Received: 4 December 2022 | Accepted: 30 March 2023

DOI: 10.1002/rcr2.1142

CASE REPORT

Respirology Case Reports OPEN ACCESS APSR WILEY

Ultrasound-controlled transthoracic true-cut needle biopsy in pulmonary nodular amyloidosis

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Associate Editor: Semra Bilaceroglu

Abstract

The current case report presents a 59-year-old man with imaging studies of the thorax showing nodular lesions in the lungs bilaterally. Based on radiographic and CT images, preliminary diagnoses for possible granulomatosis (tuberculosis) or pulmonary metastatic dissemination of a neoplastic process were made. An ultrasound-controlled transthoracic true-cut needle biopsy of a subpleural lesion was performed. Special staining with Congo red and examination with a polarizing light microscope for detection of amyloid confirmed the diagnosis of 'pulmonary nodular amyloidosis' by visualizing green birefringence.

KEYWORDS
pulmonary amyloidosis, true-cut biopsy, ultrasound-controlled

INTRODUCTION

The current article presents a rare clinical case of pulmonary nodular amyloidosis verified histologically surgically and by ultrasound-guided transthoracic true-cut needle biopsy. In terms of differential diagnosis, granulomatoses, malignancies of the lungs and systemic diseases leading to amyloid deposition in different organs should be considered. In the presented case report amyloidosis involvement of only one organ was demonstrated.

CASE REPORT

A 59-year-old man reported complaints of very easy fatigue and severe weakness and was admitted to a pulmonary clinic in November 2019. Earlier in September 2019 a surgical intervention of the cervical vertebrae was performed, and the imaging studies of the thorax showed nodular lesions in the lungs bilaterally. To the surgical team, these changes appeared suspicious for a pulmonary neoplasm, and the patient was sent to the Department of pulmonary diseases. In the pulmonology unit fiberbronchoscopy was undertaken due to this suspicion of a proliferative process of the lung with bilateral pulmonary disseminations. Endoscopically normally movable true vocal cords were visualized, no pathological changes in the larynx and trachea were detected, carina and the left part of the tracheobronchial tree applied also normal, and the presence of a second dorsal bronchus was observed on the right. Some clip biopsies, fine-needle aspiration biopsies and bronchoalveolar lavage were performed. Pathohistologically squamous cell metaplasia of the covering epithelium and a chronic nonspecific inflammatory process were veryfied, with no data directing to granulomatous inflammatory or neoplastic process. The special colouring of the specimen for fungi (Grocot) did not find any mycosis structures. The chest X-ray performed on the patient in September 2019 was highly suspicious of the presence of malignancy and this suspicion remained even though the bronchoscopy, which could not prove the

presence of neoplastic cells. Another invasive procedure for taking histological material needed to be done to reject a neoplasm.

In the anamnestic data was reported that in May 2009 because of an episode of consumptive syndrome and shortness of breath, a chest radiograph was made to the patient, describing bilateral pulmonary changes. Subsequent fiberbronchoscopy with trans-bronchial biopsies was suspicious for squamous cell carcinoma. The CT scan of the thorax at that time described a lesion with dimensions 20/30 mm in the right lung, at the top dorsally, with a central area of enlightenment and peripheral calcifications, and the same was interpreted as a probable tuberculoma. As a possible reason for the consumptive syndrome pulmonary tuberculosis was suspected. A thoracotomy with excisional biopsy of the right lung was achieved and 2 lesions were removed in the 4-th and 8-th segments near the interlobar surfaces, with dense elastic granular consistency, diameters 30/40 mm, whitish view and the same were easily separated from the surrounding parenchyma. The definitive microscopic specimen of the lesions verified pulmonary nodular amyloidosis and pulmonary emphysema. Mantoux test was then reported 8 mm, normergic, the observation of the patient continued in an outpatient setting, no presence of tuberculosis was proven. The man had a history also of abdominal surgery for a perforated duodenal ulcer many years ago and 35 package years of tobacco smoking. He had worked as a builder in a dusty environment. Regarding his family history, he reported for a father who died of oesophageal cancer.

