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Towards greater goals...

International ILD Summit

13-15 June 2025

Radisson Blu Bloomsbury Hotel
London - United Kingdom

ABSTRACT BOOK

ildsummit2025.org

INVITATION

Welcome to the International ILD Summit

June 13–15, 2025 | London

It is our great pleasure to welcome you to the International Interstitial Lung Disease (ILD) Summit, a scientific meeting convening global experts in the field of diffuse parenchymal lung diseases. This summit aims to foster meaningful academic exchange, promote interdisciplinary collaboration, and highlight the most recent advances in ILD research, diagnostics, and therapeutics.

Over the course of the meeting, participants will engage with a diverse program featuring high-level scientific presentations and interactive sessions that span the full spectrum of ILD—from early detection and imaging to novel therapies and patient-centered approaches. We are honored by your participation and confident that this summit will serve as a valuable forum for advancing both scientific knowledge and clinical excellence in ILD care.

Sincerely,

Prof. Funda Coşkun
Bursa Uludağ University Faculty of Medicine

Dr. Richard Hewitt
Royal Brompton Hospital, London
Summit Co-Chairs

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Dr. Züleyha Bingöl, Prof, İstanbul University, Capa Medical Faculty, Pulmonology Department

SCIENTIFIC PROGRAM

June 13, 2025 – Friday	
09:00-09:30	Opening Ceremony
Radiology Course **Session Chair: Peter George, Çetin Atasoy**	
09:30-10:00	Classification of Idiopathic Interstitial Pneumonias – Ahmet Ursavas
10:00-10:30	Technical Features and Required Anatomy in HRCT Evaluation – Simon Padley
Break	
Session Chair: Funda Coskun, İsmail Hanta	
11:00-11:30	Early diagnosis of ILD – Richard Hewitt
11:30-12:00	UIP/NSIP – Çetin Atasoy
Lunch	
Session Chair: Joe Jacob, Hasti Robbie	
13:30-14:00	Radiology of Connective Tissue Diseases – Hasti Robbie
14:00-14:30	Rare Lung Diseases – Cetin Atasoy
14:30-15:00	Artificial Intelligence and ILD – Joe Jacob
June 14, 2025 – Saturday	
Session Chair: Richard Hewitt, Brintha Selvarajah	
09:00-09:30	Pathophysiology: From Inflammation to Fibrosis – Brintha Selvarajah
09:30-10:00	Clinical Diagnosis of ILDs – Funda Coşkun
10:00-10:30	Defining Progression & Impact of 2022 PPF Guideline on Clinical Practice (Pulmonologist Perspective) – Göksel Altınışık
Break	
Session Chair: Gamze Kırkıl, Katherine Myall	
11:00-11:30	CTD-ILD: Importance of Early Diagnosis – Katherine Myall
11:30-12:00	SSc-ILD: Optimal Patient Management – Ender Terzioğlu
12:00-12:30	IPF/PPF: Optimal Patient Management – İsmail Hanta
Lunch	

13.00-14.00	**Poster Session**
Therapeutics Session	
Session Chair: Murat İnanç, Kemal Can Tertemiz	
14:00-14:30	Treatment of IPF/PPF – Gülfer Okumuş
14:30-15:00	Treatment of CTD-ILD – Murat İnanç
15:00-15:30	Future Drugs – Sevda Şener Cömert
Break	
Session Chair: Ahmet Ursavas, Nilgün Yılmaz Demirci	
16:00-16:30	Rare Lung Diseases 1: Eosinophilic Lung Diseases and Drug-Induced Lung Disorders – Berna Akıncı Özyürek
16:30-17:00	Rare Lung Diseases 2: Cystic Lung Diseases – Ahmet Ursavaş
June 15, 2025 – Sunday	
Keynote Speech	
09:00-09:30	"The Future of ILD Research: New Horizons" – Gisli Jenkins
Session Chair: Züleyha Bingöl, Muzaffer Onur Turan	
09:30-10:00	Treatment Challenges in Severe IPF Patients – Dildar Duman
10:00-10:30	Treatment of Group 3 Pulmonary Hypertension – Zuleyha Bingöl
Break	
Session Chairs: Elif Yelda Niksarlıoğlu, Talat Kılıç	
10:40-11:10	Sarcoidosis – Özlem Özdemir Kumbasar
11:10-11:30	Radiology of Hypersensitivity Pneumonitis – Sujal Desai
11:30-11:50	Hypersensitivity Pneumonitis – Can Sevinç
11:50-12:10	Rational Drug Use Session – Kemal Can Tertemiz
12:10	Closing

