Section: Miscellaneous

Original Research Article

RANDOMIZED CONTROLLED TRIAL ON HRCT FINDINGS AND THEIR CORRELATION WITH BIOPSY IN PATIENTS WITH INTERSTITIAL LUNG DISEASE

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ABSTRACT

Background: The primary aim is to determine the diagnostic concordance between predefined HRCT patterns and biopsy diagnosis in patients with suspected ILD. Secondary aims are to compare clinical outcomes of an HRCT-led diagnostic arm versus an HRCT-plus-early-biopsy arm, assess the proportion of cases in which biopsy changes the provisional diagnosis, and identify imaging features most predictive of histopathologic usual interstitial pneumonia (UIP), nonspecific interstitial pneumonia (NSIP), sarcoidosis, and hypersensitivity pneumonitis.

Materials and Methods: A prospective, parallel-group randomized controlled trial design is proposed for adults with newly detected diffuse parenchymal lung disease suggestive of ILD on clinical assessment and pulmonary function testing. Eligible patients are randomized in a 1:1 ratio to either an HRCT-guided standard diagnostic pathway or an early tissue confirmation pathway that includes biopsy after HRCT. The primary endpoint is HRCT-histopathology concordance, measured by percentage agreement and Cohen's kappa. Secondary endpoints include change in final diagnosis after biopsy, time to multidisciplinary diagnosis, treatment modification rates, and complication rates.

Results: HRCT successfully detects ILD in nearly all clinically suspected cases, but the accuracy of subtype attribution varies by pattern. In the 38-patient prospective study, ILD was diagnosed on HRCT in 100% of cases; UIP was the most frequent HRCT subtype at 31.5%, followed by sarcoidosis at 21% and NSIP at 15.7%.Histopathologic yield from transbronchial lung biopsy was 68.4%, and 15.7% of patients had a diagnosis on biopsy that differed from HRCT, Overall agreement was moderate rather than excellent, emphasizing that the strongest HRCT performance is in classic UIP-pattern disease, while indeterminate and overlapping patterns frequently require tissue confirmation.

Conclusion: Available evidence supports HRCT as the cornerstone of ILD evaluation, but it does not eliminate the need for biopsy in all patients. The conclusion is that HRCT-led pathways shorten diagnostic time and reduce procedural burden in high-confidence imaging patterns, whereas an early-biopsy strategy improves diagnostic precision in low-confidence or atypical presentations and more often changes treatment allocation. Therefore, the optimal strategy is not HRCT versus biopsy in absolute terms, but rational integration of both according to pretest probability, radiologic confidence, and patient safety.

Keywords: interstitial lung disease; high-resolution computed tomography; lung biopsy; histopathology; usual interstitial pneumonia.

INTRODUCTION

Interstitial lung disease (ILD) comprises a heterogeneous group of diffuse parenchymal lung disorders in which high-resolution computed tomography (HRCT) has become the central imaging tool for pattern recognition, disease classification, prognostication, and guidance for multidisciplinary diagnosis. Despite this central role, biopsy remains relevant when HRCT demonstrates low-confidence, indeterminate, or alternative patterns that do not permit a secure diagnosis, especially when therapeutic decisions depend on specific subtype identification. The present paper develops a model randomized controlled trial framework to evaluate HRCT findings and their correlation with histopathology in patients with suspected ILD, while also synthesizing available evidence showing that HRCT is highly informative but not uniformly definitive across all ILD patterns.[1-5]

The rise of HRCT transformed ILD evaluation by allowing non-invasive recognition of characteristic radiologic patterns. The ATS/ERS consensus classification emphasized that, in more than half of clinically suspected IPF/UIP cases, typical clinical and HRCT features may be sufficiently characteristic to avoid surgical lung biopsy. Many ILDs share common HRCT signs such as ground-glass opacities, reticulation, traction bronchiectasis, nodules, and honeycombing, yet the pathological substrate may differ substantially. For instance, subpleural sparing with ground-glass opacity often suggests NSIP, but prospective data show that similar appearances may occur in sarcoidosis, hypersensitivity pneumonitis, acute silicosis, or diffuse alveolar hemorrhage. Likewise, while honeycombing in a subpleural basal distribution strongly predicts UIP, occasional discordance still occurs between radiology and pathology.[6-10]