Upon the next admission to the pulmonary clinic in February 2020 for the continuation of the diagnostic process because of the suspicion of possible neoplastic disease, the patient was in a relatively good general condition, with no evidence of respiratory failure. The incoming chest X-ray from February 2020 (Figure 1) demonstrated emphysematous changes in the lung parenchyma with multiple shadows of various dimensions scattered on both sides, some with higher density and calcifications, there was no difference with the one presented from November 2019. The presented CT of the thorax and abdomen done in November 2019 before the fiberbronchoscopy (Figure 2) showed evidence of multiple emphysematous bullae and bilaterally diffusely scattered focal lesions, the larger ones with spicules along the contour, all with macrocalcifications, one with shell-type calcification. Several of the lesions were smaller and completely calcified. No enlarged hilar and mediastinal lymph nodes were visualized. Compared to the CT scan from 2009, there was a progression in the emphysema, and some of the lesions appeared with more calcium deposits. Based on radiographic and CT images, preliminary diagnoses for possible granulomatosis (tuberculosis) or a pulmonary metastatic dissemination of a neoplastic process were made. The Mantoux test was again reported normal.

The chest ultrasound examination undertaken on the patient represented a pathological subpleural located formation in the left lung between the anterior axillary and mediolavicular line, 32/26 mm in size, respiratory mobile, in which a blood flow was not clear enough visualized (Figure 3A, B). An ultrasound-controlled transthoracic true-cut needle biopsy (UC-TTNB) of the described lesion was performed under local anaesthesia with 1% Lidocaine solution and using 18G/20mm needle. The manipulation was preferred due to its minimally invasive nature during the lung parenchymal biopsy, performed under direct visual real-time control. Although the previously known and surgically proven pulmonary amyloidosis, the clinicians needed to exclude as a possible reason for this disease an underlying and undiscovered another primary disorder, causing possible secondary amyloidosis, for example a neoplasm. There was a 10 year period in which the patient was not closely followed. The pathohistological result from the performed biopsy (Figure 4) was verifying a tissue cylinder, represented by amorphous eosinophilic amyloid-like material and small areas of preserved alveolar septa with focal pneumocytic

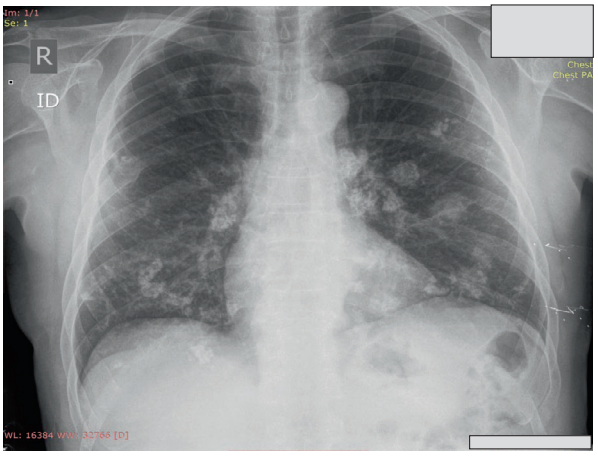

FIGURE 1 Chest X-ray at the admission in the pulmonary clinic with many diffuse bilateral nodular opacities

