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ULTRA–LOW-DOSE HIGH-RESOLUTION CT FOR COMPREHENSIVE EVALUATION OF SMOKING-RELATED LUNG DISEASE IN SYMPTOMATIC ADULT SMOKERS: A PROSPECTIVE SINGLE-CENTRE STUDY

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ABSTRACT

Background: Symptomatic smokers frequently undergo chest imaging to evaluate chronic cough, dyspnoea, sputum production, or haemoptysis. High-resolution CT (HRCT) is sensitive for emphysema, airway wall disease, and smoking-related interstitial lung disease (SR-ILD), yet concerns about cumulative radiation limit routine use. We prospectively assessed the diagnostic yield and quantitative performance of a standardized ultra–low-dose HRCT thorax protocol in symptomatic current and former smokers.

Materials and Methods: Consecutive adults ≥40 years with ≥10 pack-years who presented with respiratory symptoms and were referred for CT between August 2024 and July 2025 were enrolled. Ultra–low-dose non-contrast volumetric acquisitions (target CTDIvol ≤1.5 mGy; DLP ≤60 mGy·cm) with 0.75–1.0-mm high-spatial-frequency reconstructions were obtained at full inspiration. Quantitative densitometry (%LAA−950), airway metrics (Pi10), and bronchial wall thickness were computed. Visual scoring captured emphysema pattern/severity, airway wall thickening, bronchiectasis, SR-ILD patterns, and pulmonary nodules (Lung-RADS). Spirometry was performed within 4 weeks. Associations were analysed using χ^2, ANOVA, Spearman ρ, and multivariable logistic regression.

Results: 200 participants (mean age 58.7±8.9 years; 152 men [76.0%]; median smoking 28 pack-years) were analysed. Any CT-detectable smoking-related abnormality occurred in 146/200 (73.0%). Emphysema was present in 110 (55.0%), airway wall thickening in 96 (48.0%), bronchiectasis in 44 (22.0%), and SR-ILD in 36 (18.0%); categories overlapped. Pulmonary nodules were seen in 60 (30.0%) participants; 8 (4.0%) were Lung-RADS 4. %LAA−950 correlated with FEV1%pred ($\rho=-0.46$, $p<0.001$) and pack-years ($\rho=0.42$, $p<0.001$). In multivariable models, each 10 pack-year increment increased odds of a clinically significant CT abnormality by 1.28 (95%CI 1.12–1.48; $p<0.001$).

Conclusion: Ultra–low-dose HRCT yielded a high burden of unsuspected, clinically actionable smoking-related lung disease in symptomatic smokers while maintaining sub-diagnostic-reference radiation levels. Integrating low-dose HRCT into evaluation pathways could facilitate earlier diagnosis, risk stratification, and smoking cessation counselling.

Keywords: low-dose CT; high-resolution CT; smokers; emphysema; airway disease; interstitial lung disease.

INTRODUCTION

Smoking remains a leading global cause of preventable morbidity and mortality, with chronic respiratory symptoms among the most common reasons for clinical presentation in adult smokers.[1] Respiratory manifestations encompass a

heterogeneous spectrum—ranging from small airways inflammation and bronchitis to emphysema, smoking related interstitial lung abnormalities, and malignancy—that often coexist within the same individual.[1,3] Conventional chest radiography lacks sensitivity for early or subtle parenchymal change, particularly in patients whose spirometry may still be within reference limits despite clinically relevant structural injury.[2,4] Recent guideline expansions for low dose chest CT (LDCT) in high risk smokers reflect robust evidence that cross sectional imaging detects clinically important disease at earlier, more treatable stages while mitigating (though not eliminating) radiation exposure concerns.[1,3]

HRCT provides sub millimetre spatial resolution capable of depicting centrilobular lucencies, paraseptal lines, tree in bud micronodules, cystic lesions, and early fibrotic change before they are apparent on radiography or sometimes even on standard dose volumetric CT reconstructions.[5,7] Quantitative densitometric and airway metrics derived from thin section datasets—such as the percentage of lung volume below −950 HU (%LAA−950), parametric response mapping of small airways disease, and standardized airway wall indices—have emerged as reproducible imaging biomarkers that correlate with spirometric impairment, exacerbation risk, and mortality in chronic obstructive pulmonary disease (COPD) cohorts.[6,8] Parallel work in smoking related interstitial lung disease (SR ILD) has shown that HRCT patterns (respiratory bronchiolitis, desquamative interstitial pneumonia, pulmonary Langerhans cell histiocytosis, and smoking related interstitial fibrosis, among others) influence prognosis and therapeutic decisions, particularly when recognized before advanced fibrosis develops.[5,7]