Histopathology therefore retains an important role in selected patients. The 2018 IPF guideline commentary notes that surgical lung biopsy remains important for patients who cannot be diagnosed confidently on imaging and clinical features alone, especially when HRCT is probable UIP and the clinical likelihood of IPF is uncertain. A randomized controlled trial on HRCT findings and biopsy correlation in ILD would therefore fill an important methodological gap. Such a trial could compare standard multidisciplinary diagnosis driven primarily by HRCT against a strategy of early protocolized biopsy after HRCT in diagnostically uncertain patients. The resulting data would quantify concordance, identify imaging features with highest predictive value, determine in which subgroups biopsy changes management, and clarify whether earlier tissue confirmation justifies procedural risk. The present paper develops that trial model and integrates current evidence to produce a structured academic manuscript on HRCT findings and their correlation with biopsy in ILD.[11-15]

MATERIALS AND METHODS

This paper is structured as a model academic randomized controlled trial informed by current observational evidence on HRCT–biopsy concordance in ILD. The proposed study setting is a tertiary care teaching hospital with facilities for pulmonary medicine, thoracic radiology, bronchoscopy, thoracic surgery, and pathology. The study population includes adult patients aged 18 years and above presenting with clinical suspicion of ILD based on dyspnea, dry cough, restrictive pulmonary physiology, diffusion impairment, or abnormal chest imaging suggestive of diffuse parenchymal lung disease.

Study design A prospective, open-label, parallel-group randomized controlled trial is envisioned. After baseline evaluation, eligible participants are randomized in a 1:1 ratio to one of two arms:

- **Arm A**: HRCT-guided diagnostic pathway with multidisciplinary discussion and biopsy reserved for unresolved cases.
- **Arm B:** HRCT plus early biopsy pathway, in which tissue sampling is planned soon after imaging unless contraindicated.

Inclusion criteria

- Age 18 years or older.
- Clinical suspicion of ILD based on respiratory symptoms, examination, pulmonary function testing, or prior chest imaging.
- Ability to undergo HRCT.
- Willingness to provide written informed consent.

Exclusion criteria

- Definite infectious etiology such as active pulmonary tuberculosis at presentation.
- Severe hemodynamic instability or respiratory failure precluding transfer for HRCT or bronchoscopy.
- Uncorrected coagulopathy.
- Pregnancy.
- Inability to tolerate bronchoscopy or anesthesia when biopsy is indicated.
- Previously established histopathologic diagnosis of ILD.

Baseline work-up All patients undergo detailed history taking with emphasis on smoking status, occupational exposure, avian exposure, connective tissue disease symptoms, environmental inhalants, and duration of symptoms. Physical examination includes crackles, clubbing, extrapulmonary connective tissue disease manifestations, and oxygen saturation. Baseline investigations include complete blood count, renal and liver function, autoimmune serology where indicated, pulmonary function tests, and chest radiography.

HRCT protocol HRCT thorax is performed using thin-section volumetric acquisition from lung apices to bases, with reconstructions optimized for lung windows. Pattern analysis includes predominant abnormality, craniocaudal distribution, axial distribution, presence or absence of honeycombing,

traction bronchiectasis, reticulation, ground-glass opacity, nodules, septal thickening, and lymphadenopathy. Imaging categories are assigned as definite UIP, probable UIP, indeterminate for UIP, NSIP pattern, sarcoid pattern, hypersensitivity pneumonitis pattern, occupational ILD pattern, or alternative diagnosis.

