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CHEST IMAGING

PICTORIAL ESSAY

Radiologic patterns of interstitial lung disease: a pictorial review based on the 2025 European Respiratory Society/American Thoracic Society Classification

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ABSTRACT

Interstitial lung diseases (ILDs) are a heterogeneous group of diffuse parenchymal disorders with overlapping clinical and physiological features, making imaging central to diagnosis and management. Thin-slice computed tomography (CT) enables recognition of characteristic patterns and disease monitoring, serving as a cornerstone of multidisciplinary discussion. The 2025 European Respiratory Society/American Thoracic Society classification introduces refinements to the 2013 framework, grouping ILDs into three categories: interstitial patterns, alveolar filling disorders, and rare or unclassified disorders. This pictorial essay illustrates the updated classification with representative CT examples.

KEYWORDS

Interstitial lung diseases, computed tomography, bronchiolocentric interstitial pneumonia, alveolar macrophage pneumonia, fibrosis, usual interstitial pneumonia

Interstitial lung diseases (ILDs) are a heterogeneous group of parenchymal disorders characterized by varying degrees of inflammation and fibrosis.[1-3] Imaging, particularly thin-section computed tomography (CT), is central to diagnosis, management, and monitoring.[3-6] The 2025 European Respiratory Society/American Thoracic Society (ERS/ATS) consensus update on ILD classification refined the framework (Figure 1), reflecting advances in pathobiology, new terminology, and recognition of clinically relevant radiologic–pathologic patterns.[7] This article aims to provide radiologists with a visual reference for the updated ILD classification, illustrated with representative CT images.

Interstitial patterns

Interstitial patterns are key for ILD classification and are labeled as fibrotic or non-fibrotic. Fibrotic disease shows distortion, traction bronchiectasis, and honeycombing, indicating ongoing, often irreversible remodeling.[5-7] Non-fibrotic patterns reflect inflammation or acute injury without fibrosis. Accurate CT differentiation is vital for diagnosis, prognosis, and treatment.[7]

Usual interstitial pneumonia (UIP) is the most clinically significant form of fibrotic interstitial pneumonia and serves as the defining histopathological correlate of idiopathic pulmonary fibrosis.[1,4,7] The condition reflects chronic, progressive parenchymal remodeling with temporal and spatially heterogeneous fibrosis. Histology shows subpleural-predominant fibrosis, architectural distortion, fibroblastic foci, and honeycomb cysts.[8] CT enables non-invasive recognition, often avoiding biopsy. Hallmark findings include basal and subpleural reticulation, traction bronchiectasis, and honeycombing (Figure 2).[1,4,7] Honeycombing is the most specific sign, with ancillary features such as volume loss and distortion.

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UIP carries a poor prognosis with progressive functional decline despite therapy.[1,5-7]

Non-specific interstitial pneumonia (NSIP) is the most common pattern in connective tissue disease-associated ILD (CTD-ILD). However, it may also occur idiopathically or with drug reactions and other systemic disorders.[4-7] Histology shows uniform interstitial inflammation and fibrosis, without the temporal heterogeneity of UIP.[1-3] CT usually shows symmetric ground-glass opacities (GGOs) and fine reticulation, with lower lobe and subpleural predominance, and possible subpleural sparing, which helps distinguish NSIP from UIP (Supplementary Figure 1). Traction bronchiectasis, volume loss, and architectural distortion are frequent in the fibrotic subtype, whereas honeycombing is rare (Figure 3).[4-7,9] NSIP carries a better prognosis than UIP and often responds to immunosuppressive therapy.

Bronchiolocentric interstitial pneumonia (BIP) is a newly recognized interstitial pattern in the 2025 ERS/ATS classification, defined by fibrosis and inflammation centered around the small airways.[7] It is relevant because it may occur in diverse contexts, including hypersensitivity pneumonitis, CTD-ILD, aspiration, and drug-induced lung injury, often mimicking other fibrotic processes. On CT, BIP typically manifests as airway-centered fibrosis with associated tree-in-bud opacities and centrilobular nodules, sometimes accompanied by peribronchiolar reticulation (Figure 4).[7,10]

Diffuse alveolar damage (DAD), formerly termed acute interstitial pneumonia, is the histopathologic correlate of acute respiratory distress syndrome (ARDS) in idiopathic or secondary settings.[7,11] It is characterized by acute alveolar injury with hyaline membrane formation and interstitial edema, leading to rapid clinical deterioration.[11] On CT, DAD typically presents with diffuse GGOs and patchy consolidations, often with dependent predominance, reflecting the exudative phase of ARDS (Figure 5). In survivors, imaging may evolve toward reticulation and traction bronchiectasis, indicating fibrotic remodeling.[5-7,10,11]

Main points

- The 2025 European Respiratory Society/American Thoracic Society update organizes interstitial lung diseases (ILDs) into three computed tomography (CT)-based pattern groups.
- Recognizing key CT patterns is essential for diagnosing and managing ILD.
- This pictorial review provides practical CT examples for everyday interpretation.

Alveolar filling disorders

Alveolar filling disorders are characterized by the accumulation of specific cells or materials within the alveolar spaces, distinguishing them from fibrotic interstitial pneumonias, which primarily involve the lung interstitium.[7]

Organizing pneumonia (OP) is characterized by intra-alveolar fibroblastic plugs (Masson bodies) that preserve underlying lung architecture. The condition may be idiopathic (cryptogenic OP) or occur secondarily. Secondary causes include infection, CTD, drug reactions, radiation, or aspiration.[7,11] On CT, OP typically presents with patchy peripheral or peribronchovascular consolidations. These are often migratory in distribution. A characteristic but less common feature is the reversed halo sign, which is a central GGO surrounded by a ring of consolidation. Arcade-like opacities, representing curvilinear perilobular consolidations, may also be seen. These reflect the organization of tissue along the interstitium (Figure 6).[7,11,12] OP frequently responds to corticosteroid therapy and carries a more favorable prognosis than fibrotic ILDs.[11]

Respiratory bronchiolitis-interstitial lung disease (RB-ILD) is a smoking-related disorder characterized by the accumulation of pigmented macrophages within respiratory bronchioles and adjacent alveoli.[13] It occurs almost exclusively in current or former heavy smokers. It represents the diffuse, symptomatic form of the histologic lesion known as RB, which is otherwise common and often subclinical in smokers. On CT, RB-ILD typically demonstrates ill-defined centrilobular nodules and patchy GGOs, usually with an upper lobe predominance (Figure 7). Associated findings may include bronchial wall thickening and expiratory air trapping. The condition often improves with smoking cessation and generally carries a favorable prognosis.[7,13]

Alveolar macrophage pneumonia (AMP), formerly termed desquamative interstitial pneumonia, is an alveolar filling disorder marked by macrophage accumulation within alveolar spaces.[7,13] Although strongly linked to cigarette smoking, AMP can also, albeit rarely, occur in association with CTDs such as rheumatoid arthritis or systemic sclerosis, certain drug exposures (e.g., nitrofurantoin, amiodarone), or environmental factors (such as occupational dust). On CT, AMP shows diffuse, basally predominant GGOs with mild reticulation (Figure 8). Notably, honeycombing is rare, although small cystic changes may appear in advanced disease.[13]

Eosinophilic pneumonias comprise acute and chronic forms, both characterized by eosinophilic infiltration of alveoli and the interstitium, often associated with asthma, drug reactions, infection, or autoimmune disease.[7,13] Acute eosinophilic pneumonia typically presents with rapidly progressive respiratory failure and diffuse pulmonary involvement. CT findings include bilateral GGOs and patchy consolidations.[11,13] Chronic eosinophilic pneumonia follows a more indolent course and classically shows peripheral, upper-lobe-predominant consolidations and GGOs, sometimes described as the "photographic negative" of pulmonary edema (Figure 9). Pleural effusion is uncommon, and EP typically responds well to corticosteroid therapy.

Pulmonary alveolar proteinosis (PAP) is characterized by the intra-alveolar accumulation of surfactant material, which may be autoimmune, secondary to hematologic or environmental exposures, or congenital.[14] On CT, PAP exhibits the classic "crazy-paving" pattern, defined by diffuse or patchy GGOs superimposed with smooth interlobular septal thickening and intralobular lines. Distribution is often bilateral and symmetric, with perihilar or lower-lung predominance (Figure 10).[7,14] Imaging findings help to avoid invasive testing and guide management, including whole-lung lavage or targeted therapy in autoimmune cases.

