PULMONARY PATHOLOGY JOURNAL CLUB

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Articles for Discussion

1. Febres-Aldana CA, Vanderbilt CM, et al. Pulmonary Solid and Granular Adenocarcinomas Expressing HepPar1/CPS1: Highly Aggressive Tumors Exhibiting Mitochondrial Adaptation to STK11 Mutations Rather Than Hepatoid Differentiation. Mod Pathol. 2026;39(4):100965. PMID: 41580239. DOI: 10.1016/j.modpat.2026.100965.

Background:

Hepatoid lung carcinoma is currently a recognized variant defined by extrahepatic tumors expressing hepatocellular markers — most prominently HepPar1, an antibody that targets the mitochondrial enzyme CPS1. Recent work has shown HepPar1/CPS1 accumulation in STK11-mutant lung adenocarcinomas, presumably as a metabolic adaptation, but the implications for the "hepatoid lung carcinoma" diagnostic concept have not been formally examined.

Methods:

- 17 lung tumors prospectively diagnosed as hepatoid based on solid/trabecular growth and HepPar1 positivity were assembled and re-evaluated with an extended hepatocellular IHC panel (alpha-fetoprotein, Arginase1, Glypican3, albumin in situ hybridization).
- A parallel comparison cohort of 22 conventional acinar-predominant LUADs with HepPar1 expression was assembled to test whether the STK11 association generalizes beyond solid/granular morphology (identified by performing HepPar1 on a larger cohort of conventional LUAD).
- Pneumocytic markers (TTF1, napsin A) and mucicarmine were performed on all cases; many cases also had CK7, p40, SMARCA4 (BRG1), and SMARCA2 (BRM) performed
- Next-generation sequencing performed on all cases. Mitochondrial content was quantified (estimated) by multiple methodologies including electron microscopy, extrapolation from number of NGS reads, and SDHB IHC staining intensity.

Results:

- All 17 "hepatoid" cases were robustly HepPar1-positive but completely negative for AFP, Arginase1, Glypican3, and albumin in situ hybridization.
- Morphology was solid-trabecular with polygonal cells that had expanded granular-eosinophilic to vacuolated-clear cytoplasm (hence their proposed term “Solid and Granular Adenocarcinoma” (SAGA)); focal-to-moderate intracytoplasmic mucin was consistently present.
- STK11 loss-of-function mutations or biallelic loss were detected in 17/17 cases, an enrichment that was very statistically robust compared to unselected LUADs ($P<0.00001$, vs >2,500 LUAD controls).
- Pneumocytic markers (TTF1, napsin A) were entirely negative (except for cytoplasmic TTF1 in a subset), leading to diagnostic confusion at metastatic sites.
- All of the 17 hepatoid cases harbored a smoking-related molecular signature and mutations in other genes associated with aggressive disease (KEAP1, KRAS, TP53, and/or SMARCA4)
- Median overall survival 5.8 months vs 25 months for SAGA/hepatoid cases vs other stage-matched LUADs, respectively ($P=0.0002$); although the stage-matched controls were a historic cohort from
- In the parallel cohort of 22 conventional acinar LUADs with HepPar1 expression, many overlapping features were observed: invariable STK11 alteration, increased granular cytoplasm, lower TTF1, and poor prognosis.

Take home message:

Isolated HepPar1 positivity in a TTF-1-negative (i.e. no *nuclear* TTF1 staining) solid lung adenocarcinoma is a strong indicator of STK11-mutant LUAD with mitochondrial adaptation, rather than bona fide hepatoid differentiation.

Practical implications:

(1) Evaluate hepatoid-appearing lung tumors with an extended hepatocellular panel (AFP, Arginase1, Glypican3) before deciding on hepatoid lung carcinoma ("SAGA") vs metastatic HCC

(2) As these adenocarcinomas typically have STK11 mutations (and often SMARCA4 deficiency) they tend to have an aggressive clinical course and ICI resistance

(3) In metastatic tumors of unknown primary with this morphology, HepPar1 alone should not establish hepatocellular origin

2. Wei J, Ma X, et al. Diagnostic Potential of a Novel Immunohistochemical Marker, GFPT2, in Differentiating Mesothelioma From Its Morphological Mimics. Histopathology. 2026;88(5):1016-1026. PMID: 41392993. DOI: 10.1111/his.70062.