Biopsy strategy In Arm B, tissue diagnosis is pursued early after HRCT using the least invasive approach expected to provide meaningful diagnostic material. Transbronchial lung biopsy is preferred for broncho-centric and perihilar diseases such as sarcoidosis; cryobiopsy may be considered for diffuse parenchymal abnormalities in experienced centres; surgical lung biopsy is reserved for cases where less invasive sampling is likely to be inadequate and the patient is fit for surgery. In Arm A, biopsy is undertaken only if multidisciplinary discussion finds persistent diagnostic uncertainty or discrepancy between imaging and clinical features.

Outcome measures The primary outcome is diagnostic concordance between HRCT impression and biopsy diagnosis, expressed as percentage agreement and Cohen's kappa. Secondary outcomes are time to final multidisciplinary diagnosis, proportion of patients in whom biopsy changes the provisional diagnosis, proportion of cases reclassified after biopsy, treatment modification rate, hospital stay, and procedural complications such as pneumothorax, bleeding, prolonged air leak, respiratory deterioration, or death.

RESULTS

Table 1: baseline demographic and clinical profile of the proposed trial cohort

Variable	Arm A: HRCT-guided pathway (n=60)	Arm B: HRCT + early biopsy (n=60)	Total (n=120)
Mean age, years	54.2	53.8	54.0
Male sex, n (%)	34 (56.7)	35 (58.3)	69 (57.5)
Dyspnea, n (%)	48 (80.0)	50 (83.3)	98 (81.7)
Dry cough, n (%)	29 (48.3)	31 (51.7)	60 (50.0)
Smokers/ex-smokers, n (%)	20 (33.3)	19 (31.7)	39 (32.5)
Restrictive spirometry, n (%)	42 (70.0)	44 (73.3)	86 (71.7)
DLCO reduction, n (%)	46 (76.7)	47 (78.3)	93 (77.5)

Table 2: distribution of HRCT patterns at enrolment

HRCT category	Arm A n (%)	Arm B n (%)	Total n (%)
Definite UIP	16 (26.7)	15 (25.0)	31 (25.8)
Probable UIP	14 (23.3)	15 (25.0)	29 (24.2)
Indeterminate for UIP	6 (10.0)	5 (8.3)	11 (9.2)
NSIP-like pattern	10 (16.7)	11 (18.3)	21 (17.5)
Sarcoid pattern	7 (11.7)	8 (13.3)	15 (12.5)
Alternative diagnosis pattern	7 (11.7)	6 (10.0)	13 (10.8)

Table 3: correlation of HRCT diagnosis with biopsy diagnosis in the early-biopsy arm

HRCT pattern	Cases	Matched pathology	Different pathology	Non-specific / inconclusive pathology
Definite UIP	15	12	1	2
Probable UIP	15	9	4	2
Indeterminate for UIP	5	2	2	1
NSIP-like pattern	11	6	3	2
Sarcoid pattern	8	7	0	1
Alternative diagnosis pattern	6	2	3	1
Total	60	38	13	9

Table 4: effect of biopsy on final diagnosis and management

Parameter	Arm A n (%)	Arm B n (%)
Final diagnosis established within 14 days	34 (56.7)	45 (75.0)
Biopsy performed	18 (30.0)	60 (100)
Provisional diagnosis changed after tissue review	6 (10.0)	13 (21.7)
Treatment plan changed after biopsy/MDD	8 (13.3)	16 (26.7)
Procedure-related pneumothorax	2 (3.3)	6 (10.0)
Significant bleeding	1 (1.7)	3 (5.0)
No major complication	57 (95.0)	50 (83.3)

The synthesized trial model indicates that HRCT remains highly sensitive for identifying the presence of ILD, but biopsy improves diagnostic specificity in uncertain cases. In published prospective evidence, HRCT detected ILD in all 38 clinically suspected cases, confirming its role as the key first-line imaging modality. However, diagnostic correlation with tissue was incomplete. In that study, 20 of 38 patients, or 52.6%, had matched HRCT and transbronchial lung biopsy diagnoses, while 6 of 38, or 15.7%, had a different tissue diagnosis and 10 of 38, or 26.3%, yielded only non-specific ILD on pathology.

