

Clinical characteristics of amyloidosis with isolated respiratory system involvement: A review of 13 cases

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Abstract:

BACKGROUND: Isolated pulmonary amyloidosis is a very rare disease.

METHODS: We retrospectively reviewed the records of patients with pathologically proven isolated pulmonary amyloidosis treated at our hospital from 1990 to 2011.

RESULTS: There were 9 males and 4 females with a mean age of 54.7 years (range, 45–72 years) and the mean course of disease was 46.5 months (range, 5 months–15 years). The most common symptoms were cough (10/13), expectoration (8/13), hemoptysis (4/13), chest tightness (12/13), dyspnea (10/13), chest pain (3/13), fever (5/13), and body weight loss (2/13). Radiological findings included tracheal stenosis (2/13), bronchial stenosis with atelectasis (5/13), pulmonary nodules (3/13), lung consolidation (1/13), and lymph node enlargement with pleural effusion (2/13). Treatments included endotracheal stenting, endoscopic resection of tracheal and bronchial lesions, lung resection, and drug therapy with glucocorticoids, antineoplastic agents, or antibiotics. Four patients died of the disease within 1 year of diagnosis, 2 died of pneumonia at 3–4 years after original treatment, and the remaining patients are alive with follow-up ranging from 3 to 15 years.

CONCLUSIONS: Isolated pulmonary amyloidosis is a rare disease with a relatively high mortality and its various manifestations make diagnosis challenging. Surgical resection of lesions and chemotherapy tend to be effective treatments.

Key words: Amyloidosis, colchicines, mediastinum, melphalan, isolated pulmonary amyloidosis, respiratory system

Amyloidosis is the name given to a group of conditions in which abnormal protein material (amyloid) is deposited in the extracellular matrix resulting in disordered structure and dysfunction of tissues and organs. Amyloidosis is caused by the abnormal folding of proteins, and the amyloid is made up primarily of protein fibrils.^[1,2] Primary systemic amyloidosis is a heterogeneous disease with varied manifestations, and can involve the kidneys, gastrointestinal tract, skin, respiratory system, heart, and other organs; however, forms of localized amyloidosis affecting only one organ system are known.^[1] The incidence of amyloidosis is low,^[2] and that of amyloidosis with involvement of only the respiratory system is extremely low.^[3]

There is no gender difference in the incidence of primary amyloidosis, and the disease typically occurs in individuals >50 years of age. The clinical presentations of amyloidosis are protean, and symptoms are non-specific, which can make arriving at a diagnosis difficult.^[1,2] The condition can be an incidental finding, chronic or progressive, with death occurring within months of diagnosis in severe cases.^[1,2] No definitive treatment is available, and common treatments included glucocorticoid, melphalan, and colchicines.^[1,2]

Manifestations of isolated pulmonary amyloidosis include dyspnea, cough, and hemoptysis and clinical findings are as varied as isolated pulmonary nodules, mediastinal lymphadenopathy, and non-specific radiographic findings.^[4-6] Diseases such as pulmonary tuberculosis, connective tissue disease, malignancy, and multiple myeloma need to be ruled-out in order to avoid delaying treatment.

Because of the rarity of amyloidosis involving only the respiratory system, we searched the records of our hospital over a 21-year period and identified 13 patients with pathologically proven amyloidosis involving only the respiratory system. The purpose of this study is to present the clinical characteristics, pathological findings, treatments, and outcomes of these patients.

Methods

The medical records of 13 patients with pathologically proven amyloidosis and respiratory system involvement treated at our hospital from January 1990 to May 2011 were retrospectively reviewed. Patient demographic and clinical characteristics including gender, age, disease course, age of onset, clinical manifestations, laboratory findings, imaging

findings, and pathological findings were recorded for analysis. This study was approved by the Institutional Review Board of our hospital, and because of its retrospective nature the requirement of informed consent was waived.