Background:

The authors had previously identified GFPT2 (glutamine-fructose-6-phosphate transaminase 2) as highly expressed in mesothelioma tissues and now test its diagnostic utility against a variety of entities in the differential of mesothelioma.

Methods:

- Immunohistochemical analysis of GFPT2 on 101 mesothelioma cases plus 266 morphological mimics.
- Also tested for comparison: 100 NSCLC, 40 high-grade serous ovarian carcinoma, 6 epithelioid hemangioendothelioma, 33 solitary fibrous tumor, 23 aggressive fibromatosis, 6 synovial sarcoma, 7 dedifferentiated liposarcoma, 6 leiomyosarcoma, 4 MPNST, 8 sarcomatoid carcinoma (more sarcomatoid carcinomas, and sarcomatoid mesotheliomas for that matter, would've been nice)
- Benign comparators: 20 reactive mesothelial hyperplasia (RMH), 13 well-differentiated papillary mesothelial tumor (WDPMT).
- GFPT2 antibody and scoring threshold defined as positive if H-score ≥ 90, which was set for very high specificity while maintaining good sensitivity

Results:

- GFPT2 positive in 86/101 mesotheliomas (85.1%) and 0/20 reactive mesothelial hyperplasia (0%) and 0/13 WDPMT (0%).
- Negative in NSCLC (0/100), high-grade serous ovarian carcinoma (0/40), solitary fibrous tumor (0/33), synovial sarcoma (0/6), leiomyosarcoma (0/6).
- Low rates in EHE (1/6, 17%), aggressive fibromatosis (5/23, 22%), MPNST (1/4, 25%), sarcomatoid carcinoma (1/8, 13%).
- Notable false-positive: dedifferentiated liposarcoma (6/7, 86%).
- Reported sensitivity 85.2%, specificity 94.7% across the full diagnostic differential.

Take home message:

GFPT2 is a promising new mesothelioma IHC marker with sensitivity ~85% and specificity ~95%, with the added benefit of being negative in reactive mesothelial hyperplasia and well-differentiated papillary mesothelial tumor. Critical caveat: almost all dedifferentiated liposarcoma cases were positive (86%), so if use MDM2 IHC/FISH and other mesothelial markers/keratins if DDLS is in the differential.

Still needs to be externally validated, ideally in studies with a higher number of sarcomatoid mesotheliomas, sarcomatoid carcinomas, and benign/reactive mesothelial conditions if people are going to want to use it as a marker of malignancy

3. Saha M, Tran TV, et al. Genomic Characterization of Lung Cancer in Never-Smokers Using Deep Learning. Mod Pathol. 2026;39(4):100973. PMID: 41638573. DOI: 10.1016/j.modpat.2026.100973.

Background:

Deep learning models have been applied to H&E whole-slide images to predict molecular alterations in lung adenocarcinoma, but most prior work has been trained on smoker-dominant cohorts. Never-smoker LUAD (NS-LUAD) is molecularly and histologically distinct — enriched for EGFR mutations, ALK fusions, and a different mutational signature spectrum — and existing models perform inconsistently when applied to this population. A purpose-built model could plausibly accelerate molecular triage, particularly in tissue-limited or TAT-constrained settings.

Methods:

- Used a customized deep convolutional neural network based on ResNet50 architecture, optimized for multilabel classification – i.e. ability to simultaneously predict the presence of multiple independent molecular aberrations, including more than one of the aberrations
- Training and evaluation on 495 WSIs from 410 cases in the Sherlock-Lung never-smoker cohort (multi-institutional, international: 67% European ancestry, 29% East Asian, 4% “Admixed American”, 0.4% African).
- Training (n=346) and held-out validation (n=149) sets were patient-disjoint; 10% of training set used for stratified ten-fold cross-validation. 5,316,629 256×256 tiles extracted at 20× magnification.
- Sixteen molecular features predicted simultaneously: EGFR, KRAS, TP53, RBM10 mutations; MDM2 amplification, kataegis, CDKN2A deletion, ALK fusion, whole-genome doubling; EGFR L858R and exon 19 deletion hotspots; KRAS G12C/G12V/G12D hotspots; TMB; APOBEC mutational signature.