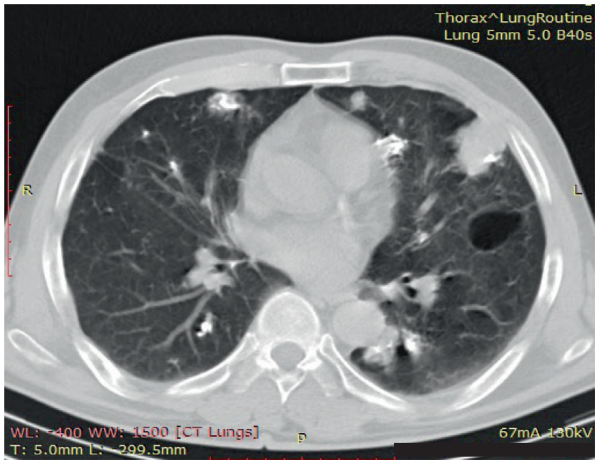

FIGURE 2 CT-scan of the patient, performed before the hospital admission, showing zones of consolidation in the lungs with calcifications and bullae

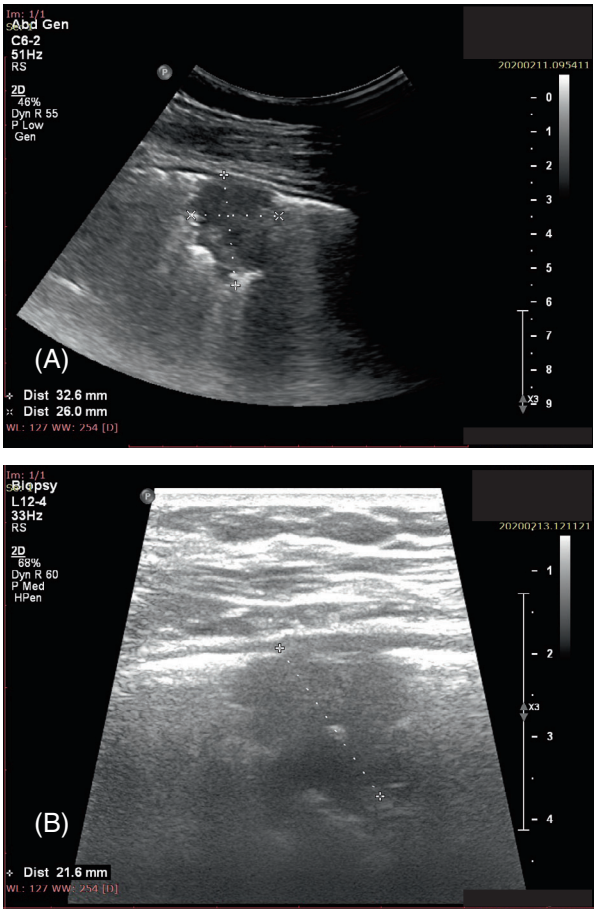

FIGURE 3 (A) The transthoracic ultrasound picture of a small subpleural nodular amyloid lesion in the left lung of the patient. (B) The moment of the performance of the ultrasound controlled transthoracic true cut needle biopsy of the same lesion