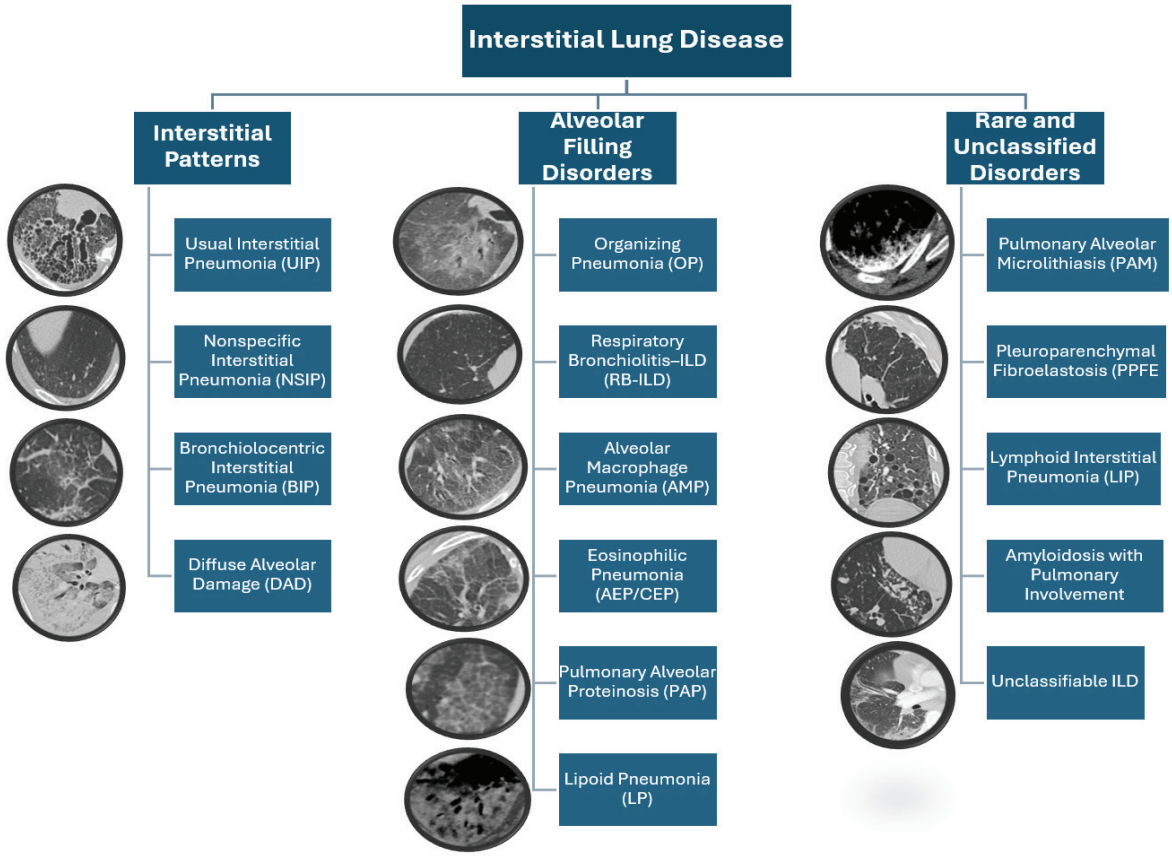

Figure 1. Radiological patterns of interstitial, alveolar filling, and uncommon or unclassified pulmonary disorders. Representative computed tomography images illustrate the predominant parenchymal patterns. ILD, interstitial lung disease.

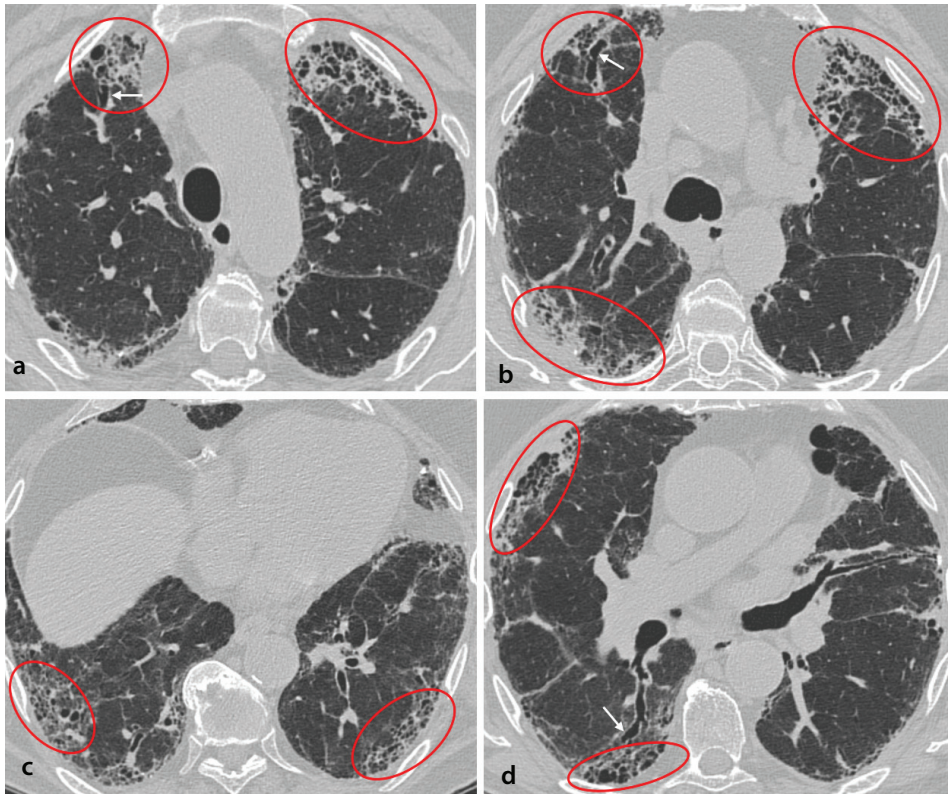

Figure 2. Usual interstitial pneumonia pattern on chest computed tomography (CT). Axial CT images **(a–d)** demonstrate characteristic UIP features, including basal and subpleural reticulation, honeycombing (red circles), and traction bronchiectasis (arrows). Fibrotic changes are heterogeneously distributed with basal and subpleural predominance and relative sparing of the central regions in a patient with idiopathic pulmonary fibrosis. UIP, usual interstitial pneumonia.

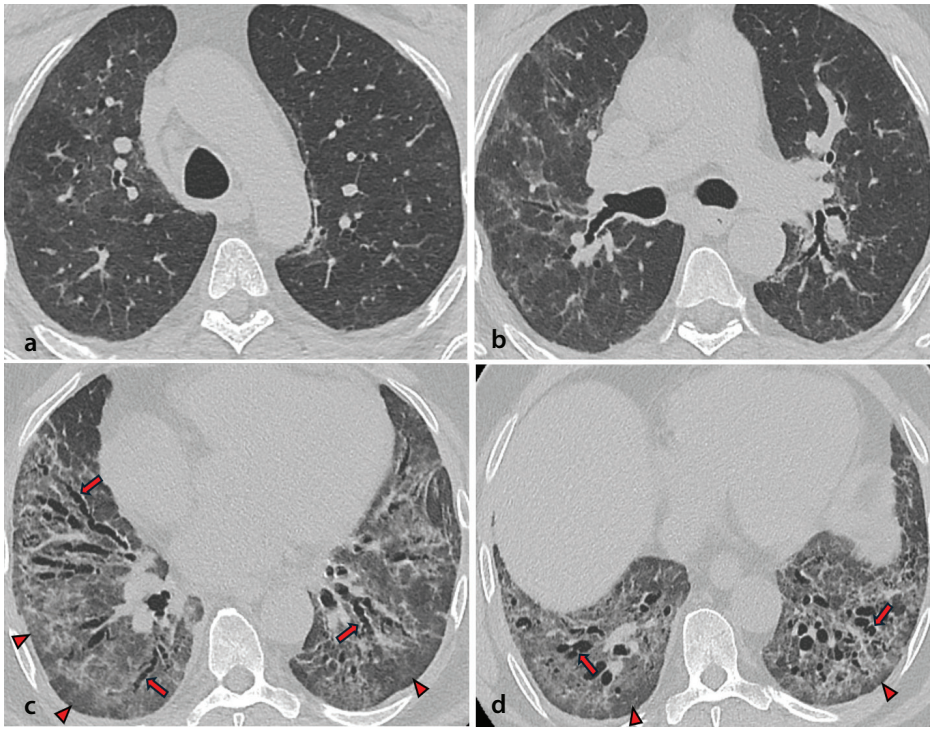

Figure 3. Non-specific interstitial pneumonia pattern on chest computed tomography (CT). Axial CT images **(a–d)** demonstrate bilateral ground-glass opacities and fine reticulation with lower-lobe predominance, accompanied by traction bronchiectasis (arrows) and areas of subpleural sparing (arrowheads). Findings are shown in a patient with systemic sclerosis–associated interstitial lung disease.