POSTER PRESENTATIONS

A Case of IPF (Idiopathic Pulmonary Fibrosis) Successfully Adhering to Antifibrotic Treatment through Adverse Effect Management

Fulsen Bozkuş, SBÜ Antalya Training and Research Hospital

Introduction: Idiopathic pulmonary fibrosis (IPF) is a chronic, progressive lung disease characterized by the irreversible deterioration of lung function and a short median survival time. It follows a course with age-standardized mortality rates ranging from 0.5 to 12 deaths per 100,000 individuals. The disease progression of IPF is unpredictable. While some cases exhibit a rapid decline, others progress slowly. Some cases may experience exacerbations, leading to shorter survival times. Scientific evidence accumulated over the past decade has altered the perspective on the disease by demonstrating the effectiveness of two medications in slowing down the loss of lung capacity. Currently, two antifibrotic agents are used in treatment. In initiating treatment and choosing the appropriate therapy, factors such as drug availability, side effect profiles, disease severity, comorbidities, and the clinician's experience play a crucial role, and a personalized treatment approach is preferred. Analyses have shown no difference between the two molecules in terms of mortality, hospitalization, and cost.

Case Report: A 65-year-old male patient, with no known history of lung disease, presented with complaints of dyspnea and cough. He reported experiencing exertional dyspnea for the past 5-6 years, which had become independent of exertion over the last year, now accompanied by a dry cough. The patient had a 35-pack-year smoking history but had quit smoking 8 years ago. His medical history was notable only for hypertension. On physical examination, his general condition was good, cooperative, and vital signs were stable. There was no edema, cyanosis, or clubbing. On respiratory system examination, bilateral velcro rales were present at both lung bases. Oxygen saturation in room air was 95%. Other system examinations were unremarkable. Laboratory findings revealed hemoglobin 14.2 g/dL, leukocytes 11,200, hematocrit 42.2%, platelet count 154,000, sedimentation 12, CRP 3.4, BUN 17 mg/dL, creatinine 0.68 mg/dL, AST 12 U/L, ALT 18 U/L, GGT 22 U/L, LDH 285 U/L, and calcium 8.5 mg/dL. Chest X-ray (Figure 1) showed a bilateral reticular pattern. High-resolution computed tomography (HRCT) of the thorax (Figure 2) revealed peripheral involvement with areas of ground-glass opacity, particularly prominent in the bilateral lower lobes, along with thickening of irregular interlobular septa, honeycombing, and traction bronchiectasis, consistent with usual interstitial pneumonia (UIP) pattern. According to the 2022 IPF guidelines, patients with a radiological UIP pattern can be diagnosed with IPF through a multidisciplinary evaluation. Therefore, the patient was diagnosed with IPF, and pirfenidone was initiated. The dose was titrated up to the full dose over a 3-week period. After reaching the full dose, the patient developed dyspeptic symptoms and nausea. As a result, the dose was reduced. However, the symptoms persisted, and the medication was discontinued until the symptoms fully resolved. Afterward, pirfenidone was reintroduced with a slower dose escalation, and when the full dose was reached, the

gastrointestinal symptoms were alleviated. Through proper management of side effects, the patient was able to continue treatment.

Conclusion: Regardless of whether the disease progresses rapidly or slowly, the management of IPF is challenging. This is due to the individual variability in patients' needs, which can change throughout the course of the disease. Treatment management requires a comprehensive approach, involving regular assessments and the application of both pharmacological and non-pharmacological treatment strategies. Currently, two antifibrotic medications are preferred in pharmacological treatment, and it is essential to be well aware of their potential side effects. In the presence of uncontrolled side effects, treatment should be discontinued. Therefore, effective management of side effects is crucial to ensure continued treatment and patient adherence in these cases.