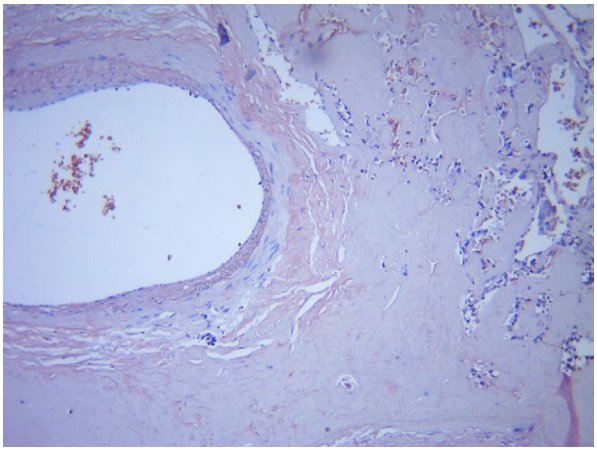

FIGURE 4 Haematoxylin – eosin staining of the amyloid deposits in the specimen

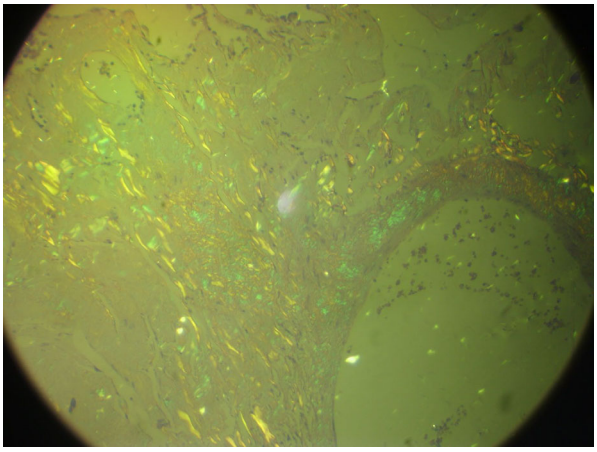

FIGURE 5 Congo red staining of the histological specimen visualizing green birefrigence under polarizing light. No neoplastic structures were observed

hyperplasia, small inflammatory foci formed by single lymphocytes and plasmatic cells. Special staining with Congo red and examination with a polarizing light microscope for detection of amyloid confirmed the diagnosis of 'pulmonary nodular amyloidosis' by visualizing green birefringence (Figure 5).[1] No neoplastic structures in the specimen were observed. A chest ultrasound due to contrast enhancement with the contrast medium sulfur hexafluoride (SonoVue) was performed in order to analyse the behaviour of this type of lesion when a contrast agent is applied (Figure 6). Subpleural consolidations bilaterally were found, with dimensions up to 35/35 mm, with preserved air bronchogram in the periphery, with disorganized structure of the parenchyma centrally. The lesion in the left lung parasternally, previously observed and biopsied by UC-TTNB, demonstrated a late contrast time to enhancement in the peripherial region and a central hypocontrasted zone without perfusion, the same respectively representing the area of the deposited amyloid.

A thorough ultrasound examination of the heart and the abdominal organs was also made. The performed echocardiography established normal sizes of the heart structures, with no evidence of pulmonary hypertension. The liver showed an upper-border dimension and a homogeneous, slightly hyperechoic structure, portal vein's diameter was measured 11 mm. Gallbladder, ductus choledochus and pancreas were normal. Both kidneys appeared with preserved cortical echogenicity. The examination did not show any pathological deposition of amyloid substances in other organs except the lungs. From the laboratory tests performed on the patient during the hospital stay no significant deviations in their parameters were found. Urine tests did not confirm the presence of renal involvement due to amyloidosis, Bens-Johnes' protein urine test was also negative. Given the possibility of an undiagnosed indolent lymphoma (MALT—lymphoma or marginal cell lymphoma) with light chain production, it was considered appropriate for the patient a serum immunofixation test.[2] The study did not reveal any evidence of paraproteinemia and a normal distribution of albumin and globulin regions was observed during the fixation with a polyvalent fixing solution. Immune