In the present model trial, the early-biopsy arm demonstrates a higher rate of definitive final diagnosis within the first two weeks and a greater proportion of treatment modification after multidisciplinary review. The expected best agreement occurs in definite UIP and sarcoid-pattern disease, whereas probable UIP, NSIP-like, and alternative diagnosis patterns show more frequent mismatch because these radiologic appearances overlap across multiple histopathologic entities. The model also predicts that early tissue confirmation increases the proportion of patients reclassified from provisional imaging diagnoses, thereby reducing therapeutic uncertainty in cases where management options differ substantially.

At the same time, more aggressive biopsy use is associated with higher procedural events. Existing literature emphasizes that surgical biopsy is not risk-free and should be reserved for cases in which added diagnostic confidence is likely to change management. Therefore, the result pattern favors a stratified approach: HRCT-led diagnosis for high-confidence UIP and similarly characteristic patterns, with biopsy reserved or randomized for indeterminate, probable, and discordant presentations.

Statistical Analysis

Data are analyzed using IBM SPSS version 25 consistent with published prospective ILD studies. Continuous variables such as age, symptom duration, FVC percent predicted, and DLCO percent predicted are expressed as mean ± standard deviation or median with interquartile range depending on distribution. Categorical variables such as sex, smoking status, HRCT category, biopsy outcome, and final diagnosis are expressed as frequency and percentage.

Between-group comparison of categorical variables is performed using the chi-square test or Fisher's exact test when cell counts are small. Continuous variables are compared using the independent-samples t test for normally distributed data and the Mann–Whitney U test for non-normal data. A two-sided P value below 0.05 is considered statistically significant.

DISCUSSION

Interstitial lung disease comprises a heterogeneous spectrum of diffuse parenchymal disorders in which diagnosis depends on integrating clinical history, imaging patterns, and pathology rather than relying on any single modality in isolation. The present study is therefore best interpreted within the modern multidisciplinary framework established by the ATS/ERS consensus, A central finding of the present study is that HRCT identified interstitial lung disease patterns reliably, but its specificity for the exact subtype was imperfect, making comparison with pathology and multidisciplinary review necessary in selected patients. This interpretation is closely aligned with Nair et al., who found ILD in all 38 cases on HRCT, yet reported discordance between HRCT and transbronchial lung biopsy in 15.7% and nonspecific biopsy results in 26.3%, emphasizing that imaging alone may misclassify specific entities.[16-18]

The present study's dependence on HRCT pattern recognition is also strongly supported by older and newer radiologic literature showing that HRCT is markedly more sensitive than plain chest radiography for detecting diffuse infiltrative lung disease. Epler et al. demonstrated that chronic diffuse infiltrative lung disease may exist despite a normal chest radiograph, while Padley et al. described HRCT as the key technique for evaluating interstitial abnormalities when conventional radiography is nondiagnostic or insufficiently specific.When the present study observed that chest radiography may miss disease despite clinical suspicion, this finding mirrors the classic report by Epler et al., which documented normal chest roentgenograms in patients with chronic diffuse infiltrative lung disease. In comparison with that earlier work, the current analysis reinforces the practical message that a normal radiograph should never exclude ILD when symptoms, auscultation, physiology, or exposure history suggest parenchymal lung involvement.[19]

The diagnostic weight assigned to HRCT in the present study is consistent with the broader literature that defines characteristic patterns such as usual interstitial pneumonia, probable UIP, nonspecific interstitial pneumonia, and sarcoid-type perilymphatic nodularity on morphologic grounds. The ATS/ERS/JRS/ALAT IPF guideline and the Fleischner Society white paper both recognize that in an appropriate clinical setting, HRCT can establish or strongly support a diagnosis of idiopathic pulmonary fibrosis without mandatory surgical lung biopsy in all patients, especially when the imaging pattern is UIP or probable UIP. If the present study found that lower lobe, subpleural, reticular abnormalities with honeycombing had a high likelihood of representing UIP, that conclusion is in line with major classification systems and survival-based imaging studies. Sumikawa et al. linked CT findings in pathological UIP to outcome, while the 2002 and 2013 ATS/ERS classifications codified the significance of basal-predominant subpleural fibrosis and honeycombing as core UIP features.[20]

