposition in NPA can be observed in MALT-type lymphomas, as they both are known to have immunosecretory traits. Therefore, the conclusion can be drawn that the two conditions are correlated with each other. Thus, the assumption can be made that amyloidosis may arise from the lymphoma itself.⁸ However, because both NPA and MALT lymphoma can arise from chronic inflammatory state, the exact sequence of aetiology may be hard to ascertain.^{6 13} In our belief, NPA may have masked the MALT lymphoma for an extended period of time. Especially in the circumstance that the patient did not improve on CyBorD therapy for few months and the improvement warranted when, although delayed, diagnosis of MALT lymphoma was established and treated.

In the last decade, there has been a change in the paradigm of treating amyloidosis with the introduction of new regimens like CyBorD.²⁷ In a European collaborative study, this regimen has been shown to be associated with an excellent partial response in a significant number of patients. Therefore, currently, this regimen is preferred over other regimens in treating AL-type amyloidosis.¹⁰ Larger studies are still needed to better characterise the outcome of patients treated with this regimen.

Learning points

- ▶ Pulmonary amyloidosis is a rare disease.
- ▶ Nodular pulmonary amyloidosis can be associated with, or even mask, B cell lymphoma, particularly mucosal-associated lymphoid tissue lymphoma.
- ▶ Different management modalities can be deployed to diagnose and treat those challenging yet potentially treatable conditions.

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