DİFFUSE PULMONARY LYMPHANGİOMATOSİS İNVOLVİNG LUNGS AND MEDİASTİNAL SOFT TİSSUE

Gülşah Günlüoğlu, Gülşah Günlüoğlu, Nurdan Kalkan, Havvanur Özçelik
Istanbul Yedikule Chest Diseases and Thoracic Surgery Training and Research Hospital

Diffuse pulmonary lymphangiomatosis (DPL) is rare in adults. It is characterized by abnormal proliferation, dilatation, and thickening of the lymphatic channels in the lungs, pleura, and mediastinal soft tissue. there are no definitive therapies for DPL.In progressive disease, surgical drainage may be performed in patients with pleural fluid accumulation.
CASE PRESENTATION
29-year-old female patient with no pulmonary symptoms applied to our hospital for screening. Chest computed tomography (CT) revealed diffuse and smooth interlobular septal thickening, peribronchovascular thickening, and patchy ground glass opacities (GGO). On the mediastinal window of the CT soft density at the hilum of the lung extending to the esophagus was seen (Fig). No abnormalities were observed on physical examination and laboratory evaluation. Mediastinal excisional biopsy and wedge biopsy of the lung performed for diagnostic purposes. Histopathologically diffuse pulmonary lymphangiomatosis involving lungs and mediastinal soft tissue was diagnosed.
DISCUSSION
DPL is rare in adults. According to a literature review, the median age of patients diagnosed with DPL was 48-year-old, of whom 40% were men. The most common symptom is cough but asymptomatic cases are seen rarely. Due to the rarity and lack of knowledge of lymphangiomatosis, it has been difficult to establish a treatment. The treatments available are palliative and aimed at managing chylous fluid accumulations and proliferation of lymphatics and alleviate symptoms that come with compression of adjacent organs. Recently, it has been shown that propranolol, a nonselective beta-blocker, may be effective in treatment of diffuse lymphangiomatosis by reducing the levels of vascular endothelial growth factor (VEGF) and the amount of chylous effusions.