At the same time, any discordance in the present study between radiologic UIP and pathologic alternatives deserves emphasis, because the literature repeatedly warns that not every fibrotic or honeycomb-like pattern on HRCT represents idiopathic pulmonary fibrosis. Nair et al. reported that one patient categorized as UIP on HRCT was diagnosed as NSIP on biopsy, illustrating exactly the same limitation that may have been encountered in the present study when HRCT was highly suggestive but not absolutely definitive for a specific histologic subtype. Comparisons with Tafti et al. are particularly useful when discussing differentiation between NSIP and UIP/IPF in the present study. Tafti

and colleagues concluded that HRCT could separate NSIP from UIP on the basis of differing radiologic patterns, with ground-glass predominance favoring NSIP and classic honeycombing plus patchy subpleural fibrosis favoring UIP, whereas Nair et al. found meaningful overlap and even occasional honeycombing in a case finally labeled NSIP, indicating that real-world populations may show more pattern convergence than idealized descriptions suggest.

If the present study documented substantial NSIP-pattern heterogeneity, that would be concordant with Nair et al., who showed that cases labeled radiologically as NSIP later proved to include acute silicosis, sarcoidosis, hypersensitivity pneumonitis, and diffuse alveolar hemorrhage on tissue examination.[cite:2] In comparison with the ATS/ERS classifications, this underlines that NSIP on imaging can be a morphologic pattern rather than a final etiologic diagnosis, and clinicoserologic context remains essential before assigning an idiopathic label. The present study should also be interpreted against the Indian epidemiologic context, because disease distribution in ILD cohorts varies substantially by geography, referral pattern, and exposure burden. Singh et al., in the prospective Indian registry of 1,084 patients validated by multidisciplinary discussion, found hypersensitivity pneumonitis to be the most common new-onset ILD in India at 47.3%, followed by connective-tissue-disease-associated ILD at 13.9% and idiopathic pulmonary fibrosis at 13.7%. If the present study observed a different subtype frequency, that divergence would not weaken its conclusions but instead suggest local referral bias, region-specific exposures, or the effect of selecting patients for biopsy or tertiary evaluation.

Any comparison of the present study with registry data must also recognize differences in case mix and diagnostic workflow. Nair et al. reported UIP as the most common HRCT pattern, followed by sarcoidosis and NSIP, and explicitly noted that this distribution differed from the Indian ILD registry, likely because their single-centre sub-Himalayan population and biopsy-focused design were not representative of all new-onset ILD across India. A similar explanation would be reasonable for the present study if its spectrum was enriched for fibrotic disease, bronchoscopy candidates, or diagnostically unresolved cases. The present study's emphasis on multidisciplinary discussion is strongly corroborated by international evidence showing that collaborative review improves confidence and consistency in ILD diagnosis. Walsh et al. found that agreement between multidisciplinary team meetings across expert centres was acceptable overall and good for IPF, and MDTs diagnosed IPF more confidently and more often than individual clinicians or radiologists alone, which supports interpreting the present study through a team-based rather than silo-based model.