Results:

- AUROC 0.84-0.93 for 11 features: EGFR (0.93), KRAS (0.92), TP53 (0.91), RBM10 (0.90), MDM2 amplification (0.93), kataegis (0.91), CDKN2A deletion (0.89), ALK fusion (0.86), whole-genome doubling (0.84), EGFR L858R (0.92), EGFR exon 19 del (0.86).
- Low performance: TMB (AUROC 0.67), APOBEC signature (0.57), KRAS G12C (0.74), KRAS G12V (0.55), KRAS G12D (0.43) – model had less KRAS-mutated cases to train on in it’s defense
- Customized model significantly outperformed Inception-v3 across most features (DeLong $P<0.01$ for several drivers) and was numerically better than standard ResNet50.
- Class-activation maps highlighted spatial regions contributing to molecular predictions, with EGFR-mutant predictions concentrating on lepidic/papillary areas.

Take home message:

A purpose-built CNN can predict 11 clinically relevant molecular alterations from H&E whole-slide images in never-smoker LUAD with AUROC 0.84-0.93 (outperforming other CNNs - Inception-v3 and standard ResNet50). The likely use case is molecular triage - prioritizing tissue or TAT-limited cases for reflex testing and flagging morphologic ambiguity. External validation on tissue-limited biopsies and prospective evaluation in a TAT-driven workflow would be the best next steps to move towards applicability.

4. Weissferdt A, Moran CA. Solid, Cystic, and Mucinous Spindle Cell Thymomas (WHO Type A): A Clinicopathological and Immunohistochemical Study of 7 Cases. Hum Pathol. 2026;170:106050. PMID: 41580205.

Background/Methods:

Description of 7 cases of a morphologic variant of WHO type A (spindle cell) thymoma characterized by the combination of solid areas, cystic changes, and focal mucinous differentiation.

Cohort:

- 5 men, 2 women; aged 43–57 years (mean 50).
- All had anterior mediastinal masses on imaging, and all underwent surgical resection via thoracotomy.

Gross Pathology

All well circumscribed, 2.7–4.8 cm in greatest diameter; 6/7 tumors were encapsulated without overt invasion; one showed minimal invasion outside the capsule

Histologic Features

The tumors exhibited a triphasic morphologic pattern:

- Solid spindle cell component (WHO type A appearance), cystic changes, plus areas of mucinous differentiation
- The admixed areas of mucinous differentiation are the somewhat novel/unique finding, although the same pair of authors seemingly described a set of 12 cases that sound quite similar in 2013 (PMID: 23528863):
    - In this current study, they included glandular structures lined by mucinous epithelium, scattered individual mucinous cells, extracellular mucin pools, and partial lining of cyst walls by mucinous epithelium. Mucicarmine histochemical staining confirmed the presence of intracellular mucin.

Immunohistochemical Profile

- Spindle cell component: the classic immunoprofile of type A thymoma
- Mucinous cell component: Variably positive for CK7, CK20, and CDX2

Take home:

This paper builds on the same group's earlier work describing thymomas with prominent glandular differentiation (12 cases, 2013), in which 7 of 12 tumors were also type A and 4 showed mucinous features — though the current series specifically argues that the combined solid-cystic-mucinous pattern is a distinct morphologic variant. The recognition of glandular and mucinous differentiation in type A thymomas is consistent with the known morphologic versatility of this tumor type, which can also exhibit rosette-like, pseudoglandular, microcystic, papillary, and glomeruloid growth patterns.