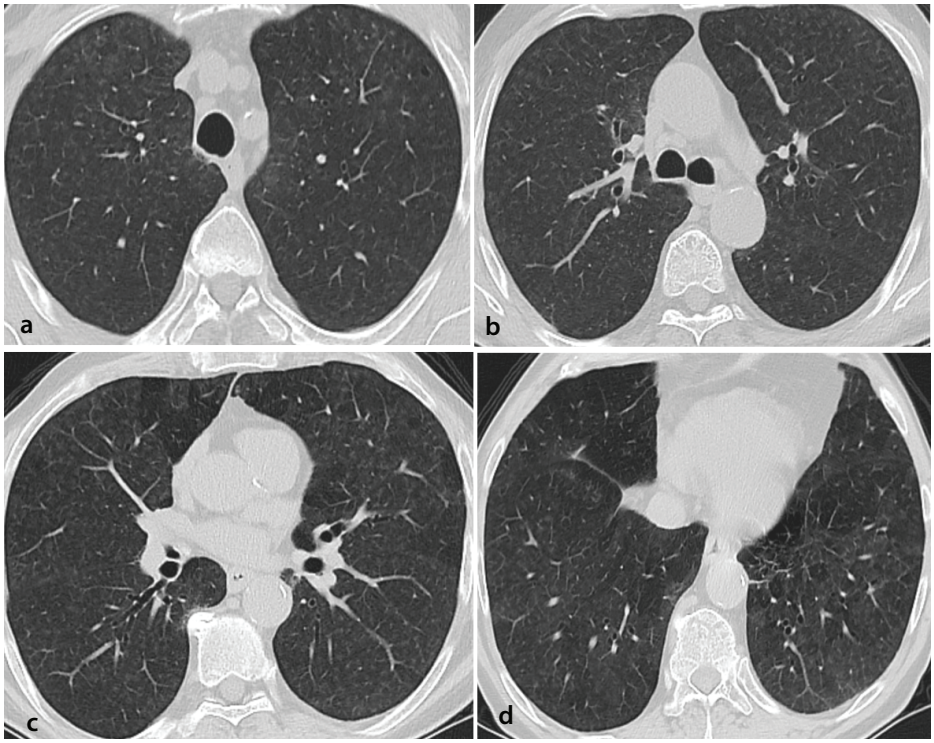

Figure 4. Bronchiolocentric interstitial pneumonia pattern on chest computed tomography (CT). Axial CT images **(a–d)** demonstrate diffusely distributed centrilobular nodules and ground-glass opacities, consistent with an airway-centered interstitial process. Basilar-predominant mosaic attenuation with associated lucent areas suggests air trapping. Based on integration of the imaging findings, histopathologic correlation, and exposure history, this pattern was interpreted as bronchiolocentric interstitial pneumonia, consistent with non-fibrotic hypersensitivity pneumonitis in the appropriate clinical context.

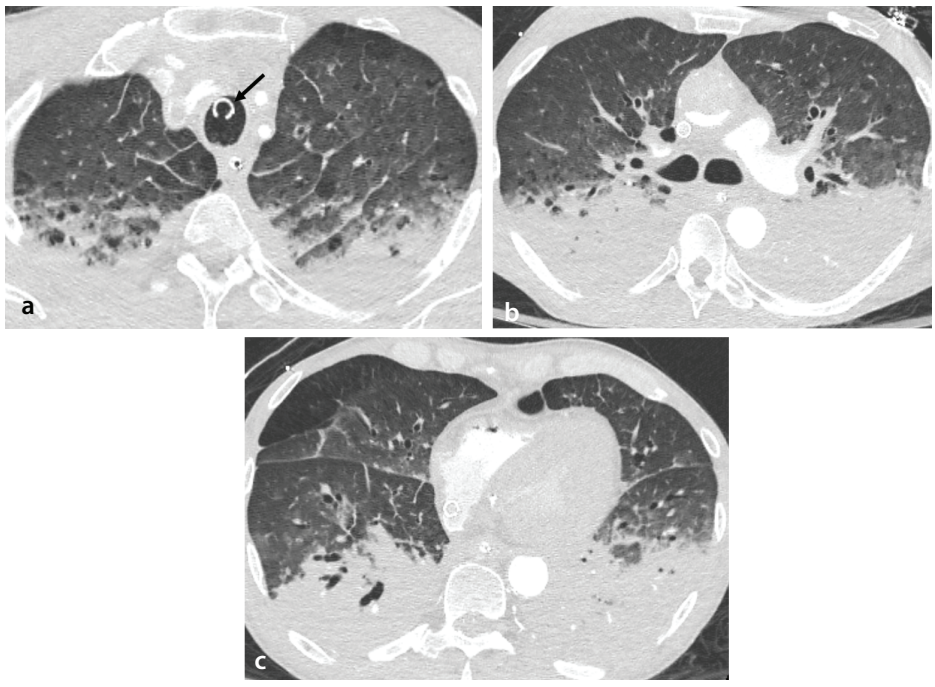

Figure 5. Diffuse alveolar damage pattern on chest computed tomography (CT). Axial CT images **(a–c)** demonstrate dependent gradient of consolidations, consistent with the exudative phase of diffuse alveolar damage. The patient experienced respiratory deterioration and met clinical criteria for acute respiratory distress syndrome. The endotracheal tube is noted (arrow), along with interstitial thickening, suggestive of underlying pulmonary edema.