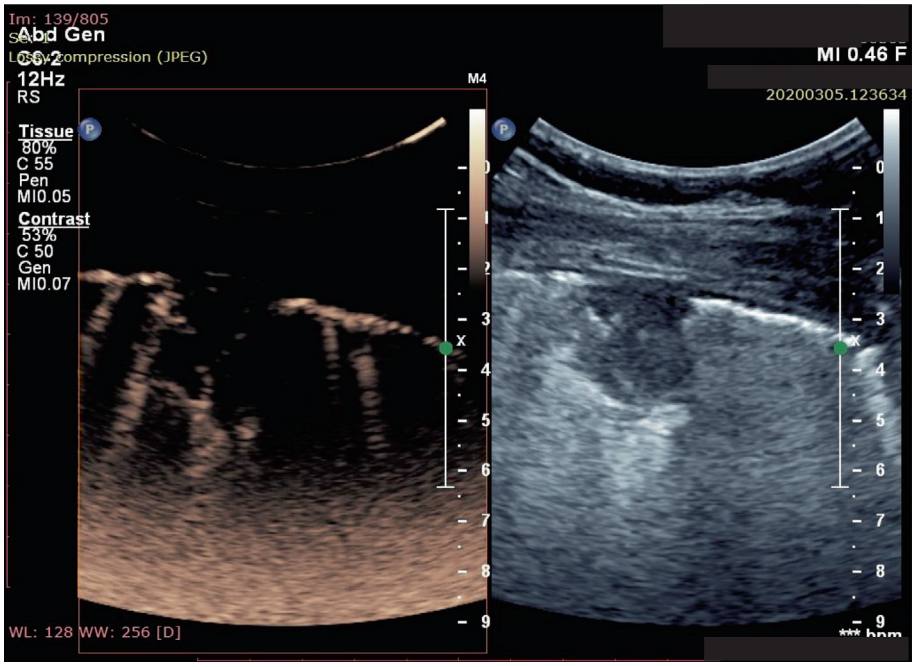

FIGURE 6 Chest ultrasound with contrast enhancement using the contrast medium sulfur hexafluoride (SonoVue). The lesion demonstrated a late contrast time to enhancement in the peripherial region and a central hypocontrasted zone without perfusion, the same respectively representing the area of the deposited amyloid

precipitates of IgG, IgA, IgM, as well as light chains of both types showed no qualitative changes.

Laboratory, imaging and invasive biopsy investigations could not reveal the presence of malignant disease, no clinical data were available to support the suspicion of a systemic connective tissue disease. As a final diagnosis the monoorganic localization of amyloidosis was accepted—respectively, pulmonary nodular amyloidosis. Nowadays the patient remains under the continuous supervision of a general practitioner and the annual examinations of a pulmonologist by monitoring of his lung function, X-ray image and ANA—titre, until now without any dynamics in direction deterioration.

DISCUSSION

The current case presents the diagnostic process in the rare disorder pulmonary nodular amyloidosis, established in the patient in 2009 by excisional surgical biopsy and confirmed in 2020 after a UC-TTNB of a small, subpleural localized lung lesion. The precisely performed UC-TTNB of the pulmonary lesion verified the same histological result as the previously performed thoracotomy, sparing the patient a new major interventional procedure under general anaesthesia and rejecting two differential diagnoses. The patient was subsequently examined in the conditions of transthoracic contrast-enhanced ultrasound in order to analyse the behaviour of this type of lesion when a contrast agent is applied. All imaging and laboratory tests did not prove the presence of systemic amyloidosis involvement, and the hypotheses of the presence of neoplastic disease or tuberculosis were not confirmed. No clinical data from the patient were available to support the thesis for a systemic connective tissue disease. At this stage, he remains still under the supervision of pulmonologist and general practitioner.

Amyloidosis is a disease caused by conformational changes and extracellular accumulation of autologous proteins that deposit in tissues in the form of aggregates of fibrils, all of them sharing a common ultrastructure.[3–5] It can be idiopathic (primary form) or associated with various inflammatory, hereditary or neoplastic pathogeneses (secondary or reactive form).[6] The disease can be a manifestation of a widespread process, involving many organs, or it may be localized to one organ, for example – the lung. Almost 15 forms of systemic amyloidosis are known and classified according to the different amyloidogenic precursor proteins.[7] The estimated incidence of systemic amyloidosis is nearly 10 cases per million people per year, ranging it among the rare diseases.[8] Unlike systemic amyloidosis, localized pulmonary involvement usually follows a benign course, is rarely symptomatic and males are more affected than females, with mean age of onset 55–60 years.[9,10]