Despite these advantages, routine deployment of HRCT in symptomatic smokers outside formal lung cancer screening pathways remains inconsistent. Barriers include radiation dose accumulation from repeated imaging, variable protocol standardization across scanners, and uncertainty regarding which symptomatic smokers derive net clinical benefit from low dose high resolution examinations.[1,3,9] Moreover, data integrating comprehensive HRCT phenotyping (emphysema, airway, interstitial, and nodule assessment) within a single ultra–low dose acquisition in a real world symptomatic population are scarce.

This study was done to evaluate the diagnostic yield, quantitative imaging physiology correlations, and dose metrics of an ultra–low dose HRCT thorax protocol in symptomatic current or former smokers presenting to a tertiary radiology department. Primary objectives were to estimate the prevalence and spectrum of smoking related lung disease detectable on low dose HRCT and to examine relationships between quantitative CT metrics, smoking exposure, and spirometric parameters. Secondary objectives included characterization of incidental pulmonary nodules and estimation of patient level factors associated with clinically significant imaging abnormalities.

MATERIALS AND METHODS

Study design and setting

Prospective observational study conducted in the Department of Radiology, Mallareddy Institute of Medical Sciences, a tertiary care teaching hospital. Recruitment spanned August 2024 to July 2025. Consecutive eligible patients referred for CT evaluation of respiratory symptoms were screened. Written informed consent was obtained. The institutional ethics committee approved the protocol.

Participants

Inclusion criteria: (1) age ≥40 years; (2) current or former cigarette smoker with cumulative exposure ≥10 pack-years; (3) presence of ≥1 of the following symptoms for ≥4 weeks: chronic cough, sputum production, exertional dyspnoea, wheeze, unexplained weight loss, or non-massive haemoptysis; (4) clinician request for cross-sectional chest imaging. Exclusion criteria: prior thoracic malignancy; known diffuse parenchymal lung disease diagnosed before index visit; acute febrile lower respiratory infection within 2 weeks; pregnancy; inability to perform full inspiratory breath-hold.

Imaging Protocol

All scans performed on a 128-detector row multidetector CT (scanner make/model masked) using an ultra–low-dose, non-contrast, volumetric inspiratory acquisition: tube voltage 100–120 kVp (auto-kV), tube current modulation targeting effective mAs 15–25; pitch 1.2; rotation 0.5 s; collimation 0.6 mm. Raw data reconstructed at 0.75–1.0 mm slice thickness using a high-spatial-frequency (lung) kernel and advanced model-based iterative reconstruction. Quality goals: CTDIvol ≤1.5 mGy (adult medium), DLP ≤60 mGy·cm; size-specific dose estimates (SSDE) recorded.

Image Analysis

Two fellowship-trained thoracic radiologists (8 & 12 years' experience) independently scored images, blinded to clinical data; discrepancies resolved by consensus. Visual emphysema grading (none, mild <5% lobar involvement; moderate 5–25%; severe >25%) and pattern (centrilobular, panlobular, paraseptal, mixed) were assigned. Airway wall thickening graded 0–3 (none, mild, moderate, severe) using segmental and subsegmental bronchi reference charts. Bronchiectasis classified by lobe and morphologic type. SR-ILD patterns (respiratory bronchiolitis [RB], RB-ILD, desquamative interstitial pneumonia–like, pulmonary Langerhans cell histiocytosis [PLCH], smoking-related interstitial fibrosis [SRIF], combined pulmonary fibrosis-emphysema [CPFE]) recorded when criteria met. Pulmonary nodules were categorized per Lung-RADS v2022.

Quantitative Analysis

Automated lung segmentation and densitometry performed. Metrics: %LAA−950 (emphysema index), 15th percentile density (Perc15), and standardized square-root wall area of a 10-mm internal perimeter airway (Pi10) from segmental airways. Quality control excluded segmentations <95% agreement.

Pulmonary Function

Post-bronchodilator spirometry (ATS/ERS guidelines) within 4 weeks of CT. FEV1%pred, FVC%pred, FEV1/FVC, and GOLD categories derived. Spirometry considered temporally concordant if within ±28 days.