Articles for Notation

Neoplastic

Severson DT, Freyaldenhoven S, et al. Multiomic, Histologic, and scRNA-seq Profiling of Pleural Mesothelioma Reveals Negative Prognosis Associated With a Novel Uncommitted Molecular Phenotype. J Thorac Oncol. 2026;21(4):103529. PMID: 41319863. DOI: 10.1016/j.jtho.2025.11.019.

Tumor samples from 35 patients with pleural mesothelioma underwent integrated profiling: single-cell RNA sequencing, bulk exome and RNA sequencing, optical genome mapping, spatial transcriptomics, and standardized histologic review (19 epithelioid, 13 biphasic, 3 sarcomatoid; plus 3 additional non-malignant pleural tissue controls). Goal was to identified candidate drivers and prognostic biomarkers of the "malignant cell state".

Take home: Pleural mesothelioma comprises three malignant cell states (epithelioid, sarcomatoid, uncommitted) that coexist within individual clones rather than being "clonally segregated" by morphologic subtype. MEST and MSLN IHC are potential surrogates for the uncommitted and epithelioid states respectively (High MEST staining → shorter overall survival in epithelioid meso; high MSLN staining → longer overall survival in epithelioid and biphasic meso)

4. Sun L, Cui Y, et al. Non-Small Cell Lung Carcinomas With Diffuse Co-Expression of TTF-1 and p40: Clinical, Pathological and Molecular Characterization of a Tumor Subtype. Virchows Arch. 2026;488(4):897-907. PMID: 41665643. DOI: 10.1007/s00428-026-04436-y.

Background:

Comprehensive literature review of previously reported cases of NSCLC with diffuse TTF-1/p40 co-expression, with survival curves constructed from published data.

Take home: Diffuse TTF-1 and p40 co-expression in a single NSCLC morphologic population may represent a discrete biphenotypic clinicopathologic subtype with poor prognosis, distinct from adenocarcinoma, squamous cell carcinoma, and adenosquamous carcinoma

He Y, Waltman P, et al. Longitudinal Genomic Analysis of Five Cases of Recurrent Thymomas. J Thorac Oncol. 2026;21(4):103521. PMID: 41274415.

WES on 3 primary and 11 recurrent thymoma samples from 5 patients documents clonal evolution with higher TMB in recurrence samples (no statistical tests done as it's only n = 5) and one case of "histologic transformation from type AB to B2/B3" *(….or treatment-induced selection of a B2/B3 component; although given the images they provided in figure 2 I'm not really sure if it was ever type AB in the first place…)*

Take home: The authors argue that recurrent thymomas may be associated with molecular (and sometimes histologic) progression via clonal evolution

Miyakoshi J, Shiraishi K, et al. Chromosome 15q15 Deletion Drives Brain Metastasis in NSCLC. J Thorac Oncol. 2026;21(4):103511. PMID: 41207477.

WGS/WES of >1,000 primary NSCLCs plus matched primary-brain metastasis pairs identifies 15q15 deletion as a driver of brain metastasis. 15q15 deletions significantly co-occurred with EGFR mutations, but tumors with 15q15 deletion consistently had higher cumulative risk of brain metastases regardless of whether there was a co-mutation in EGFR.

Nicoś M, Stokowy T, et al. Mutational Landscapes of Brain Metastases Across Various Histological Subtypes of Lung Cancer. Lung Cancer. 2026;215:109342. PMID: 41747607.

NGS of 142 lung cancer brain metastases — TP53, H3F3A, and PMS2 mutated in >20% across histologies; LUAD-BM showed broader driver repertoires.

Pirlog R, Hofman V, et al. Single-Center Experience Using Reflex-Targeted Next-Generation Sequencing at Diagnosis of Squamous Cell Lung Carcinoma in Daily Practice. Virchows Arch. 2026;488(4):841-852. PMID: 40608133.

108 consecutive lung squamous cell carcinomas with reflex DNA + RNA NGS — ~4% harbored a TKI-actionable alteration.