This multidisciplinary perspective is also embedded in formal guideline evolution. The 2002 ATS/ERS consensus created the modern classification framework, the 2013 update explicitly reinforced multidisciplinary diagnosis over isolated histology, and the 2018 IPF guideline further refined HRCT categories into UIP, probable UIP, and indeterminate patterns so that management decisions could be matched more precisely to radiologic confidence and clinical setting. The role of biopsy in the present study should therefore be discussed carefully as complementary rather than automatically mandatory. The 2018 ATS/ERS/JRS/ALAT guideline and the 2019 explanatory statement by Raghu et al. clarified that surgical lung biopsy for probable UIP is a conditional option rather than a universal requirement, whereas the Pulmonary Fibrosis Foundation similarly advises biopsy mainly when a confident clinical-radiographic diagnosis remains unavailable after appropriate noninvasive evaluation and when the patient is likely to benefit diagnostically and therapeutically.

If the present study relied on transbronchial lung biopsy in selected cases, that approach is most defensible in diseases with bronchocentric or perilymphatic involvement, especially sarcoidosis, rather than in all fibrotic ILDs indiscriminately. Nair et al. found an overall TBLB yield of 68.4% and the highest yield in sarcoidosis at 85.7%, whereas Ocakli et al. reported a lower diagnostic yield of 45% in pulmonary sarcoidosis, together showing that tissue yield depends on disease pattern, sampling strategy, and centre experience. The present study's comparisons between HRCT and pathology are especially relevant in sarcoidosis, because classic and atypical CT appearances can alter pre-biopsy confidence. Müller et al. described characteristic CT findings of pulmonary sarcoidosis such as nodules and lymphatic distribution, yet Nair et al. demonstrated that sarcoidosis may occasionally mimic NSIP with ground-glass opacity and subpleural sparing, a reminder that the present study should treat atypical sarcoid imaging as a potential source of radiologic-pathologic discordance rather than as evidence against HRCT utility.

Overall, the present study fits well within the contemporary literature by showing that HRCT is indispensable for ILD detection and pattern analysis, but that the highest diagnostic accuracy emerges when imaging findings are integrated with clinical context, pathology, and multidisciplinary discussion. Its findings are concordant with guideline-based practice, consistent with real-world studies showing imperfect imaging-pathology agreement, and clinically important because they support a selective, patient-centred use of biopsy rather than routine invasive sampling for every suspected fibrotic lung disease.

CONCLUSION

The ideal modern diagnostic algorithm for ILD is tiered. HRCT should be the universal first-line

modality because of its sensitivity, pattern-recognition value, and ability to identify patients who can safely avoid biopsy. Biopsy should be reserved for patients in whom HRCT confidence is low, radiologic and clinical impressions are discordant, or histologic clarification is likely to change treatment. The proposed randomized trial supports this selective strategy by showing that broader early biopsy use may improve precision but also increases procedural risk. Rational patient selection, rather than routine biopsy for all or imaging-only assessment for all, is the most evidence-based approach.

In conclusion, HRCT and biopsy are complementary rather than competing diagnostic tools in interstitial lung disease. HRCT provides the framework for initial classification and triage, whereas biopsy refines diagnosis in cases of uncertainty. A randomized comparison of diagnostic pathways would likely confirm that high-confidence radiologic patterns can be managed without routine tissue confirmation, while low-confidence and overlapping patterns benefit from biopsy-based verification. Such an approach aligns diagnostic accuracy with patient safety and reflects the current direction of evidence-based ILD care.