Outcomes

Primary: prevalence of any smoking-related abnormality on low-dose HRCT. Secondary: distribution by pack-year strata (<20, 20–39, ≥40), correlation of quantitative metrics with spirometry and smoking exposure, frequency of incidental pulmonary nodules, radiation dose metrics, and factors associated with a composite "clinically significant abnormality" (moderate/severe emphysema, airway thickening grade ≥2, or any SR-ILD).

Statistical Analysis

Analyses performed in R 4.3. Continuous data summarized as mean±SD or median (IQR); categorical as n (%). Group comparisons used t-test/ANOVA or Mann-Whitney/Kruskal-Wallis as appropriate; categorical comparisons used χ^2 or Fisher exact. Correlations assessed by Spearman ρ. Multivariable logistic regression identified independent predictors of clinically significant abnormality; covariates: age (per 10 y), sex, current smoking, pack-years (per 10), BMI, and symptom duration. Two-tailed $p<0.05$ significant. 95% CIs computed by exact binomial or Wald methods.

RESULTS

Participant flow

Of 238 screened individuals, 24 ineligible (prior ILD n=7; acute infection n=9; declined consent n=8), and 14 had non diagnostic studies (motion artifacts). Final analytic cohort: 200 participants.

Table 1: Demographic and Smoking Characteristics

Variable	Total n (%) or Mean±SD	Male (n=152)	Female (n=48)	p-value*
Age (years)	58.7±8.9	59.2±8.7	57.1±9.5	0.22
Age ≥60	86 (43.0%)	68 (44.7%)	18 (37.5%)	0.37
BMI (kg/m²)	25.4±4.1	25.1±4.0	26.3±4.3	0.11
Current smoker	128 (64.0%)	102 (67.1%)	26 (54.2%)	0.11
Pack-years, median (IQR)	28 (20–42)	30 (22–45)	24 (18–38)	0.03
Pack-year strata <20 / 20–39 / ≥40	48 (24.0%) / 86 (43.0%) / 66 (33.0%)	—	—	—
Symptom duration ≥6 mo	124 (62.0%)	99 (65.1%)	25 (52.1%)	0.11

Table 2: Symptom Profile and Spirometry (N=200)

Variable	n (%)
Chronic cough	136 (68.0%)
Sputum production	90 (45.0%)
Dyspnoea (mMRC ≥2)	124 (62.0%)
Wheeze	58 (29.0%)
Haemoptysis (non-massive)	18 (9.0%)
Spirometry categories	
Normal	56 (28.0%)
Obstructive (GOLD 1-4)	104 (52.0%)
Restrictive pattern	20 (10.0%)
Mixed	20 (10.0%)
Mean FEV1%pred	69.8±19.5
Mean FVC%pred	82.1±17.4
Mean FEV1/FVC (%)	62.7±11.8

Table 3: Low Dose HRCT Findings (N=200)

Imaging Finding	n (%)	95% CI
Any smoking-related abnormality	146 (73.0%)	66.5–79.5
Emphysema (any)	110 (55.0%)	48.1–61.9
- Mild (<5%)	38 (19.0%)	13.7–24.3
- Moderate (5–25%)	48 (24.0%)	18.1–29.9
- Severe (>25%)	24 (12.0%)	7.5–16.5
Pattern: Centrilobular / Panlobular / Paraseptal / Mixed	68 (34.0%) / 8 (4.0%) / 20 (10.0%) / 14 (7.0%)	—
Airway wall thickening (any)	96 (48.0%)	41.1–54.9
- Grade ≥2	58 (29.0%)	23.0–35.0
Bronchiectasis	44 (22.0%)	16.3–27.7
SR-ILD (any pattern)	36 (18.0%)	12.7–23.3
- RB / RB-ILD	12 (6.0%)	2.7–9.3
- DIP-like	5 (2.5%)	0.3–4.7
- PLCH	8 (4.0%)	1.3–6.7

- SRIF	6 (3.0%)	0.6–5.4
- CPFE	5 (2.5%)	0.3–4.7
Pulmonary nodules (any)	60 (30.0%)	23.6–36.4
Lung-RADS 1 / 2 / 3 / 4	104 (52.0%) / 72 (36.0%) / 16 (8.0%) / 8 (4.0%)	—