Take home: Quantifies the small-but-real yield of reflex NGS in SqCC of the lung;

Suzuki K, Miyazaki K, et al. Treatment and Outcomes of Pulmonary Mucosa-Associated Lymphoid Tissue Lymphoma: A Multicenter Analysis of 186 Patients. Cancer. 2026;132(8):e70390. PMID: 41954337.

Multicenter cohort of 186 pulmonary MALT lymphoma patients (2013-2022); 4-year OS 96/92/90% across stages IE, IIE, and IV respectively, independent of first-line modality including watchful waiting.

Take home: Confident pathology diagnosis of pulmonary MALT enables the choice of watch-and-wait strategy that performs as well as immediate therapy (as virtually all patients had a favorable prognosis)

Sun Z, Liu T, et al. RET Fusion-Positive Lung Adenocarcinoma: Partner-Specific Clinicopathological Characteristics, Co-Mutation Profiles, and Implications for Targeted and Immunotherapy. Lung Cancer. 2026;214:109314. PMID: 41707338.

268 RET fusion-positive LUAD: KIF5B-RET dominates (65%), CCDC6-RET 16%; non-KIF5B partners enriched for CDKN2A co-mutations and showed longer pralsetinib PFS.

Take home: NGS reports for RET-fusion lung cancer should specify the partner and CDKN2A status — partner identity changes therapy outcome

Fernandez-Bussy S, Yu Lee-Mateus A, et al. Bronchoscopic Pulsed-Electric Field Ablation for Carcinoid Tumor: Robotic-Assisted and Non-Robotic Approach. Lung Cancer. 2026;214:109333. PMID: 41762471.

Multicenter retrospective series of bronchoscopic pulsed-electric field ablation for pulmonary carcinoid tumors in non-surgical candidates.

Take home: Pathologists should start to familiarize themselves what the PEF-related treatment effect looks like histologically *(from what I've read it's typically just bland fibrosis, often a gradient from tumor to paucicellular scar in incompletely treated tumors)*

Treatment/Clinical Trials Section:

Heymach JV, Yamamoto N, et al. First-Line Zongertinib in Advanced HER2-Mutant Non-Small-Cell Lung Cancer. N Engl J Med. 2026;394(17):1675-1684. PMID: 41985129.

Beamion LUNG-1 phase 1a-1b: zongertinib (HER2-selective oral irreversible TKI) in 74 first-line HER2-mutant NSCLC; overall response rate: 76%, 14.4 months PFS, 47% intracranial response. Molecular inclusion criteria = a HER2 mutation involving the tyrosine kinase domain.

Take home: Trial reinforces the need for ERBB2/HER2 mutation testing to be part of all multigene panel NGS tests for NSCLC (also, IHC/FISH HER2 amplification tests are not appropriate for this indication).

Park W, Kasi A, et al. Setidegrasib in Advanced Non-Small-Cell Lung Cancer and Pancreatic Cancer. N Engl J Med. 2026;394(14):1409-1420. PMID: 41879829.

Phase 1 of setidegrasib (ASP3082), a first-in-class KRAS G12D-targeted protein degrader; objective response rate: 36%, all partial responses in NSCLC at the 600 mg dose; 12-month OS: 59%

Take home: Eliminates the prior "untargetable" status of KRAS G12D, seemingly not a great response rate, but apparently the G12D mutation has been notoriously difficult to target

Jänne PA, Planchard D, et al. Survival With Osimertinib Plus Chemotherapy in EGFR-Mutated Advanced NSCLC. N Engl J Med. 2026;394(1):27-38. PMID: 41104938.

FLAURA2 final OS analysis: first-line osimertinib + platinum-pemetrexed gave median OS 47.5 vs 37.6 months for monotherapy (HR 0.77).

Take home: Reinforces the importance of accurate, fast EGFR genotyping as the osimertinib-chemo combination has a real OS gain over monotherapy

Fang W, Wu L, et al. Sacituzumab Tirumotecan in EGFR-TKI-Resistant, EGFR-Mutated Advanced NSCLC. N Engl J Med. 2026;394(1):13-26. PMID: 41124220.