Results

Patients

There were 9 males and 4 females with a mean age of 54.7 years (range, 45–72 years) included in the analysis. The mean duration from symptoms onset to confirmed pathological diagnosis was 11.2 months (range, 1–51 months). The mean course of disease was 46.5 months (range, 5 months–15 years). Patient demographic and clinical characteristics are summarized in Table 1.

The clinical manifestations at presentation included cough ($n = 10$), expectoration ($n = 8$), hemoptysis ($n = 4$), chest tightness ($n = 12$), dyspnea ($n = 10$), chest pain ($n = 3$), intermittent fever ($n = 5$), and body weight loss ($n = 2$). Superficial lymph node enlargement was not found in any patient. The respiratory sounds were reduced and dullness was noted on percussion in 2 patients, wheezing on auscultation was found on inhalation in 6 patients, and moist rales were found in 4 patients.

Enlargement of the liver and spleen was absent in all patients, and work-up for tuberculosis, syphilis, chronic renal disease, hypergammaglobulinemia, rheumatoid arthritis, malignancies, and multiple myeloma was negative in all patients.

Laboratory examination

All patients were negative for HIV. The mean peripheral white blood cell (WBC) count was $6.2 \pm 1.9 \times 10^9/L$ (neutrophils, $0.61 \pm 0.05\%$); platelet count, $245 \pm 39 \times 10^9/L$; hemoglobin (Hb), $12.1 \pm 1.6 \text{ g/L}$; and erythrocyte sedimentation rate (ESR) was $22 \pm 8 \text{ mm/h}$. All patients had normal kidney and liver function testing, and normal serum electrolyte levels. Bone marrow examination was done in 2 patients, and was normal in both.

Imaging examinations

Abdominal ultrasonography did not reveal enlargement of the liver, spleen, or retroperitoneal lymph nodes in any patients. Chest radiograph findings were non-specific in all patients. Computed tomography (CT) or magnetic resonance imaging (MRI) revealed rugged mucosa in the trachea and adjacent major main bronchus resulting in tracheal stenosis in 2 patients and bronchial stenosis with atelectasis in 5 patients [Figure 1a and b]. Lung consolidation was found in 1 patient [Figure 2]. Nodular shadows in the lung were

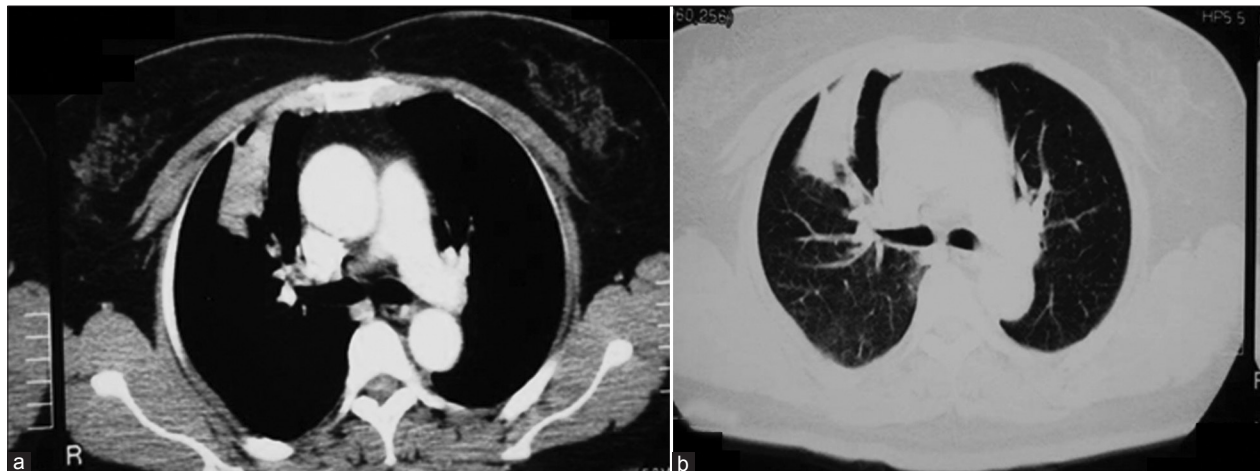


Figure 1: Computed tomography from a 49-year-old female (case no. 5) showing narrowed bronchi and atelectasis