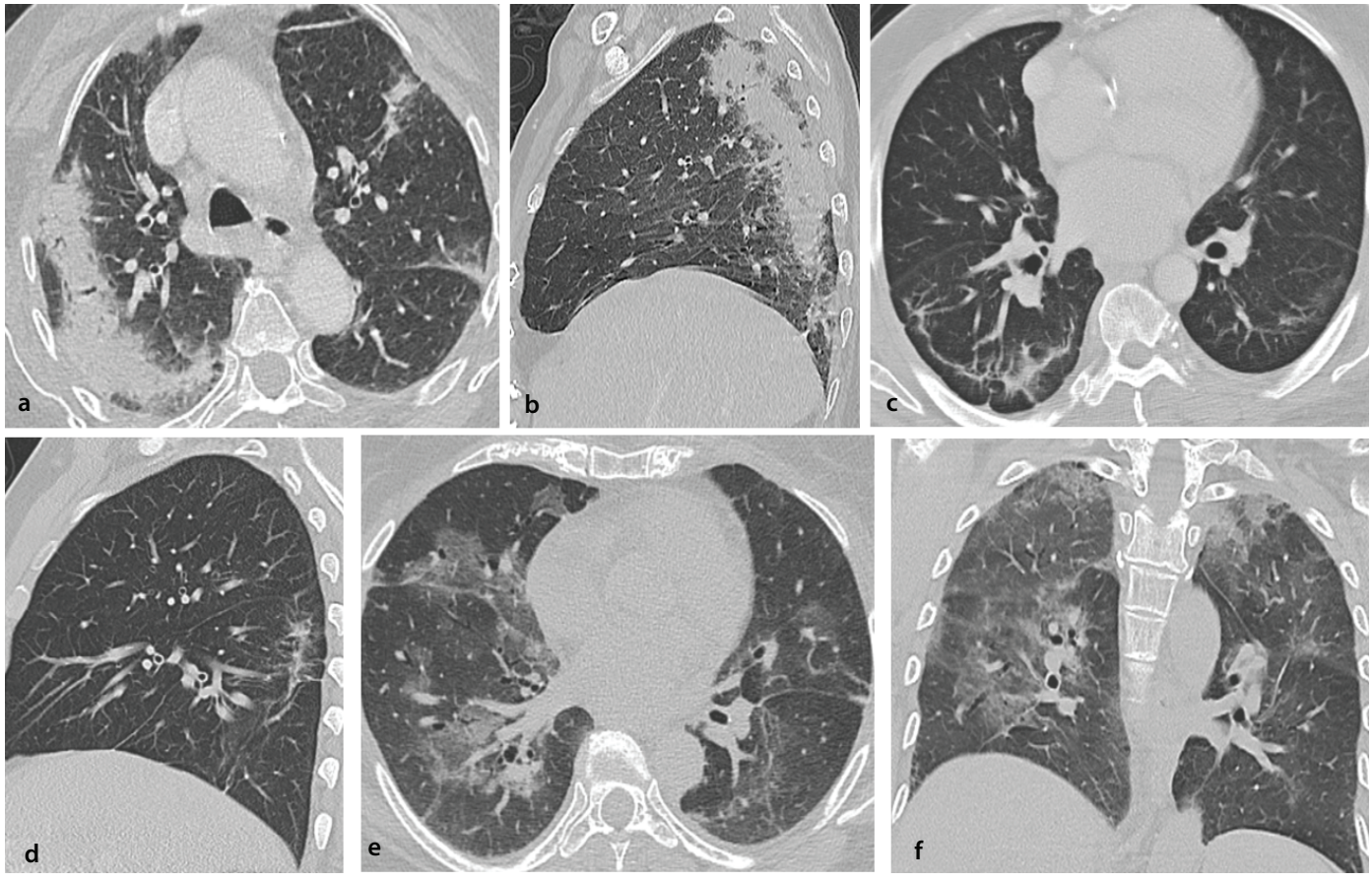

Figure 6. Organizing pneumonia patterns on chest computed tomography (CT). Axial **(a)** and sagittal **(b)** CT images demonstrate patchy peripheral consolidations, more prominent on the right side and involving both upper and lower lobes. Axial **(c)** and sagittal **(d)** images show peripheral, arcade-like opacities with areas of subpleural sparing. Axial **(e)** and coronal **(f)** images reveal patchy peripheral and peribronchial ground-glass opacities.

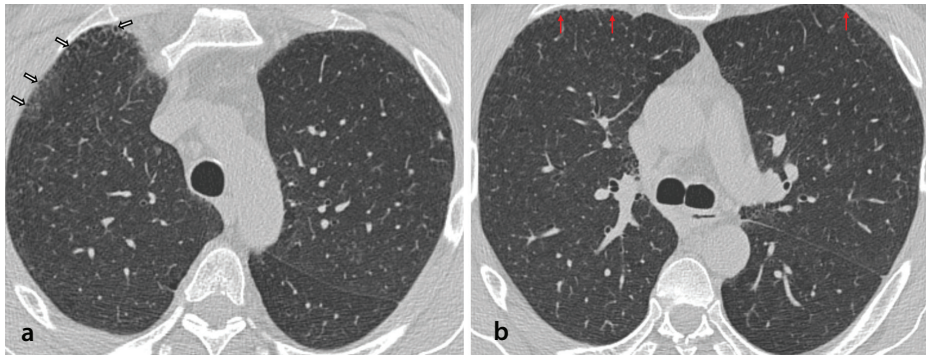

Figure 7. Respiratory bronchiolitis-interstitial lung disease (RB-ILD) pattern on chest computed tomography (CT). Axial CT images **(a, b)** demonstrate ill-defined centrilobular nodules and patchy, mild ground-glass opacities with upper-lobe predominance, representing the key imaging features of RB-ILD. Associated paraseptal emphysema is present (red arrows), consistent with a smoking-related ILD. Minimal subpleural reticulation and bronchiolectasis (white arrows) are incidental but are not defining features of RB-ILD.

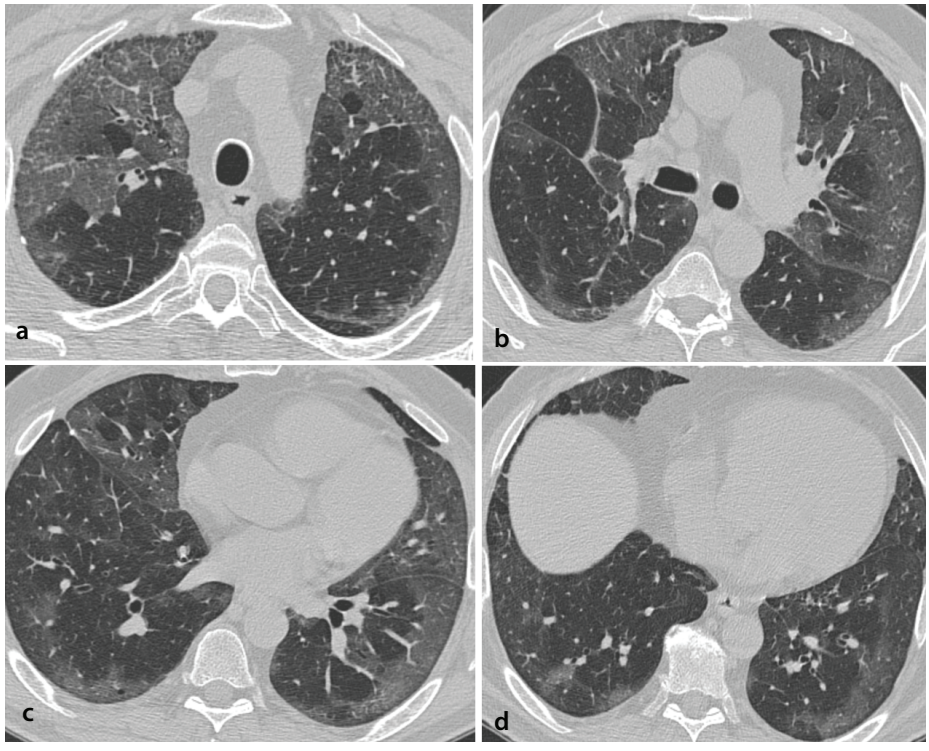

Figure 8. Alveolar macrophage pneumonia pattern, formerly termed desquamative interstitial pneumonia, on chest computed tomography (CT). Axial CT images **(a–d)** demonstrate diffuse, basilar-predominant ground-glass opacities in a patient with a 16 pack-year smoking history. The diagnosis was histopathologically confirmed.

Lipoid pneumonia (LP) is a rare disorder caused by the accumulation of lipid material within the alveoli, which may be exogenous (e.g., aspiration of mineral oils or fatty substances) or endogenous (associated with airway obstruction, lipid storage disorders, or tissue breakdown).[7] On CT, LP typically manifests as consolidations or nodules with fat attenuation, often demonstrating negative Hounsfield unit values. These lesions may be focal or multifocal and are sometimes accompanied by GGOs or a crazy-paving pattern when inflammation coexists (Figure 11).[15] Identification of fat density within pulmonary opacities is highly suggestive of LP and helps distinguish it from infectious or neoplastic processes.

Rare and unclassified disorders

Rare and unclassified ILDs represent a heterogeneous group outside the major interstitial and alveolar filling patterns.[7] Some cases show overlapping or atypical findings and are labeled unclassifiable ILD.