Phase 3 OptiTROP-Lung04 of sac-TMT (TROP2-directed antibody-drug conjugate) vs platinum-pemetrexed after EGFR-TKI failure. TROP2 can be highly expressed in EGFR-mutated NSCLC and is associated with EGFR-TKI resistance. Overall survival (sacituzumab vs chemotherapy): HR for death 0.60 (95% CI, 0.44–0.82; P=0.001); 18-month OS: 65.8% vs. 48.0%.

Take home: Oncologists could soon be requesting TROP2 IHC on (re)biopsies of TKI-resistant EGFR+ NSCLC — labs may want to consider validating a TROP2 assay and standardize scoring methodology proactively.

Non-neoplastic

Zhang Y, Liu E, et al. Primary Pulmonary Langerhans Cell Histiocytosis: Comprehensive Clinicopathologic and Molecular Genetic Analysis of 13 Cases. Hum Pathol. 2026;170:106034. PMID: 41539487. DOI: 10.1016/j.humpath.2026.106034.

Retrospective analysis of 13 pulmonary Langerhans cell histiocytosis (PLCH) cases at a single institution that includes summary of clinical data, imaging review, histopathologic findings, IHC, BRAF V600E mutation status, and broader DNA-based NGS on 6 of the cases *(*for the purpose of this summary, I'm including PLCH in non-neoplastic category as I assume most of us think of it more as a diffuse lung disease rather than lumping it in with conventional neoplasms)*

Take home: The molecular landscape of PLCH extends beyond BRAF V600E (only 1/4 tested cases positive) to alternative MAPK activators (BRAF deletions, MAP2K1, DUSP4, RRAS) and DNA-damage repair gene alterations.

Shacker M, Wang L, et al. Pleural Space Complications After Lung Transplantation. J Thorac Cardiovasc Surg. 2026;171(4):860-870. PMID: 41338447.

Single-institution study of 840 bilateral lung-tx recipients comparing those undergoing pleural space surgery vs not.

Take home: Pleural rind, organizing pleuritis, fibrothorax, and post-tx empyema specimens are increasingly common

Ormsby AL, Matson SM, et al. Subclinical Interstitial Lung Disease in Rheumatoid Arthritis: Implications for Early Detection and Management. Chest. 2026;169(4):1018-1025. PMID: 41354403.

Review of subclinical RA-ILD; ~25% of RA patients have subclinical ILD, with MUC5B promoter variant, MMP-7, PARC, and SP-D as key biomarkers.

Take home: When evaluating lung specimens from RA patients, pathologist may be the first part of the patient's team to take note of an ILD/chronic interstitial pneumonia or other interstitial abnormalities

Seeliger B, Ruwisch J, et al. Differential Effects of Antifibrotic Treatment on Outcome Prediction via Serial Matrix Metalloproteinase-Degraded C-Reactive Protein Neoepitope Levels in Idiopathic Pulmonary Fibrosis. Chest. 2026;169(4):991-1005. PMID: 41033547.

203 IPF patients with serial MMP-degraded CRP neoepitopes during antifibrotic therapy.

Take home: Matrix-degradation biomarker activity is thought to reflect what pathologists see morphologically as fibroblastic foci progression (may eventually complement histologic UIP diagnosis?).

Chao WC, Liao SY. Comparative Outcomes and Side Effects of Immunosuppressants and Tumor Necrosis Factor Inhibitors in Sarcoidosis: A Real-World Data Analysis. Chest. 2026;169(4):1006-1017. PMID: 41077383.

Real-world comparison of second-line agents (immunosuppressants vs TNF inhibitors) in refractory sarcoidosis.

Take home: Post-treatment biopsies of sarcoidosis may show altered granuloma morphology (decreased cellularity, increased fibrosis) — pathologists should anticipate this in patients on second-line therapy.

Tiew PY, Narayana JK, et al. Aspergillus Fumigatus Sensitization Is Associated With High-Risk Bronchiectasis. Chest. 2026;169(4):932-946. PMID: 41386457.