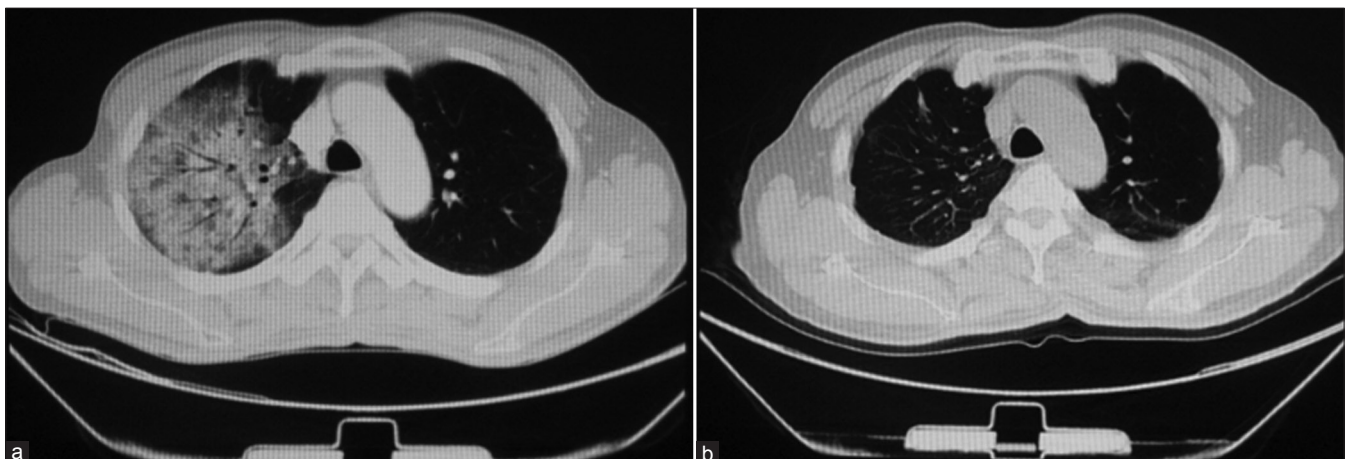


Figure 2: (a) Computed tomography from a 63-year-old male (case no. 13) revealing lung consolidation. (b) Consolidation was resolved after 2 months of treatment