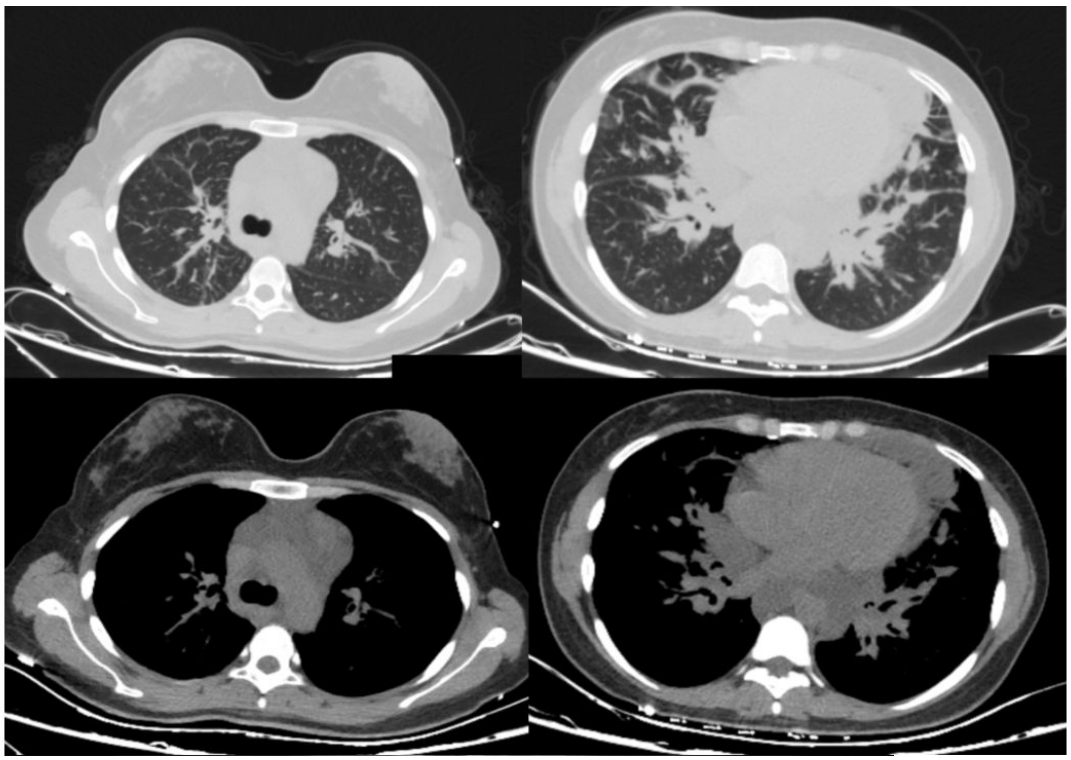

Figure. Chest computed tomography (CT) revealed diffuse and smooth interlobular septal thickening, peribronchovascular thickening, and patchy ground glass opacities (GGO). On the mediastinal window of the CT soft density at the hilum of the lung extending to the esophagus was seen

CATAMENIAL PNEUMOTHORAX IN A YOUNG WOMAN WITH PELVIC ENDOMETRIOSIS AND OVARIAN ENDOMETRIOMA: A RARE CASE

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1Department of Chest Diseases, University of Health Sciences Turkey , Yedikule Chest Diseases and Thoracic Surgery Education and Research Hospital - Istanbul (Turkey)

Introduction: Catamenial pneumothorax (CP) is a rare clinical entity characterized by recurrent spontaneous pneumothorax episodes, typically occurring on the right hemithorax in temporal association with menstruation. It is the most common manifestation of thoracic endometriosis syndrome and predominantly affects women of reproductive age, particularly those with a history of pelvic endometriosis. Diagnosis is primarily based on clinical suspicion supported by menstrual synchrony, imaging findings, and thoracoscopic identification of diaphragmatic or pleural lesions, although these may not always be evident. In terms of management, tube thoracostomy is the initial approach, while video-assisted thoracoscopic surgery (VATS) and adjunctive hormonal suppression are recommended to reduce recurrence. Herein, we present a case of CP diagnosed in a 32-year-old woman who presented with chest pain and dyspnea.

Case: A 32-year-old female presented to our outpatient clinic with complaints of chest pain and shortness of breath. She had a history of smoking (5 pack-years), but had ceased smoking two years prior. Professionally, she worked as a public relations specialist. Her past medical history was notable for pelvic endometriosis and ovarian endometrioma (chocolate cyst), with no regular medication use. On physical examination, the patient's vital signs were stable (SpO_2: 96%, pulse: 93 bpm). Auscultation revealed decreased breath sounds over the right posterior hemithorax. Digital clubbing and pretibial edema were absent. Pulmonary function testing showed normal values: FVC: 3.52 L (98%), FEV_1: 2.98 L (96%), FEV_1/FVC: 84%, with no bronchodilator response. Thoracic computed tomography (CT) revealed right-sided pneumothorax (Image1). The patient had experienced two previous episodes of spontaneous pneumothorax in March 2023 and May 2024, both temporally associated with her menstrual cycle and managed with tube thoracostomy.Based on clinical and radiologic findings, catamenial pneumothorax was suspected and the patient underwent VATS. During surgery, wedge resections were performed from the right middle and lower lobes. Histopathological examination of the resected lung specimens revealed bullous cystic changes, emphysema, chronic pleuritis, subpleural fibrosis, and evidence of both old and recent hemorrhage. Chronic nonspecific pleuritis was observed in the parietal pleura. Taken together, the clinical, surgical, and histopathological findings strongly supported a diagnosis of catamenial pneumothorax secondary to thoracic endometriosis.