International multicenter cohort showing Aspergillus sensitization is a marker of high-risk bronchiectasis.

Uzunhan Y, Diou C, et al. Consensus Definition of Rapidly Progressive Interstitial Lung Disease: A Critical Unmet Need to Be Led by Pneumologists. Eur Respir J. 2026;67(4):2502125. PMID: 41927078.

Letter calling for harmonised definition of rapidly progressive ILD in the CTD-ILD context.

Take home: Pathologists who sign out wedge biopsies of acutely declining ILD patients should track this debate —case definitions affect when biopsies are pursued and what the clinical questions are

Perch M, Corris P, et al. A European Multicentre, Randomised Controlled Trial of Pirfenidone in Bronchiolitis Obliterans Syndrome After Bilateral Lung Transplantation. Eur Respir J. 2026;67(4):2402154. PMID: 41232942.

Negative phase II RCT of pirfenidone vs placebo for progressive BOS after bilateral lung-tx. Total n of only 90 so more of an exploratory trial, but results between groups didn't hint at any efficacy.

Alnababteh M, Keller MB, et al. Early Post-Transplant Recipient Tissue Injury Predicts Allograft Function, Rejection and Survival in Lung Transplant Recipients: Evidence From Cell-Free DNA. Eur Respir J. 2026;67(4):2402537. PMID: 40744691.

Multicenter prospective cohort showing recipient-derived cfDNA early post-tx predicts allograft function, rejection, and survival; higher recipient-derived cfDNA levels → increased risk of acute rejection (HR = 2.3, p=0.03)) and death (HR = 3.15, p <0.001)

Take home: Adds a non-tissue, longitudinal biomarker that complements transbronchial biopsy — pathologists should anticipate cfDNA results being correlated with their histologic evaluations of acute cellular rejection.

Reviews, editorials, letters, and case reports

Leduc C, Rahimi K, et al. Well-Differentiated Papillary Mesothelial Tumour: Histologic, Molecular and Genetic Features Support a Benign Clonal Neoplasm Despite Diffuse Peritoneal Involvement. Histopathology. 2026;88(5):1104-1106. PMID: 41424264.

Letter presenting a unique case of multifocal/diffuse WDPMT arguing that this entity is clonal but biologically benign even with diffuse peritoneal involvement; includes summary of whole exome sequencing performed on tumor from three separate intraperitoneal sites

Poletti V, Ryerson CJ, et al. Tissue-Based Categorisation of Fibrotic Interstitial Pneumonias: When Early Diagnosis Can Make the Difference (Editorial). Eur Respir J. 2026;67(4):2501903. PMID: 41927071.

Editorial in the wake of the 2025 ERS/ATS multidisciplinary classification update — argues that tissue-based categorisation remains essential for prognosis and therapy.

Take home: Reaffirms the surgical pathologist's role at the center of fibrotic-ILD diagnosis even as "progressive pulmonary fibrosis" becomes a clinical phenotype.

Brugière O, Saez Gimenez B. What Can We Learn From a Negative Randomised Controlled Trial in Bronchiolitis Obliterans Syndrome After Lung Transplantation? (Editorial). Eur Respir J. 2026;67(4):2502412. PMID: 41927073.

Editorial accompanying Perch et al. (negative pirfenidone-in-BOS RCT).

Williams T, Bourdin A, et al. Reimagining CLAD as a Damage-Repair Paradigm: Biomarkers, Mechanisms and Opportunities (Editorial). Eur Respir J. 2026;67(4):2600161. PMID: 41991210.

Editorial reframing CLAD as recurrent injury-repair rather than a static fibrotic endpoint.

Lötscher J, Baarts J. Pulmonary Mucormycosis. N Engl J Med. 2026;394(7):699. PMID: 41671484.

Durand ML, Siedner MJ, et al. Case 2-2026: A 63-Year-Old Man With Pulmonary Nodules, Liver Mass, and Vision Loss. N Engl J Med. 2026;394(3):282-294. PMID: 41534046.