Table 1: Patient demographic and clinical characteristics

No.	Age	Sex	Pulmonary diseases; Smoking	Duration between symptoms onset and pathological diagnosis (months)	Clinical manifestations	Computed tomography or magnetic resonance imaging findings	Lung function	Bronchoscopy	Diagnostic procedure	Treatment	Out-come
1	48	M	No history of lung disease; 20 cigarettes/day x 30 years	15	Cough, expectoration, chest tightness, dyspnea	Tracheostenosis	Moderate mixed ventilatory dysfunction, moderately compromised lung diffusion capacity	Tracheal stenosis, rugged mucosa with nodular deposits	Bronchoscopy with biopsy	Glucocorticoid	Died of respiratory failure and pneumonia after 4 years
2	47	F	No history of lung disease or smoking	9	Cough, expectoration, chest tightness, dyspnea	Tracheostenosis	Mild mixed ventilatory dysfunction, mildly compromised lung diffusion capacity	Tracheal stenosis, rugged mucosa with nodular deposits	Bronchoscopy with biopsy	Endotracheal stenting	Stable disease after 3 years
3	59	M	No history of lung disease; 30 cigarettes/day x 25 years	6	Cough, expectoration, hemoptysis, chest tightness, dyspnea, chest pain, intermittent fever	Stenosis of right upper bronchus and atelectasis	Moderate mixed ventilatory dysfunction, moderately compromised lung diffusion capacity	Stenosis of right upper bronchus, rugged mucosa with nodular deposits	Bronchoscopy with biopsy	Glucocorticoid and antibiotics	Died of pneumonia and hemoptysis after 3 years
4	45	F	No history of lung disease or smoking	3	Cough, expectoration, hemoptysis, chest tightness, intermittent fever	Stenosis of left upper bronchus and atelectasis	Mild mixed ventilatory dysfunction, mildly compromised lung diffusion capacity	Stenosis of left upper bronchus, rugged mucosa with nodular deposits	Bronchoscopy with biopsy	Thoracotomy with lobectomy of left upper lobe	Stable disease after 15 years
5	49	F	No history of lung disease or smoking	18	Cough, expectoration, chest tightness, dyspnea, intermittent fever, hemoptysis	Stenosis of right upper bronchus and atelectasis	Moderate mixed ventilatory dysfunction, mildly compromised lung diffusion capacity	Stenosis of right upper bronchus, rugged mucosa with nodular deposits	Bronchoscopy with biopsy	Thoracotomy with lobectomy of right upper lobe	Stable disease after 5 years
6	52	M	No history of lung disease or smoking	6	Cough, expectoration, chest tightness, dyspnea, intermittent fever, hemoptysis	Stenosis of left lower bronchus and atelectasis	Mild mixed ventilatory dysfunction, moderately compromised lung diffusion capacity	Stenosis of left lower bronchus, rugged mucosa with nodular deposits	Bronchoscopy with biopsy	Glucocorticoid, intermittent antibiotics	Stable disease after 3 years
7	61	M	No history of lung disease; 20 cigarettes/day x 40 years	13	Cough, expectoration, chest tightness, dyspnea	Stenosis of left upper bronchus and atelectasis and inflammation	Mild mixed ventilatory dysfunction, moderately compromised lung diffusion capacity	Stenosis of left upper bronchus, rugged mucosa with nodular deposits	Bronchoscopy with biopsy	Thoracotomy with lobectomy of left lower lobe	Recurrence 9 months after original treatment; died of pneumonia 18 months after further resection
8	55	M	No history of lung disease or smoking	51	Cough, expectoration, chest tightness, dyspnea, chest pain, intermittent fever	Diffuse shadows and nodules	Mild mixed ventilatory dysfunction, moderately compromised lung diffusion capacity	No abnormalities	Thoracoscopic biopsy	Glucocorticoid, intermittent antibiotics	Died of respiratory failure 1 year after diagnosis

contd....

Table 1: Contd...

No.	Age	Sex	Pulmonary diseases; Smoking	Duration between symptoms onset and pathological diagnosis (months)	Clinical manifestations	Computed tomography or magnetic resonance imaging findings	Lung function	Bronchoscopy	Diagnostic procedure	Treatment	Out-come
9	48	F	No history of lung disease or smoking	11	Chest tightness	Nodular shadow in left lower lobe (23 x 25 mm)	Normal pulmonary ventilation, normal lung diffusion capacity	No abnormalities	Thoracotomy with Wedge resection of the nodule	Thoracotomy with wedge resection of the nodule	No recurrence after 8 years
10	63	M	No history of lung disease; 20 cigarettes/day x 30 years	5	Chest pain	Nodular shadow in right lower lobe (15 x 18 mm)	Normal pulmonary ventilation, normal lung diffusion capacity	No abnormalities	Thoracoscopy with wedge resection of the nodule	Thoracoscopy with wedge resection of the nodule	No recurrence after 3.5 years
11	49	M	No history of lung disease; 30 cigarettes/day x 25 years	5	Chest tightness, dyspnea, body weight loss	Enlarged mediastinal lymph nodes and bilateral pleural effusion	Mild mixed ventilatory dysfunction, moderately compromised lung diffusion capacity	No abnormalities	Mediastinoscopic biopsy of lymph nodes and thoracoscopy biopsy of pleura simultaneously	Glucocorticoid, and methotrexate	Died of respiratory failure and pneumonia 9 months after diagnosis
12	72	M	No history of lung disease; 20 cigarette/day x 50 years	3	Cough, chest tightness, dyspnea, body weight loss	Enlarged mediastinal lymph nodes and bilateral pleural effusion	Mild mixed ventilatory dysfunction, moderately compromised lung diffusion capacity	No abnormalities	Mediastinoscopic biopsy of lymph nodes and thoracoscopy biopsy of pleura simultaneously	Traditional Chinese medicine	Died of respiratory failure and pneumonia 5 months after diagnosis
13	63	M	No history of lung disease; 20 cigarette/day x 40 years	1	Cough, chest tightness, dyspnea	Massive consolidation in right lung	Normal pulmonary ventilation, mildly compromised lung diffusion capacity	No abnormalities	Percutaneous lung biopsy	Glucocorticoid, melphalan, and antibiotics for 2 months; lesions resolved and therapy was discontinued	Stable disease at 1.5 years after diagnosis