Pulmonary alveolar microlithiasis (PAM) is a rare genetic disorder caused by mutations in the *SLC34A2* gene, leading to intra-alveolar accumulation of calcium phosphate microliths and potentially resulting in restrictive lung physiology. The disease often progresses insidiously, with imaging findings preceding symptoms by years.[7,16] On CT, PAM is characterized by diffuse, sand-like calcific micronodules that produce a dense "ground-glass" appearance and marked subpleural calcification (Figure 12). The black pleura sign, indicating relative subpleural lucency due to adjacent dense calcification, can be seen.[16]

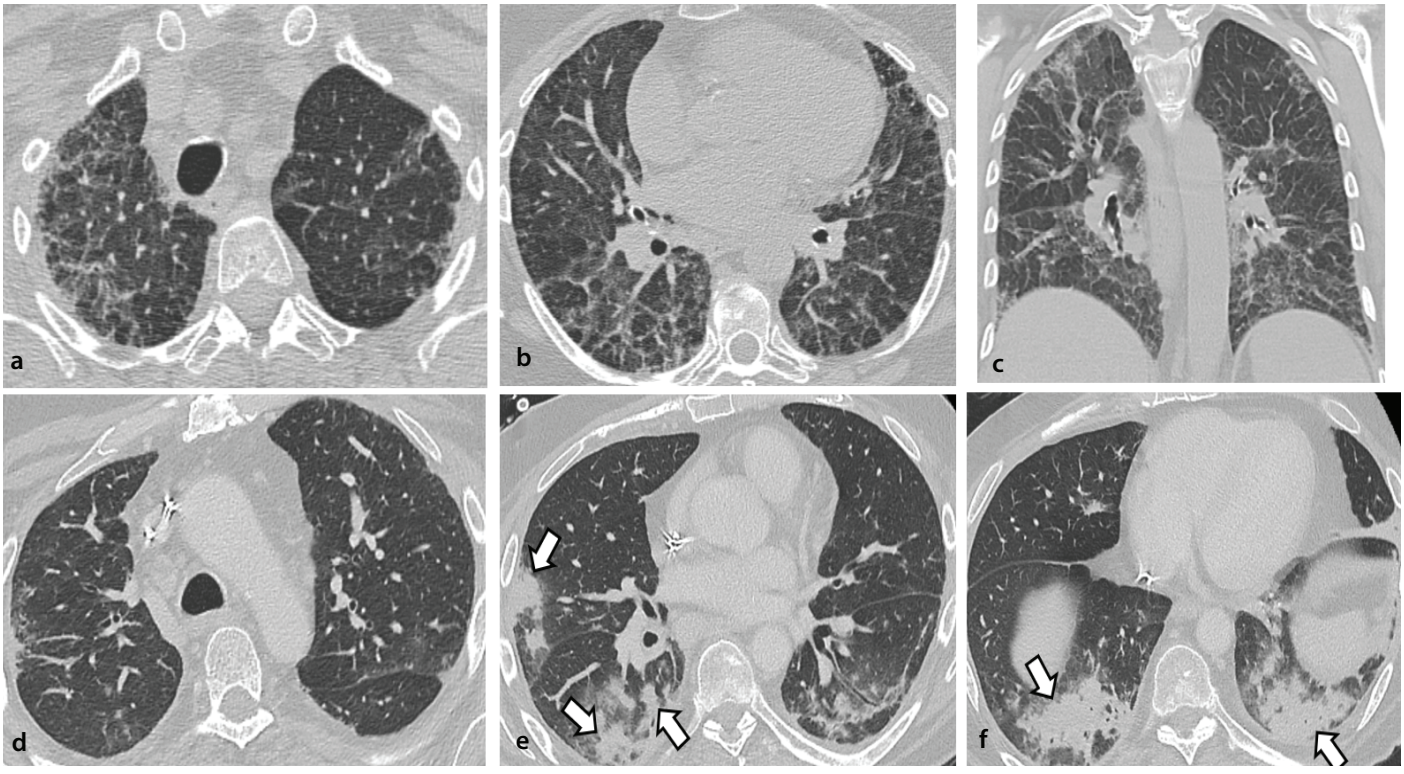

Figure 9. Eosinophilic pneumonia patterns on chest computed tomography (CT). Axial **(a, b)** and coronal **(c)** CT images demonstrate chronic eosinophilic pneumonia, characterized by peripheral, upper-lobe–predominant ground-glass opacities (GGOs), architectural distortion, and reticulations in a patient with a subacute-to-chronic clinical course. Axial CT images **(d–f)** demonstrate acute eosinophilic pneumonia with bilateral patchy consolidations (arrows) and ground-glass opacities in the setting of acute respiratory symptoms.

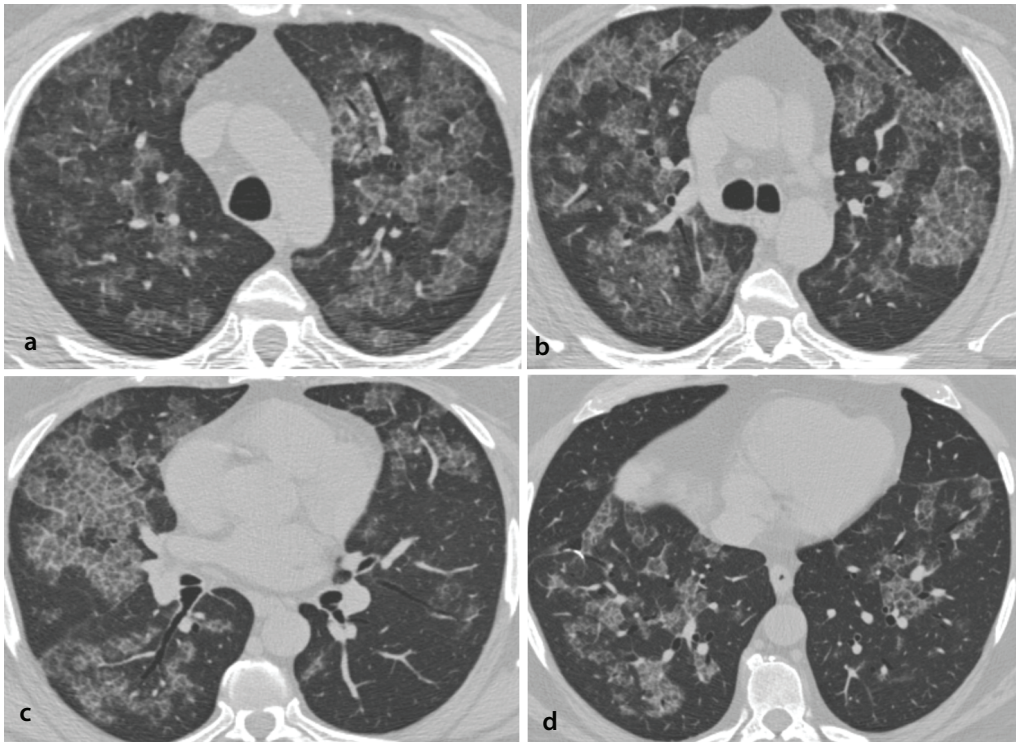

Figure 10. Pulmonary alveolar proteinosis pattern on chest computed tomography (CT). Axial CT images **(a–d)** demonstrate the characteristic "crazy-paving" pattern, consisting of diffuse or patchy ground-glass opacities superimposed with smooth interlobular septal thickening and intralobular lines. The distribution is bilateral and symmetric.

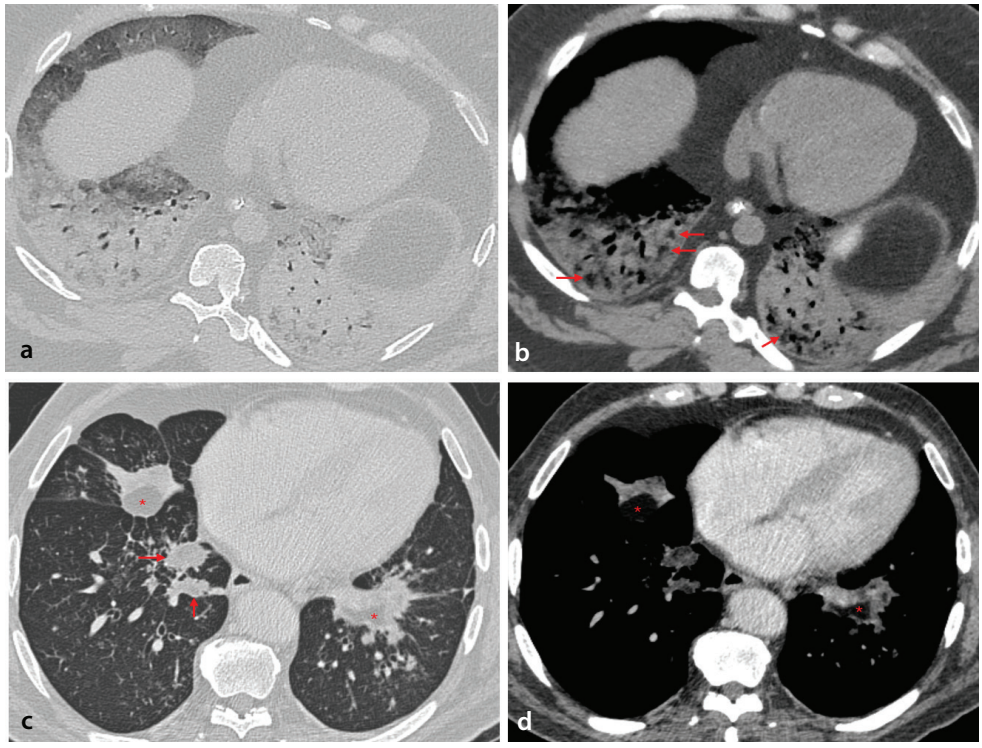

Figure 11. Lipoid pneumonia patterns on chest computed tomography (CT). Axial CT images **(a, b)** show bilateral lower-lobe consolidations with areas of negative attenuation (arrows) in a patient with acute respiratory failure, consistent with endogenous lipoid pneumonia. Axial CT images **(c, d)** demonstrate mixed-attenuation masses and nodules containing fat-density areas (asterisks) in a patient with chronic constipation and a history of oral oil-based laxative use, consistent with exogenous lipoid pneumonia due to aspiration.