Keywords: Catamenial pneumothorax, Pelvic endometriosis, Spontaneous pneumothorax, V ATS, Menstruation- related pneumothorax
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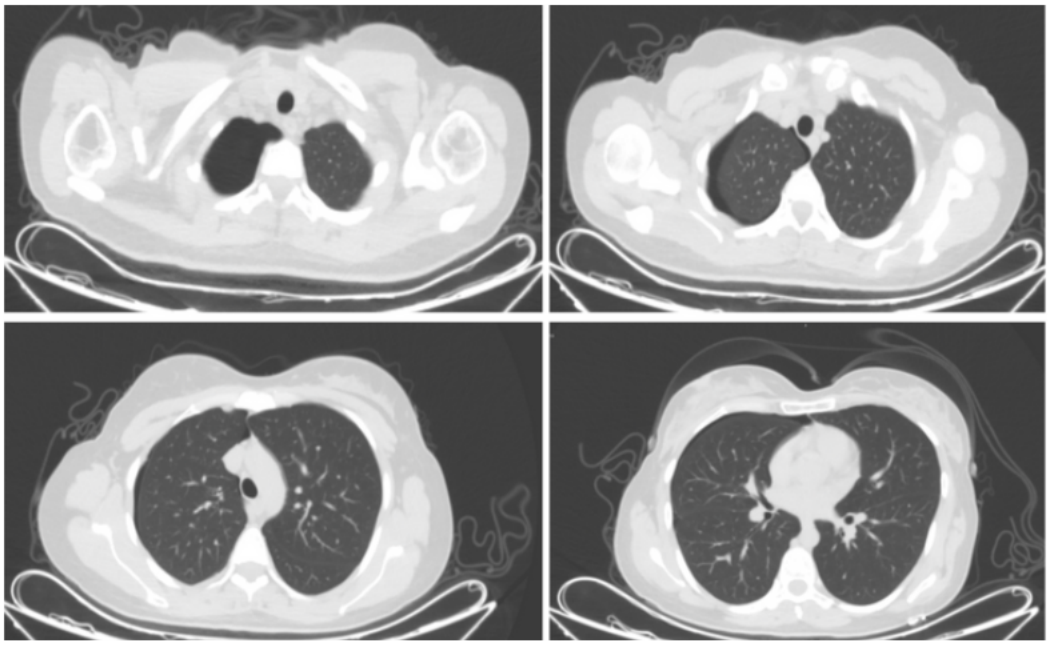

Discussion: Catamenial pneumothorax represents the most frequent manifestation of thoracic endometriosis and is characterized by spontaneous pneumothorax episodes that typically occur within 72 hours of menstruation onset. The present case exhibited hallmark features of CP , including right-sided pneumothorax temporally associated with menses and a documented history of pelvic endometriosis and ovarian endometrioma. Radiologic identification of CP can be limited, as was the case here, highlighting the importance of a thorough clinical history and surgical exploration. V ATS allowed for direct visualization and tissue sampling, confirming multiple findings consistent with thoracic endometriosis, including subpleural fibrosis, hemorrhage, and chronic inflammation. While definitive histological identification of endometrial glands and stroma may not always be possible, consistent morphologic and clinical findings can substantiate the diagnosis, as supported by previous literature. This case emphasizes the need to consider CP in women of reproductive age presenting with recurrent spontaneous pneumothorax, particularly when temporally related to menstruation. Multidisciplinary evaluation, including gynecologic consultation, is essential. V ATS not only serves as a diagnostic modality but also enables definitive surgical treatment. Postoperative hormonal suppression therapy is considered critical in preventing recurrence and improving long-term outcomes.

Evaluation of the Relationship Between Carotid Intima-Media Thickness, Lipid Profile, and Biochemical Parameters in Patients with Idiopathic Pulmonary Fibrosis (IPF) and a Healthy Population

Aydın balcı, Yaşar İnkaya
Afyonkarahisar Health Sciences University, Chest Disease Departmen

Background:
Idiopathic Pulmonary Fibrosis (IPF) is a progressive interstitial lung disease characterized by poor prognosis and increasing evidence of systemic involvement. Recent studies suggest that IPF may be associated with subclinical atherosclerosis and vascular dysfunction.

Objective:
To evaluate carotid intima-media thickness (CIMT), lipid profiles, atherogenic indices, and visual analog scale (VAS) scores in IPF patients compared to a healthy population, and to investigate their potential associations.

Methods:
This retrospective study included 117 IPF patients and 104 age- and sex-matched healthy controls. Participants underwent pulmonary function tests, laboratory evaluations, and carotid Doppler ultrasonography. CIMT, atherogenic indices (AC, CRR, AI), and VAS scores were analyzed.

Results:
CIMT and CRR were significantly higher in the IPF group, while HDL levels were lower and VLDL levels were higher compared to controls. Pulmonary function and 6-minute walk test distances were reduced in IPF patients. Significant correlations were observed between CIMT and 6MWT, and between VAS and several biochemical and respiratory parameters.