noted in 3 patients; solitary nodule in 2 and multiple nodules in 1. Enlargement of mediastinal lymph nodes accompanied by bilateral pleural effusion was found in 2 patient [Figure 3].

Bronchoscopy

Bronchoscopy was performed in all patients, and in 7 the mucosa of trachea and bronchi was rugged and nodular deposits were noted. Congestion and edema were also found in the mucosa, which was susceptible to bleeding on touching [Figure 4].

Pathological examination

Bronchoscopic biopsy was performed in 7 patients, thoracoscopic biopsy in 1, percutaneous lung biopsy in 1, and wedge resection in 2. Two patients underwent mediastinoscopic biopsy of lymph nodes and thoracoscopic biopsy of pleura simultaneously. Pathological examination under a light microscopy and hematoxylin and eosin (H&E) staining revealed homogeneous, pink eosinophilic amyloid without cellular structure, and Congo red-stained tissue exhibited saffron yellow substances under light microscopy and red-green birefringence under polarizing microscopy

[Figure 5].

Treatment and prognosis

One of the two patients with endotracheal amyloidosis received endotracheal stenting and was stable at 3-years follow-up. The other one died of respiratory failure 4 years later. Three of five patients who were diagnosed with endobronchial amyloidosis received thoracotomy with lobectomy, and two were stable at 5-years follow-up and 15-years follow-up, respectively, but one developed recurrence 9 months later and died of pneumonia and respiratory failure 18 months after surgery. The other two patients with endobronchial amyloidosis were only treated with glucocorticoid and antibiotics, and one was stable at 3-years follow-up and one died of pneumonia and respiratory failure 3 years later.

Two patients with solitary lung nodule underwent wedge resection by thoracotomy and thoracoscopy, respectively, and were followed up for 3.5 years and 8 years without recurrence. One patient with multiple nodules treated with glucocorticoid and antibiotics died of respiratory failure and pneumonia one year later. One patient with enlarged mediastinal lymph node

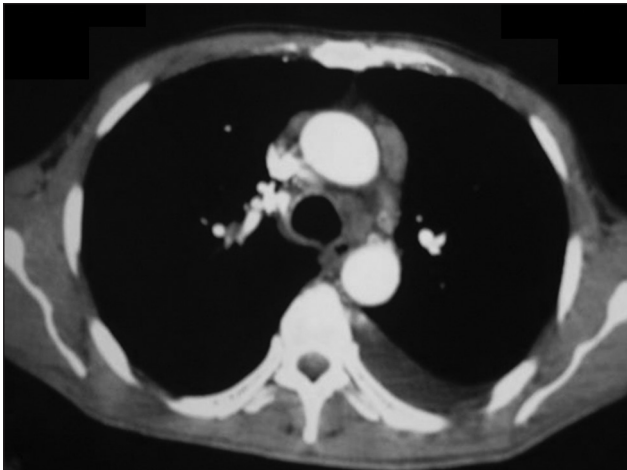


Figure 3: Computed tomography from a 49-year-old male (case no. 11) showing enlargement of mediastinal lymph nodes accompanied by bilateral pleural effusion