The systemic disease can be caused by increased synthesis of amyloid proteins, such as in chronic inflammation (apolipoprotein serum amyloid A), plasma cell dyscrasias (monoclonal immunoglobulin light chains, forming amyloid AL), malignancies such as medullary thyroid carcinoma,[2] as genetic disorders like hereditary amyloidosis (due to mutated transthyretin, ATTRm) or senile amyloidosis (acquired, age-related, caused by the wild-type transthyretin,

and ATTRwt). The diagnosis of amyloidosis is based on tissue biopsy and Congo red staining.[1]

Respiratory manifestations can be observed both in systemic and localized forms of amyloidosis. Amyloidosis can appear in the lung in three different forms: nodular pulmonary amyloidosis with single or multiple nodules in the lung parenchyma, diffuse alveolar-septal (interstitial) amyloidosis and tracheobronchial amyloidosis with submucosal deposits in the airways.[1,10–13]

Nodular pulmonary amyloidosis is a usually localized form which is an incidental finding on chest radiography, defined as one or more nodular amyloid deposits involving the lung, usually consisting on immunoglobulin light chain deposits or mixed light chain/heavy chain ones.[14,15] Some rare cases of systemic amyloidosis with lung nodular involvement are reported.[16–18] Nodulary amyloidosis usually presents with peripheral subleural bilateral localizations of variable size.[19] Amyloid nodules are generally localized to the lower lobes, in the peripheral and subpleural areas.[20] They have four characteristic features on CT: (1) Sharp and lobulated contours; (2) Calcification, often central or in an irregular pattern within the nodule (seen in about 50% of cases)[10,12,13,20,21]; (3) Multiple shapes and sizes varying from 0.5 to 15 cm,[12,21] and (4) Slow growth, often over years, with no regression.[10,12,20] Cavitation is very rare.[22] The prognosis is generally very good.

In some cases, nodular pulmonary amyloidosis can result from an underlying lymphoproliferative disorder in the spectrum of extranodal marginal zone lymphoma of mucosa-associated lymphoid tissue (MALT lymphoma).[14] The underlying lymphoproliferative disorder may be subtle (indolent and mildly symptomatic), but sensitive methods reveal a clonal B-cell population in most cases, the same expressing light chains forming the amyloid deposits.[14,23] The finding of monotypic lymphoid cells on immunohistochemical analysis confirms the diagnosis of lymphoma. The localized AL amyloid differs from its systemic counterpart by the morphological appearance of the amyloid and the presence of clonal plasma cells and giant cells. The κ light chains in localized form are more frequent than the λ chains, in contrast to the systemic form.[14] Histologically, the nodules are well-circumscribed and are composed of homogeneous, densely eosinophilic material, with small aggregates of lymphocytes and plasma cells adjacent to them.

Sjögren's disease was found to be associated with pulmonary amyloidosis and lymphoproliferative disorders. It manifests with multiple pulmonary large bullae, multiple nodules, parenchymal opacity and bronchiectasis, so this differential diagnosis should be also considered.[24,25]

Diffuse alveolar-septal amyloidosis is also known as diffuse parenchymal amyloidosis, characterized by the presence of amyloid deposits in the alveolar septa and vessel walls. Usually, it is a manifestation of the systemic form, but localized cases are also reported.[1,26,27]

The pulmonary impairment rarely dominates the clinical picture, so most often the diffuse alveolar-septal amyloidosis is a post mortem finding, especially in systemic AL amyloidosis. The pathohistological presentation demonstrates widespread amyloid deposition in the small vessels and the interstitium, with affection sometimes of all pulmonary lobes and the visceral pleura. The lesions are typically hypocellular, but scant plasma cells may be present.[1] Reticular opacities, interlobular septal thickening, micronodules, ground-glass opacification, traction bronchiectasis and honeycombing can be visualized at high-resolution computed tomography (CT), eventually accompanied by mediastinal lymphadenopathy and pleural effusions.[22,26] Radiologically the described anomalies show the pattern of nonspecific diffuse interstitial or alveolar opacities (confluent consolidations located predominantly in the subpleural regions), remaining stable over time.[28] The interlobular septal thickening frequently have also a predominant basilar and peripheral distribution.[22] Small well defined nodules (2–4 mm in diameter) are also detected, some of them showing calcifications.[21]