Conclusion:
Subclinical atherosclerosis and vascular risk may be elevated in patients with IPF. CIMT and atherogenic indices can serve as non-invasive markers for cardiovascular assessment. Symptom severity (VAS) appears to be associated with both pulmonary and vascular dysfunction, suggesting a need for holistic patient evaluation.

A Rare Case of Primary Amyloidosis Presenting with Pulmonary and Cardiac Involvement

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Introduction – Objective: Amyloidosis is a rare systemic disease characterized by extracellular deposition of abnormally folded precursor proteins. Various subtypes exist depending on the type of precursor protein involved, most commonly affecting the kidneys, liver, heart, nervous system, and gastrointestinal tract. Pulmonary involvement is rare and typically manifests as tracheobronchial amyloid deposits, parenchymal nodules, or diffuse interstitial infiltrates. Cardiac involvement, particularly in AL (primary) amyloidosis, is associated with poor prognosis.

Histopathological confirmation with Congo red positivity and apple-green birefringence under polarized light remains the diagnostic gold standard. In this case, we present a patient initially treated for presumed tuberculosis due to progressive dyspnea, weight loss, and interstitial lung infiltrates, who was ultimately diagnosed with pulmonary amyloidosis via cryobiopsy. Further evaluation revealed concurrent cardiac involvement, confirming a diagnosis of systemic AL amyloidosis associated with multiple myeloma. This case highlights a rare presentation of primary amyloidosis with both pulmonary and cardiac manifestations.

Case Report: A 59-year-old female presented to an external center with a 6-month history of exertional dyspnea, chest pain, and dry cough. Empirical antibiotics were initiated with no clinical improvement. Over the past month, she reported a 7 kg weight loss, totaling 20 kg in the past year. Chest radiography performed in Sivas revealed cardiomegaly and bilateral—predominantly right-sided—reticulonodular infiltrates, leading to the initiation of quadruple anti-tuberculosis therapy. After one month of treatment, she developed nausea, vomiting, and abdominal pain. Laboratory evaluation showed elevated transaminases, suggesting drug-induced hepatotoxicity, and therapy was discontinued. Following normalization of liver enzymes, treatment was restarted. However, two months into therapy, the patient experienced worsening dyspnea, profound fatigue, and hemoptysis, prompting admission to our center. Her medical history included diabetes mellitus and hypertension, for which she was on ramipril 5 mg/day. She had a 40 pack-year smoking history. On examination: blood pressure 135/80 mmHg, heart rate 88 bpm, respiratory rate 18/min, and SpO_2 96% on room air. Bilateral fine inspiratory crackles were audible on auscultation. Clubbing and peripheral edema were absent. Thoracic computed tomography (CT) revealed diffuse interlobular septal thickening observed in all regions, which was dominant in the upper and middle lobes, centrilobular nodules, bilateral predominantly lower lobes ground-glass opacities, and scattered areas of consolidation (Figure 1A-B). Given the inconsistency between clinical/radiological findings and tuberculosis, further investigations were pursued. Bronchoscopy revealed patent airways bilaterally; bronchoalveolar lavage (BAL) cultures, PCR, and cytology were all negative. Rheumatologic and vasculitic markers were also unremarkable. Laboratory findings included ESR: 106 mm/h, and serum ACE: 96 U/L.

Due to persistent interstitial infiltrates, rigid bronchoscopy with fluoroscopic-guided cryobiopsies was performed from the anterior and posterior segments of the right upper lobe. Histopathology revealed thickened alveolar septa and vascular walls with amorphous eosinophilic material forming occasional nodules, Congo red-positive, and exhibiting apple-green birefringence under polarized light—findings consistent with pulmonary amyloidosis.

Further etiological evaluation showed a free kappa/lambda ratio of 8.27 (kappa: 83.6 mg/L; lambda: 10.1 mg/L). Bone marrow biopsy demonstrated 15% CD138-positive plasma cells expressing kappa light chains with Congo red-positive material in interstitial and vascular compartments—diagnostic of AL amyloidosis (Figure 2). PET- CT showed no evidence of plasmacytoma or lytic lesions. Subcutaneous fat aspiration was negative for amyloid. Cardiac workup revealed elevated pro-BNP (1552 pg/mL) and troponin T (17.66 pg/mL). Echocardiography showed concentric left ventricular hypertrophy and granular thickening of the interventricular septum—suggestive of cardiac amyloidosis.