Figure 4: Bronchoscopy from a 52-year-old male (case no. 6) showing congestion, edema, and nodular deposits at mucosa of left lower bronchus

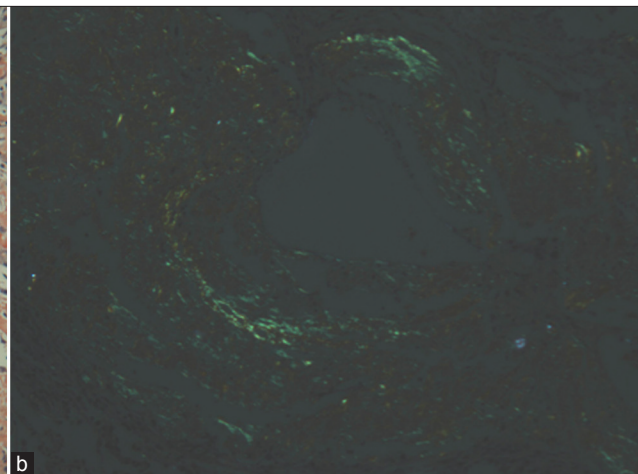
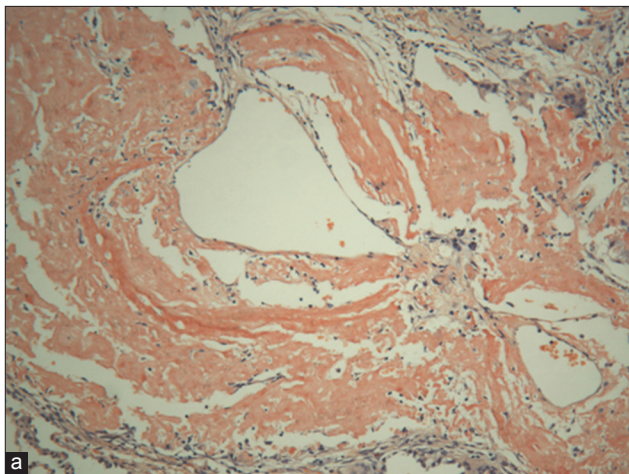


Figure 5: Histopathological examination of biopsy specimen from a 52-year-old male (case no. 6). (a) Congo red staining showed saffron yellow substances under light microscopy ($\times 100$). (b) Under polarized microscopy, green amyloid is seen on Congo red-stained tissue ($\times 100$)

and bilateral pleural effusion underwent glucocorticoid and methotrexate treatment, but died of pneumonia 9 months later. The other patient with enlarged mediastinal lymph node and bilateral pleural effusion took traditional Chinese medicine, but died 5 months later. One patient with massive pulmonary consolidation [Figure 2a] was treated with glucocorticoid, melphalan and antibiotics; the lesion resolved 2 months later [Figure 2b] and at 1.5 years follow-up he was alive and well.

Discussion

Amyloidosis can be primary or secondary to other diseases including tuberculosis, syphilis, chronic renal disease, hypergammaglobulinemia, rheumatoid arthritis, malignancies, and multiple myeloma.^[1,2] Secondary amyloidosis seldom involves the lungs, but primary systemic amyloidosis frequently involves multiple systems including the heart, gastrointestinal tract, kidneys, and skin.^[1,2] Primary amyloidosis with involvement of only 1 system (localized amyloidosis) is uncommon, and disease with only respiratory system involvement alone is rare.^[7-11] In patients with primary respiratory amyloidosis, amyloid is deposited in the pulmonary parenchyma and submucosa of the bronchi and trachea, pleura and lymph nodes in the mediastinum, and hilum of lungs.^[11] Utz *et al.*^[7] retrospectively analyzed the clinical record of patients seen at the Mayo Clinic over a 13-year period (1980–1993), and of 55 patients with pathologically proven amyloidosis only 11 had the disease confined to the respiratory system.