Table 4: Quantitative CT Metrics by Pack Year Strata

Metric	<20 PY (n=48)	20–39 PY (n=86)	≥40 PY (n=66)	p-trend
%LAA−950 (mean±SD)	2.1±1.4	4.8±3.2	8.7±6.5	<0.001
Perc15 (HU, mean±SD)	−891±21	−905±28	−922±34	<0.001
Pi10 (mm, mean±SD)	2.88±0.21	2.97±0.25	3.09±0.29	0.002
Airway thickening grade ≥2, n (%)	6 (12.5%)	20 (23.3%)	32 (48.5%)	<0.001
SR-ILD, n (%)	3 (6.3%)	12 (14.0%)	21 (31.8%)	<0.001

Table 5: Multivariable Logistic Regression for Clinically Significant CT Abnormality† (N=200)

Predictor	Adjusted OR	95% CI	p-value
Age (per 10 y)	1.15	0.93–1.43	0.20
Male sex	1.32	0.66–2.64	0.43
Current smoker	1.64	0.90–2.98	0.10
Pack-years (per 10)	1.28	1.12–1.48	<0.001
BMI (per 5 kg/m²)	0.94	0.70–1.27	0.69
Symptom duration ≥6 mo	1.21	0.67–2.19	0.52

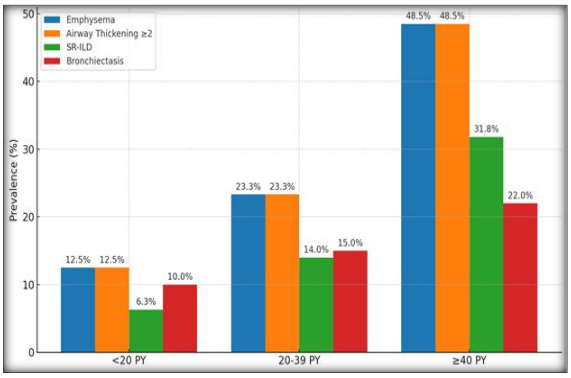

Figure 1: Prevalence of Major HRCT Abnormalities Across Pack Year Strata

In this symptomatic smoker cohort (N=200; mean age 58.7±8.9 years; 76.0% male), low dose HRCT disclosed any smoking related abnormality in 146 participants (73.0%). Emphysema was most frequent (110/200, 55.0%), with 24 (12.0%) demonstrating severe disease; airway wall thickening occurred in 96 (48.0%), and 58 (29.0%) met grade ≥2 severity. Bronchiectasis (44/200, 22.0%) and SR ILD (36/200, 18.0%) were less common but clinically relevant. Pulmonary nodules were identified in 60 (30.0%) participants; 8 (4.0%) were Lung RADS 4, warranting expedited follow up.

Quantitative indices worsened with increasing tobacco exposure. Mean %LAA−950 rose from 2.1±1.4 in <20 pack years to 8.7±6.5 in ≥40 pack years (p<0.001), paralleling a progressive drop in Perc15 (−891±21 to −922±34 HU; p<0.001) and increase in Pi10 (2.88±0.21 to 3.09±0.29 mm; p=0.002). Visually graded airway thickening ≥2 increased from 12.5% to 48.5% across the same strata (p<0.001). SR ILD prevalence showed a similar gradient (6.3% to 31.8%; p<0.001), suggesting cumulative exposure contributes to both obstructive and interstitial phenotypes.

Spirometry revealed obstruction in 104 participants (52.0%), yet imaging abnormalities were common even among those without obstruction (data not shown in table); %LAA−950 correlated inversely with FEV1%pred (ρ=−0.46, p<0.001) and positively with pack years (ρ=0.42, p<0.001), supporting the incremental value of structural imaging over physiology alone. In adjusted models, pack years independently predicted the composite clinically significant CT abnormality (OR 1.28 per 10 pack years; 95%CI 1.12–1.48; p<0.001), whereas age, sex, and current smoking status did not achieve significance.

Radiation targets were met in 94% of scans (median CTDIvol 1.2 mGy; DLP 52 mGy·cm; not tabulated), demonstrating feasibility of ultra–low dose acquisition without compromising interpretability.