The clinical presentation of the diffuse pulmonary amyloidosis is more severe, because the fibril deposits affect the interstitium and disturb the gas exchange due to alveolar membrane and capillary damage.[1,28] The patients complain from dyspnoea with pulmonary origin because of the progressive character of the interstitial lung disease. The high resolution CT represents an infiltrative imaging pattern, sometimes also cysts and calcifications.[29] As a differential diagnosis cardiac failure needs to be excluded due to either congestive or restrictive cardiomyopathy. Vascular deposits can be the reason for the development of pulmonary hypertension—primary by arterial deposits in the media, or secondary because of left-sided restrictive cardiomyopathy or the diffuse lung involvement.[30–32]

Interstitial amyloidosis occasionally occurs as a consequence of lung infiltration of B-cell malignancies[30,33] producing amyloidogenic monoclonal protein, as well as in rare cases of lung metastases of medullary carcinoma of the thyroid.[34] The diffuse form of the disease is treated according to the underlying systemic amyloidosis, current treatment derives from chemotherapy schemes developed for multiple myeloma, but not with the same success.[35]

Tracheobronchial amyloidosis is an organ-limited amyloidosis, and is most often presenting as multifocal submucosal plaques, usually not associated with detectable systemic lymphoplasmocytic clonal proliferation.[26,33,36–39] The association with multiple myeloma is extremely rare.[40] The pulmonary parenchyma is typically not involved, but colocalisation of laryngeal, tracheal and bronchial amyloidosis has been described, with fixed airflow obstruction at spirometry due to stenosis from the amyloid deposits.[41] Patient may be asymptomatic, or may present with dyspnoea, cough and haemoptysis. Narrowing of airways can cause wheezing, distal atelectasis, recurrent pneumonia or lobar collapse, and solitary nodules may be mistaken for endobronchial neopasia.[42,43] Possible calcification can cause tracheal and bronchial wall thickening, which can be observed at CT scan.[44,45] The fiberbronchoscopy demonstrates irregular whitish deposits in the airways, most often

diffuse, narrowing the airway lumen more or less completely (multifocal submucosal plaques, associated with plasma cells and giant cells), and very fragile.[36] Squamous cell metaplasia may affect the epithelium and could be confused with carcinoma.[46] The management of this form of amyloidosis includes treatment with a laser of forceps debridement or external beam radiation, which can suppress the responsible clonal B-cells within the tissue.[44,47,48]

Amyloidosis of the pleura is associated with effusions refractory to maximal diuretic therapy and thoracentesis, possibly because of impairment of resorption of pleural fluids. As a diagnostic procedure, percutaneous or thoracoscopic pleural biopsy may be considered.[49,50]

Despite the current advances in the diagnosis and pathogenetic aspects of amyloidosis, many issues concerning this pathological unit remain unresolved. There is still no clear and specific treatment algorithm.

AUTHOR CONTRIBUTIONS

Teodora Z. Mihalova and Rosen E. Petkov are responsible for the initial draft of this manuscript. All authors provided substantial contributions to the conception of the work as well as critical revision for the initial draft and the revised draft. All authors also provided final approval for this manuscript.

CONFLICT OF INTEREST STATEMENT

None declared.

DATA AVAILABILITY STATEMENT

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

ETHICS STATEMENT

The authors declare that appropriate written informed consent was obtained for the publication of this manuscript and accompanying images.

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How to cite this article: Mihalova TZ, Ivanova SS, Mekov EV, Yamakova YT, Petkov RE. Ultrasound-controlled transthoracic true-cut needle biopsy in pulmonary nodular amyloidosis. Respirology Case Reports. 2023;11:e01142. https://doi.org/10.1002/rcr2.1142