Primary pulmonary amyloidosis is classified into 4 types according to the site of the lesions^[7]: tracheobronchial amyloidosis; nodular amyloidosis; infiltrating interstitial amyloidosis, and lymph node amyloidosis. Tracheobronchial amyloidosis is the most frequently seen in clinical practice, and the clinical manifestations included dyspnea, chest tightness, cough, expectoration, and hemoptysis.^[7,8] Chest radiographs and CT show increased lung markings, obstructive pneumonia and atelectasis, localized or diffuse stenosis of the trachea, thickened trachea and bronchus and nodular shadows in the lumen with occasional calcifications. Bronchoscopy shows single or multiple protrusions or generalized thickening of the bronchial wall and bronchial stenosis. The smooth protrusions have no nodules, are of different sizes, and are susceptible to bleeding on touch. Occasionally, the protrusions may obstruct the bronchus resulting in secondary infection. In some cases, the whole submucosa of the bronchus is infiltrated with amyloid substance leading to bronchial stenosis. In the present study, 7 patients developed tracheobronchial stenosis (53.8%) and their clinical manifestations were similar to previously reported.

Patients with nodular amyloidosis usually have a cough or hemoptysis, and imaging findings show single or multiple nodules or patchy shadows, and involvement of other organs is seldom found.^[12] In the present study, 3 patients had nodular amyloidosis, 2 of whom received surgical resection of solitary nodular lesions.

Infiltrating interstitial amyloidosis is characterized by massive deposition of amyloid substances in small blood vessels and the pulmonary interstitium.^[13] High resolution CT shows thickening

of interlobular septum, network-like blurred shadows, or multiple subpleural nodules (2–4 mm in size). Lymph node amyloidosis is characterized by deposition of amyloid substances in lymph nodes in the mediastinum and pulmonary hilum.^[14] The condition is rarely seen in patients without systemic amyloidosis, and was found in only 2 patient in the present study.

Pathological examination of a tissue specimen is the gold standard for the diagnosis of amyloidosis. Under a light microscope, hematoxylin & eosin (H&E) staining shows homogeneous, pink amyloid substances without cellular structure that are usually accompanied by fibrosis, and characteristic red-green birefringence is seen under polarizing microscopy in Congo red-stained tissue.^[2] The edge of cord or mass-like amyloid substances were blur and their outlines become light gradually.

The principles in the treatment of amyloidosis are to inhibit the synthesis of amyloid and its extracellular deposition, reduce the production of amyloid precursors, and promote the degradation of amyloid.^[1,2] To date, there are no completely effective therapies and the effectiveness of glucocorticoid, vitamin E, colchicine, and other drugs is not clear.^[11] Resection of intratracheal and bronchial lesions with Nd-YAG laser or endoscopic clipping has been reported.^[10] External beam radiation therapy has also been attempted with promising results, in spite of the underlying mechanisms.^[15] Systemic chemotherapy consisting of 0.15 mg/kg/d melphalan plus 20 mg/d prednisone for 4 weeks (MP regimen) is one of the most commonly used treatments and is believed to inhibit the production and deposition of amyloid.^[8] In the present study, only 1 patients were treated with the MP regimen with good results. Kyle *et al.*^[16] treated patients with primary systemic amyloidosis with colchicine ($n = 72$), MP regimen ($n = 77$), or MP regimen in combination with colchicine ($n = 71$) and reported median survival times of 8.5 months, 18 months, and 17 months, respectively.

The primary limitation of this study is its retrospective nature. However, amyloidosis involving only the respiratory system is very rare and this report is of a relatively large number of patients.

Conclusions

In summary, isolated pulmonary amyloidosis is a rarely seen disease and its manifestation are numerous making diagnosis challenging. A high index of suspicion is required to arrive at a prompt diagnosis. Outcomes are varied, and surgical removal of lesions and chemotherapy can be effective.

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