A multidisciplinary board confirmed the diagnosis of systemic AL amyloidosis with pulmonary and cardiac involvement associated with multiple myeloma. Treatment was initiated with Daratumumab, Bortezomib, Cyclophosphamide, and Dexamethasone, along with tetracycline as an anti-amyloid adjunct.

Discussion – Conclusion: Amyloidosis is a rare disease with systemic manifestations, commonly affecting the kidneys, heart, and gastrointestinal tract. Pulmonary involvement is uncommon and often diagnosed late. As demonstrated in this case, pulmonary amyloidosis may mimic interstitial lung diseases and be misdiagnosed as tuberculosis, malignancy, or connective tissue disorders. Our patient was initially treated for presumed tuberculosis, with no improvement, prompting further workup. Cryobiopsy is crucial in confirming pulmonary amyloidosis, with Congo red positivity and birefringence under polarized light being diagnostic hallmarks. Concomitant cardiac involvement was identified, leading to a final diagnosis of AL amyloidosis. Cardiac involvement, frequently seen in AL amyloidosis, significantly worsens prognosis. Granular myocardial appearance and elevated cardiac biomarkers were key diagnostic clues in this case.

Early diagnosis and initiation of therapy are essential in primary AL amyloidosis. In our case, associated with multiple myeloma, chemotherapy and anti-amyloid therapy were promptly started. The case underscores the high diagnostic yield of minimally invasive techniques like cryobiopsy in suspected interstitial lung disease and highlights the importance of considering amyloidosis in the differential diagnosis.

A comprehensive evaluation, including cardiac assessment, and a multidisciplinary approach are fundamental to effective patient management in systemic amyloidosis.

Keywords: Amyloidosis, Pulmonary Amyloidosis, Cardiac Amyloidosis, Interstitial Lung Disease, Cryobiopsy, AL Amyloidosis, Multiple Myeloma