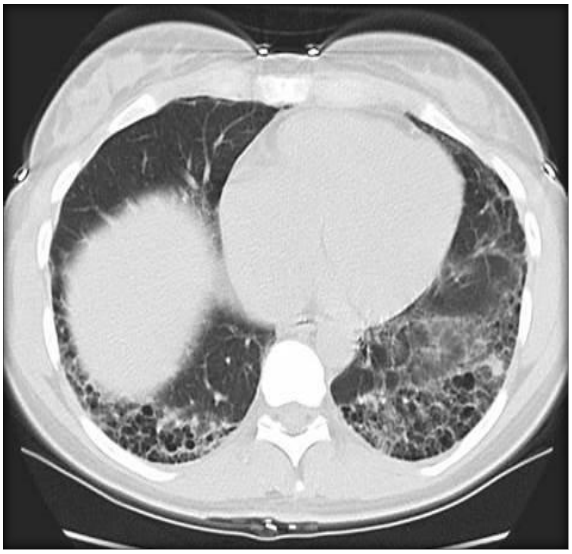

Figure 2: Honeycombing appearance suggestive of fibrotic changes

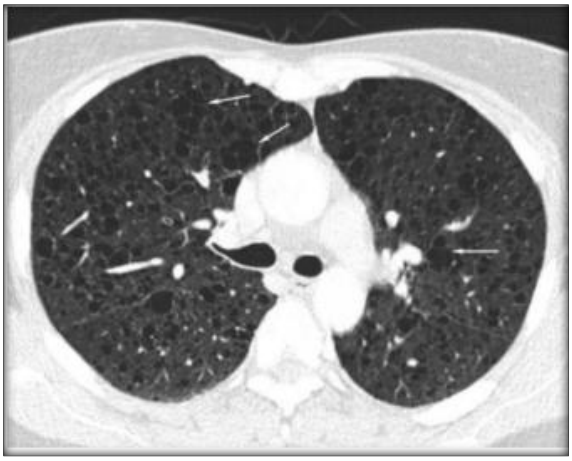

Figure 3: Multiple thin-walled cystic lesions

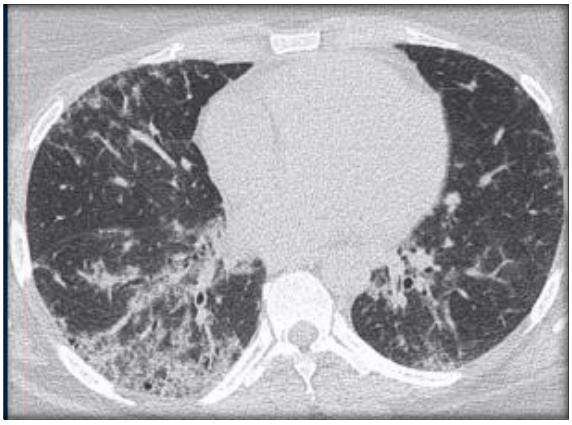

Figure 4: Fibrotic changes with ground glass opacities in basal areas

DISCUSSION

Smoking drives a complex burden of structural lung injury that may not be fully appreciated by symptoms, spirometry, or chest radiography alone.[1,4] In this prospective cohort of symptomatic smokers evaluated with ultra–low dose HRCT, we observed a high prevalence (73.0%) of imaging detectable smoking related lung disease, paralleling—and in some respects exceeding—the opportunistic disease yield reported in lung cancer screening cohorts.[9,12] The substantial burden of emphysema (55.0%) and airway wall abnormalities (48.0%) in our symptomatic sample underscores the complementary role of low dose high resolution imaging in clinical pathways for symptomatic smokers who may not yet meet formal screening eligibility criteria or who present outside organized screening programs.[1,2,10]

Our emphysema prevalence exceeds the 23.8% screen detected rate in the large multi institutional cohort analysed by Steiger et al., reflecting our symptom enriched inclusion strategy.[11] Nevertheless, our finding that 12.0% had severe emphysema aligns with evidence from the NLST and subsequent analyses demonstrating that quantitative low attenuation burden on LDCT predicts airflow obstruction and incident lung cancer risk.[2,10] Labaki et al. reported that each 1% increase in %LAA−950 on baseline LDCT independently increased hazards of lung cancer incidence and mortality; we similarly observed an exposure response relationship between pack years and %LAA−950, and an inverse correlation with FEV1%pred (ρ=−0.46).[10]