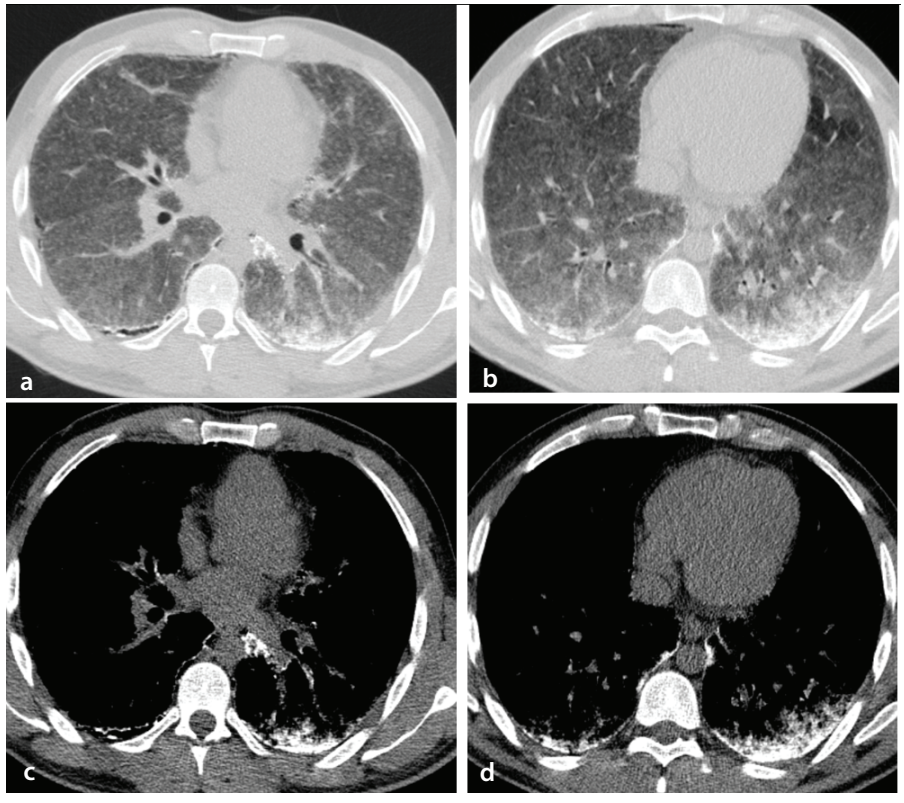

Figure 12. Pulmonary alveolar microlithiasis pattern on chest computed tomography (CT). Axial CT images **(a, b)** in lung windows show diffuse, sand-like calcific micronodules creating a dense "ground-glass" appearance with subpleural predominance. Corresponding mediastinal window images **(c, d)** highlight extensive subpleural and parenchymal calcifications.

Pleuroparenchymal fibroelastosis is characterized by dense fibroelastotic thickening of the pleura and adjacent subpleural lung parenchyma, predominantly in the upper lobes. It may arise idiopathically or secondary to transplantation, chemotherapy, autoimmune disease, or recurrent infections.[5-7] CT typically shows upper-lobe pleural and subpleural fibrosis with significant volume loss, resulting in the characteristic platythorax (flattened chest) appearance. Additional findings can include traction bronchiectasis, interlobular septal thickening, and honeycombing (Figure 13).[5,6] Pleuroparenchymal fibroelastosis follows a progressive course with poor prognosis, frequently leading to respiratory failure.

Lymphoid interstitial pneumonia (LIP) is characterized by diffuse lymphoid infiltration of the interstitium, most often associated with autoimmune diseases, immunodeficiency, or viral infections, although idiopathic cases may occur.[7] On CT, LIP typically manifests as a combination of thin-walled cysts, GGOs, and interlobular septal thickening, often with lower-lobe predominance (Figure 14). Small nodules and peribronchovascular thickening may also be present.[17,18] Although some patients respond to immunosuppressive therapy, LIP carries a risk of progression to lymphoma.

Pulmonary amyloidosis results from extracellular deposition of amyloid fibrils within the lung parenchyma, airways, or vasculature and may manifest in either systemic or localized forms.[7,19] On CT, amyloidosis can appear as calcified or non-calcified parenchymal nodules, tracheobronchial wall thickening, or interstitial infiltration. In cases of interstitial infiltration, imaging findings include patchy GGOs, smooth or nodular interlobular septal thickening, perilymphatic micronodules, and occasional architectural distortion (Figure 15).[19,20]

Unclassifiable ILD refers to cases in which a confident diagnosis cannot be established despite comprehensive clinical, radiologic, and pathologic evaluation.[7] This may result from overlapping features, atypical or indeterminate patterns, or limited biopsy samples. On CT, unclassifiable ILD often shows a mixture of findings, such as fibrotic changes coexisting with OP-like opacities, making assignment to a single category challenging (Supplementary Figure 2).[5-7,11]

The 2025 ERS/ATS classification offers an updated framework for ILD, emphasizing CT's central role in distinguishing key radiologic patterns with clinical and prognostic relevance (Table 1).