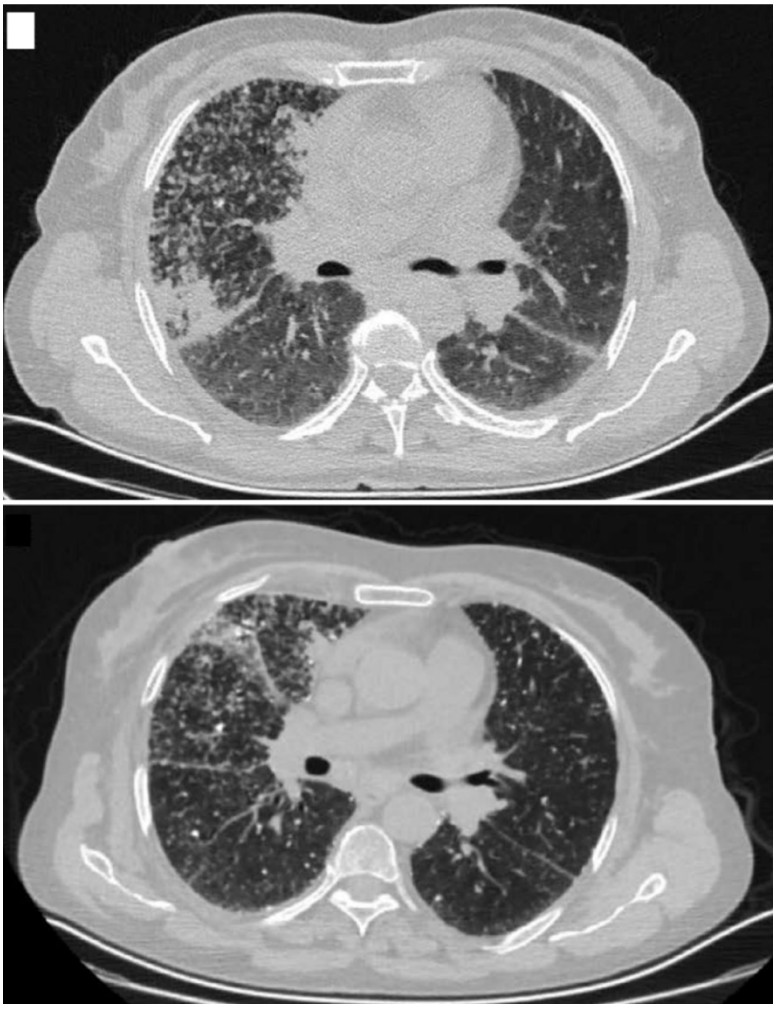

Figure 1A-B: Thoracic computed tomography (CT) revealed diffuse interlobular septal thickening observed in all regions, which was dominant in the upper and middle lobes, centrilobular nodules, bilateral predominantly lower lobes ground-glass opacities, and scattered areas of consolidation

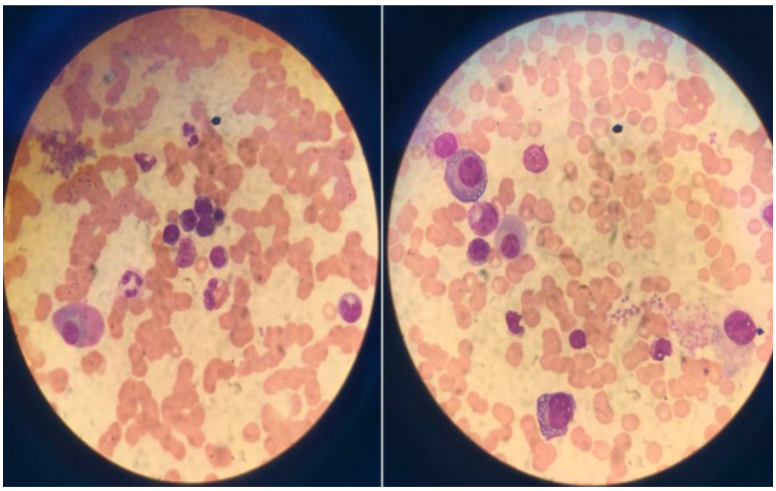

Figure 2: Bone marrow biopsy demonstrated 15% CD138-positive plasma cells expressing kappa light chains with Congo red-positive material in interstitial and vascular compartments—diagnostic of AL amyloidosis

Alveolar Sarcoidosis with Atypical Findings: A Case Report

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Introduction:
Although sarcoidosis is a systemic granulomatous disease, it most commonly affects the lungs and intrathoracic lymph nodes. It can present with a wide range of clinical and radiological manifestations. Here, we present a case of sarcoidosis with atypical radiological findings that required differential diagnosis from various clinical conditions.

Case Report:
A 57-year-old female patient presented to our clinic with progressively worsening dyspnea and cough. Thoracic computed tomography (CT) revealed bilateral dense ground-glass opacities, septal thickening, atelectatic bands, parenchymal distortion, and volume loss (Figure 1). The patient was evaluated for interstitial lung disease. Rheumatologic tests were negative, serum ACE level was 149 U/L, and there was no history of occupational or environmental exposure, medication use, or bird breeding. She had a 1.5 pack/year smoking history but had quit. Given the widespread ground-glass opacities on thoracic CT, alveolar proteinosis or hypersensitivity pneumonitis was initially suspected. Bronchoscopy was performed and bronchoalveolar lavage (BAL) fluid was collected. Histopathological examination of the BAL material did not support a diagnosis of alveolar proteinosis. The case was discussed in a surgical council, and for definitive pathological diagnosis, wedge resection of the right lower lobe was performed. Pathological evaluation of the resection specimen revealed “non-necrotizing granulomatous inflammation, consistent with sarcoidosis.” The patient was started on prednisolone and is under ongoing follow-up.

Discussion and Conclusion:
Alveolar sarcoidosis is an atypical form observed in approximately 20–25% of cases. This can lead to delays in diagnosis and treatment, potentially resulting in permanent organ damage. We presented this case to highlight that sarcoidosis can mimic many clinical conditions and to emphasize the importance of considering sarcoidosis, known as the “great mimicker,” especially in the presence of atypical parenchymal findings, in order to avoid diagnostic and therapeutic delays.

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