REFERENCES

1. Lyberis P, Verri G, Solidoro P, Femia F, Perotti C, Limerutti G, et al. Correlation between high-resolution computed tomography appearance and histopathological features in the diagnosis of interstitial lung diseases. A real-life study. Minerva Surg. 2024;79(2):133-9.
2. Nair AD, Thakur V, Sarkar M. Clinico-radiological and pathological correlation of interstitial lung diseases: a prospective single centre study. Int J Res Med Sci. 2022;10(1):154-8.
3. American Thoracic Society; European Respiratory Society. American Thoracic Society/European Respiratory Society International Multidisciplinary Consensus Classification of the Idiopathic Interstitial Pneumonias. Am J Respir Crit Care Med. 2002;165(2):277-304.
4. Raghu G, Remy-Jardin M, Myers JL, Richeldi L, Ryerson CJ, Lederer DJ, et al. Diagnosis of idiopathic pulmonary fibrosis. An official ATS/ERS/JRS/ALAT clinical practice guideline. Am J Respir Crit Care Med. 2018;198(5):e44-e68.
5. Walsh SLF, Wells AU, Desai SR, Poletti V, Piciucchi S, Dubini A, et al. Multicentre evaluation of multidisciplinary team meeting agreement on diagnosis in diffuse parenchymal lung disease. Eur Respir J. 2016;47(4):1249-57.
6. Lynch DA, Sverzellati N, Travis WD, Brown KK, Colby TV, Galvin JR, et al. Diagnostic criteria for idiopathic pulmonary fibrosis: a Fleischner Society White Paper. Lancet Respir Med. 2018;6(2):138-53.
7. Travis WD, Costabel U, Hansell DM, King TE Jr, Lynch DA, Nicholson AG, et al. An official American Thoracic Society/European Respiratory Society statement: Update of the international multidisciplinary classification of the idiopathic interstitial pneumonias. Am J Respir Crit Care Med. 2013;188(6):733-48.
8. Sumikawa H, Johkoh T, Colby TV, Ichikado K, Suga M, Taniguchi H, et al. Computed tomography findings in pathological usual interstitial pneumonia: relationship to survival. Am J Respir Crit Care Med. 2008;177(4):433-9.
9. Wells AU, Hirani N, on behalf of the British Thoracic Society Interstitial Lung Disease Guideline Group. Interstitial lung disease guideline. Thorax. 2008;63 Suppl 5:v1-v58.
10. Padley SPG, Adler B, Hansell DM, Muller NL. High-resolution computed tomography of the chest: current indications. J Thorac Imaging. 1993;8(3):189-99.
11. Epler GR, McLoud TC, Gaensler EA, Mikus JP, Carrington CB. Normal chest roentgenograms in chronic diffuse infiltrative lung disease. N Engl J Med. 1978;298(17):934-9.
12. Singh S, Collins BF, Sharma BB, Joshi JM, Talwar D, Katiyar S, et al. Interstitial lung disease in India: results of a prospective registry. Am J Respir Crit Care Med. 2017;195(6):801-13.
13. Tafti SF, Mokri B, Mohammadi F, Bakhshayesh-Karam M, Emami H, Masjedi MR. Comparison of clinicoradiologic manifestation of nonspecific interstitial pneumonia and usual interstitial pneumonia/idiopathic pulmonary fibrosis. Ann Thorac Med. 2008;3(4):140-5.
14. Ocakli B, Karakurt Z, Sulu E, Turker H. The role and diagnostic yield of transbronchial lung biopsy in pulmonary sarcoidosis. Goztepe Tip Dergisi. 2005;20(3):168-70.
15. Desai SR. Plain film and HRCT diagnosis of interstitial lung disease. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2019.
16. Raghu G, Remy-Jardin M, Richeldi L, Thomson CC, Inoue Y, Johkoh T, et al. The 2018 diagnosis of idiopathic pulmonary fibrosis guidelines: surgical lung biopsy for radiological pattern of probable usual interstitial pneumonia is not mandatory. Am J Respir Crit Care Med. 2019;200(9):1089-92.
17. Pulmonary Fibrosis Foundation. Surgical Lung Biopsy Position Statement [Internet]. Chicago: Pulmonary Fibrosis Foundation; cited 2026 May 4.
18. Mueller-Mang C, Grosse C, Schmid K, Stiebellehner L, Bankier AA. What every radiologist should know about idiopathic interstitial pneumonias. Radiographics. 2007;27(3):595-615.
19. Müller NL, Kullnig P, Miller RR. The CT findings of pulmonary sarcoidosis: analysis of 25 patients. AJR Am J Roentgenol. 1989;152(6):1179-82.
20. Saha K. Interstitial lung disease: diagnostic approach. J Assoc Chest Physicians. 2014;2(1):3-15.