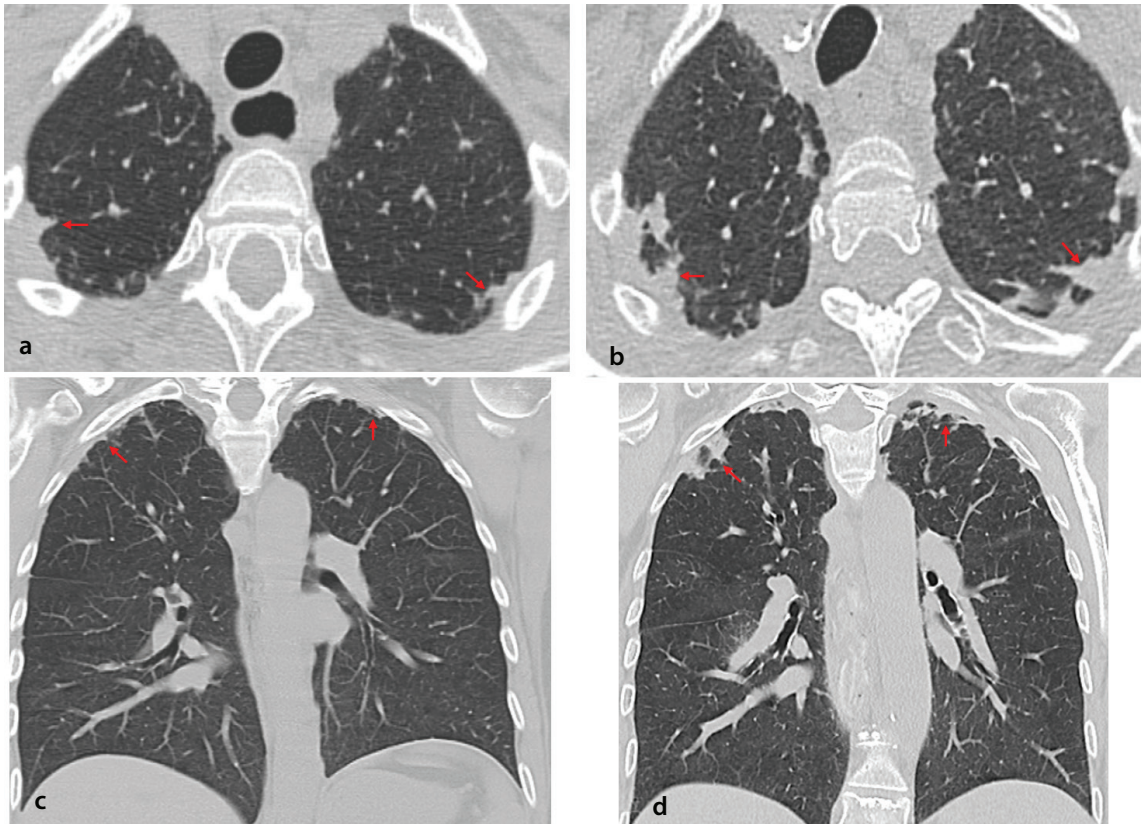

Figure 13. Pleuroparenchymal fibroelastosis pattern on high-resolution chest computed tomography (CT). Initial axial **(a)** and coronal **(c)** CT images demonstrate dense pleural and subpleural fibrosis with upper-lobe predominance. Follow-up CT images obtained one year later **(b, d)** show marked progression of fibrosis with associated volume loss and architectural distortion (arrows).

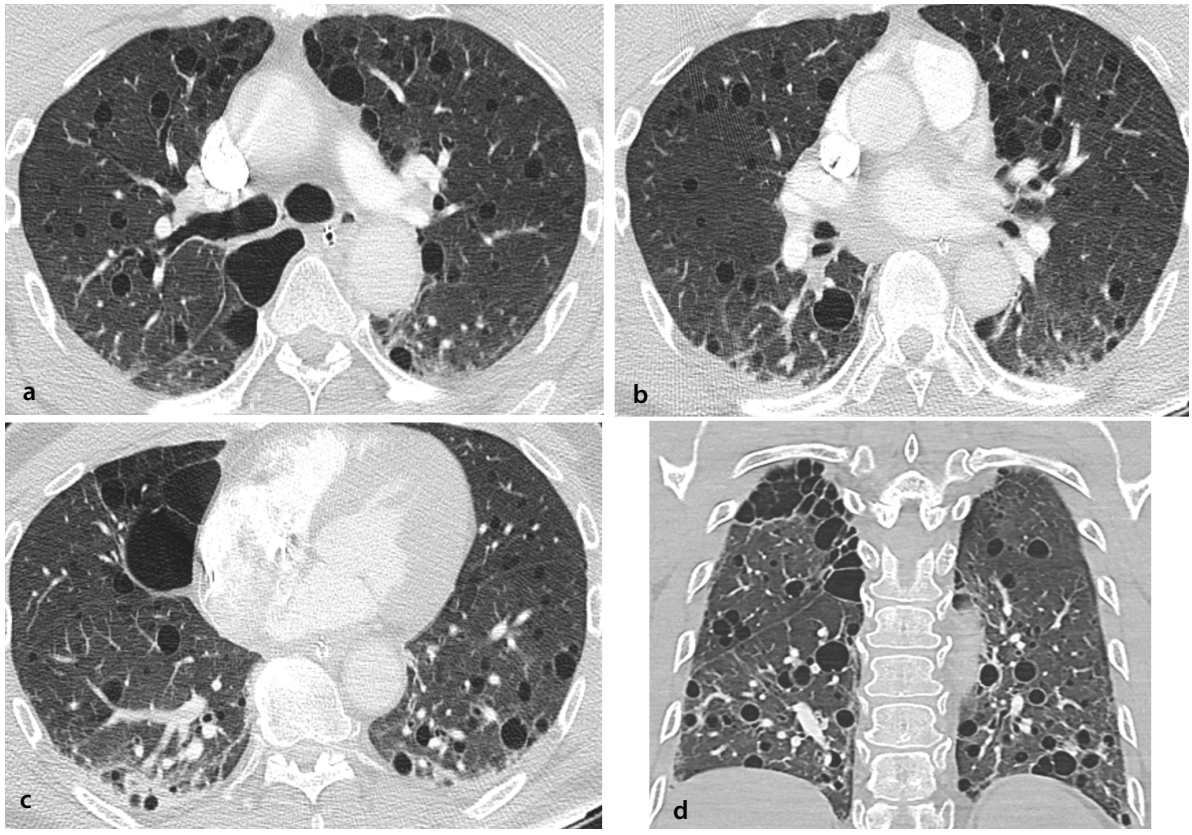

Figure 14. Lymphoid interstitial pneumonia (LIP) pattern on chest computed tomography (CT) in a patient with underlying Sjögren's disease. Axial **(a–c)** and coronal **(d)** CT images show numerous thin-walled cysts of variable size, lower-lobe–predominant ground-glass opacities, and interlobular septal thickening, characteristic of LIP.

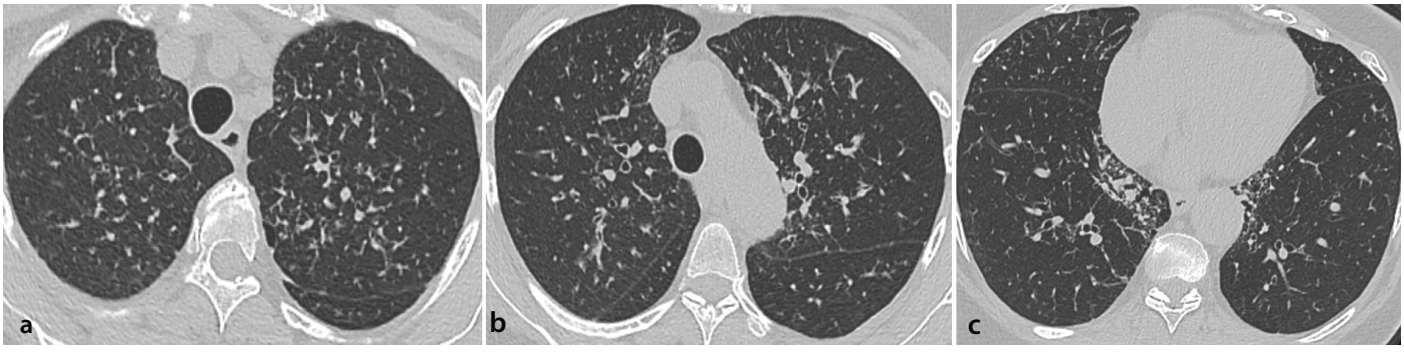

Figure 15. Pulmonary amyloidosis pattern on chest computed tomography (CT). Axial CT images **(a–c)** demonstrate nodular interlobular septal thickening, scattered perilymphatic micronodules, and patchy ground-glass opacities. The imaging findings were suggestive of pulmonary amyloidosis in a patient with known systemic disease, and were supported by histopathologic correlation.

Table 1. Radiological subtypes of interstitial, alveolar filling, and rare/unclassified disorders

Subtype/pattern	Definition/key features	Imaging characteristics [Radiograph/chest computed tomography (CT)]
Interstitial patterns		
Usual interstitial pneumonia	Chronic fibrosing interstitial lung disease (ILD); histopathologic and radiologic hallmark of idiopathic pulmonary fibrosis.	Radiograph: reticular opacities, basal volume loss. CT: basal and subpleural fibrosis, honeycombing, traction bronchiectasis.
Non-specific interstitial pneumonia	Temporally homogeneous interstitial pneumonia with varying degrees of inflammation and fibrosis; includes non-fibrotic and fibrotic subtypes; commonly associated with connective tissue disease (CTD) but may be idiopathic or secondary.	Radiograph: bilateral hazy opacities. CT: diffuse ground-glass opacities (GGOs) with or without fine reticulation, traction bronchiectasis in fibrotic subtype, lower-lobe predominance, possible subpleural sparing.
Bronchiolocentric interstitial pneumonia	Airway-centered interstitial pneumonia characterized by bronchiolocentric inflammation with or without fibrosis; may be seen in hypersensitivity pneumonitis, CTD-associated ILD, aspiration, or drug-related lung disease.	CT: peribronchiolar interstitial thickening or reticulation, centrilobular nodules, tree-in-bud opacities; may be associated with air trapping on expiratory imaging.
Diffuse alveolar damage	Radiologic–pathologic pattern of acute lung injury, seen in acute respiratory distress syndrome (ARDS) and acute exacerbations of ILDs; ARDS represents a clinical syndrome.	Radiograph: diffuse bilateral opacities. CT: diffuse GGOs and patchy consolidation, often with dependent predominance.
Alveolar filling disorders		
Organizing pneumonia (OP)	Intra-alveolar fibroblastic tissue plugs with preservation of lung architecture; idiopathic (cryptogenic OP) or secondary to infection, CTD, drugs, radiation, or aspiration.	Radiograph: patchy, often peripheral opacities. CT: peripheral or peribronchovascular consolidations, perilobular or arcade-like opacities, reversed halo sign.
Respiratory bronchiolitis-ILD	Smoking-related ILD characterized by accumulation of pigmented macrophages within respiratory bronchioles and adjacent alveoli.	CT: ill-defined centrilobular ground-glass nodules and patchy GGOs, often with upper-lobe predominance.
Alveolar macrophage pneumonia (desquamative interstitial pneumonia)	Smoking-related alveolar filling disorder with diffuse intra-alveolar macrophage accumulation; less commonly associated with CTD or drug exposure.	Radiograph: diffuse hazy opacities. CT: diffuse GGOs with basal predominance, mild reticulation, and occasional small cysts.
Eosinophilic pneumonia [acute eosinophilic pneumonia (AEP)/ chronic eosinophilic pneumonia (CEP)]	Acute and chronic eosinophilic lung diseases characterized by eosinophilic infiltration of alveoli and interstitium; AEP presents with acute respiratory symptoms, whereas CEP follows a subacute to chronic course.	CT: AEP-diffuse bilateral GGOs and/or consolidation. CEP–peripheral and often upper-lobe–predominant consolidations and GGOs ("photographic negative" of pulmonary edema).
Pulmonary alveolar proteinosis	Intra-alveolar accumulation of surfactant material; most commonly autoimmune in origin.	Radiograph: bilateral perihilar opacities. CT: crazy-paving pattern (GGOs with superimposed septal thickening).
Lipoid pneumonia	Exogenous or endogenous accumulation of lipid material within the alveoli.	CT: consolidations or nodules with fat attenuation (negative Hounsfield units), sometimes with associated GGOs.
Rare and unclassified disorders		
Pulmonary alveolar microlithiasis	Rare genetic disorder with widespread intra-alveolar calcium phosphate microliths.	Radiograph: diffuse sand-like micronodules. CT: diffuse calcified micronodules with subpleural involvement; black pleura sign.
Pleuroparenchymal fibroelastosis	Rare fibrosing ILD characterized by upper-lobe–predominant pleural and subpleural fibroelastosis.	Radiograph: apical pleural thickening. CT: dense subpleural upper-lobe fibrosis, volume loss, platythorax.
Lymphoid interstitial pneumonia	Lymphoproliferative ILD; frequently associated with autoimmune disease or immunodeficiency.	CT: GGOs, interlobular septal thickening, scattered thin-walled cysts.
Pulmonary amyloidosis	Extracellular deposition of amyloid protein within lung parenchyma, airways, or vasculature.	CT: pulmonary nodules with or without calcification, tracheobronchial wall thickening, and interstitial infiltration.
Unclassifiable ILD	ILD with indeterminate or overlapping features that do not fit established categories despite multidisciplinary evaluation.	CT: mixed or overlapping patterns, often combining fibrotic changes with OP-like features.

Footnotes

Conflict of interest disclosure

Furkan Ufuk, MD, serves as Section Editor for Diagnostic and Interventional Radiology. He had no involvement in the peer review of this article and had no access to information regarding its peer review.

Supplementary Figures 1-2: https://d2v96fxpocvxx.cloudfront.net/beb8919b-f013-4ea1-b1c8-40332e840fe1/content-images/6d601dda-86a1-4f50-8964-b276c887a632.pdf

References

1. Raghu G, Remy-Jardin M, Richeldi L, et al. Idiopathic pulmonary fibrosis (an update) and progressive pulmonary fibrosis in adults: an official ATS/ERS/JRS/ALAT clinical practice guideline. *Am J Respir Crit Care Med*. 2022;205(9):e18-e47. [Crossref]
2. Rajan SK, Cottin V, Dhar R, et al. Progressive pulmonary fibrosis: an expert group consensus statement. *Eur Respir J*. 2023;61(3):2103187. [Crossref]
3. Marinescu DC, Hague CJ, Muller NL, et al. Integration and application of radiologic patterns from clinical practice guidelines on idiopathic pulmonary fibrosis and fibrotic hypersensitivity pneumonitis. *Chest*. 2023;164(6):1466-1475. [Crossref]
4. Chung JH, Cox CW, Montner SM, et al. CT features of the usual interstitial pneumonia pattern: differentiating connective tissue disease-associated interstitial lung disease from idiopathic pulmonary fibrosis. *AJR Am J Roentgenol*. 2018;210(2):307-313. [Crossref]
5. Lee KS, Han J, Wada N, et al. Imaging of pulmonary fibrosis: an update, from the AJR special series on imaging of fibrosis. *AJR Am J Roentgenol*. 2024;222(2):e2329119. [Crossref]
6. Chelala L, Brixey AG, Hobbs SB, et al. Current state of fibrotic interstitial lung disease imaging. *Radiology*. 2025;316(1):e242531. [Crossref]
7. Ryerson CJ, Adegunsoye A, Piciucchi S, et al. Update of the international multidisciplinary classification of the interstitial pneumonias: an ERS/ATS statement. *Eur Respir J*. 2025;66(6):2500158. [Crossref]
8. Mukhopadhyay S. Usual interstitial pneumonia (UIP): a clinically significant pathologic diagnosis. *Mod Pathol*. 2022;35(5):580-588. [Crossref]
9. Shih AR, Nitiwarangkul C, Little BP, et al. Practical application and validation of the 2018 ATS/ERS/JRS/ALAT and Fleischner Society guidelines for the diagnosis of idiopathic pulmonary fibrosis. *Respir Res*. 2021;22(1):124. [Crossref]
10. Fernández Pérez ER, Travis WD, Lynch DA. Rethinking the CT and histopathological nomenclature of hypersensitivity pneumonitis: unveiling bronchiolocentric patterns of interstitial pneumonia. *Am J Respir Crit Care Med*. 2025;211(10):1738-1740. [Crossref]
11. Marquis KM, Hammer MM, Steinbrecher K, et al. CT approach to lung injury. *Radiographics*. 2023;43(7):e220176. [Crossref]
12. Ufuk F, Ocak I, Chelala L, Landeras L. The reverse halo sign in emergency chest CT: a diagnostic guide across diverse pulmonary conditions. *Emerg Radiol*. 2025;32(3):435-445. [Crossref]
13. Escalon JG, Girvin F. Smoking-related interstitial lung disease and emphysema. *Clin Chest Med*. 2024;45(2):461-473. [Crossref]
14. Trapnell BC, Nakata K, Bonella F, et al. Pulmonary alveolar proteinosis. *Nat Rev Dis Primers*. 2019;5(1):16. [Crossref]
15. Bučić D, Koršić M, Smojver-Ježek S, Hrabak-Paar M. Exogenous lipoid pneumonia after long-term nasal petroleum jelly application. *Radiol Cardiothorac Imaging*. 2025;7(1):e240289. [Crossref]
16. Ufuk F. Pulmonary alveolar microlithiasis. *Radiology*. 2021;298(3):567. [Crossref]
17. Kuykendall K, Raptis C, Bhalla S. Interstitial lung disease in connective tissue disease. *Radiol Clin North Am*. 2025;63(4):595-607. [Crossref]
18. Ufuk F. Lymphoid interstitial pneumonia. *N Engl J Med*. 2025;392(8):806. [Crossref]
19. Li F, Li J, Li Y, et al. Imaging and clinical features of nodular pulmonary amyloidosis. *J Thorac Imaging*. 2025;40(5):e0830. [Crossref]
20. Eggleston RH, Hartman TE, Walkoff LA, Yi ES, Ryu JH, Baqir M. Clinical, radiologic, and pathologic features and outcomes of pulmonary transthyretin amyloidosis. *Respir Med*. 2022;194:106761. [Crossref]