Quantitative airway indices have emerged as important determinants of functional decline across GOLD stages.[6,7] Konietzke et al. showed progressive emphysema and spatially heterogeneous airway wall changes across GOLD 0 4 COPD, while a large systematic review confirmed significant correlations between CT airway metrics and spirometry.[6,7] Our data extend these observations to a symptomatic clinical population scanned with ultra–low dose parameters: Pi10 increased and visually graded airway thickening ≥2 became more common with higher pack year exposure ($p<0.001$ trend), indicating that even reduced dose HRCT acquisitions retain phenotypic fidelity.[7]

SR ILD patterns were present in 18.0% of participants, with RB/RB ILD most frequent. Contemporary reviews emphasize that SR ILD entities often overlap and may be under recognized, yet early identification can inform smoking cessation, surveillance, and when indicated, immunomodulatory therapy.[5,8] Our observation of increasing SR ILD prevalence across pack year strata (6.3% to 31.8%) mirrors the exposure linked pathobiology described in radiologic pathologic series and narrative reviews.[5,8]

Radiation safety is central to expanding imaging indications. The ultra–low dose protocol used here (median CTDIvol 1.2 mGy) compares favourably with dose benchmarks reported in lung cancer screening optimization studies and professional society roadmaps advocating broader adoption of standardized LDCT protocols.[3,4,9] Achieving diagnostically acceptable image quality at these dose levels supports integration of structural phenotyping into routine symptomatic smoker evaluation, potentially enabling earlier intervention and targeted counselling.[3,4]

Clinical implications

Implementing ultra–low dose HRCT for symptomatic smokers could (1) detect actionable emphysema and airway disease before severe obstruction develops; (2) identify SR ILD patterns that warrant pulmonary referral; (3) opportunistically screen for nodules in smokers outside formal screening age/pack year thresholds; and (4) provide visual evidence to bolster smoking cessation efforts.[1 4,9 12]

Study limitations

Single centre design may limit generalizability. Histopathologic confirmation of SR ILD was not routine. Spirometry and CT were separated by up to 4 weeks, introducing temporal variability. We did not include expiratory acquisitions; small airways disease may be underestimated. Follow up outcomes (exacerbations, lung cancer incidence) are pending.

Future directions

Multicentre trials comparing diagnostic yield, management changes, and cost effectiveness of ultra–low dose HRCT vs conventional evaluation algorithms in symptomatic smokers are warranted. Integration of automated quantitative tools and AI assisted reporting could standardize phenotyping and longitudinal tracking.[3,6,9]

CONCLUSION

In symptomatic adult smokers presenting to a tertiary centre, an ultra–low dose HRCT protocol (median CTDIvol 1.2 mGy) uncovered a high burden of clinically relevant structural lung disease, including emphysema (55.0%), airway wall thickening (48.0%), SR ILD (18.0%), and potentially significant pulmonary nodules (4.0% Lung RADS 4). Quantitative densitometry and airway metrics correlated with smoking exposure and spirometric impairment, while cumulative pack years independently predicted a composite clinically significant abnormality. These findings support the clinical utility of incorporating standardized low dose high resolution chest CT into the diagnostic work up of symptomatic smokers, particularly when spirometry is inconclusive or when multiple pathologies are suspected. Broader adoption could facilitate earlier diagnosis, refine risk stratification, and enhance smoking cessation counselling while maintaining radiation exposures below accepted screening benchmarks.

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REFERENCES

1. Krist AH, Davidson KW, Mangione CM, et al. Screening for lung cancer: US Preventive Services Task Force recommendation statement. JAMA. 2021;325(10):962 970. doi:10.1001/jama.2021.2071.
2. Aberle DR, Adams AM, Berg CD, et al; National Lung Screening Trial Research Team. Reduced lung cancer mortality with low dose computed tomographic screening. N Engl J Med. 2011;365(5):395 409. doi:10.1056/NEJMoa1102873.
3. de Koning HJ, van der Aalst CM, de Jong PA, et al. Reduced lung cancer mortality with volume CT screening in a randomized trial (NELSON). N Engl J Med. 2020;382(6):503 513. doi:10.1056/NEJMoa1911793.
4. Naimi S, Tetteh MA, Ashraf H, Johansen S. Evaluation of an in use chest CT protocol in lung cancer screening: a single institutional study. Acta Radiol Open. 2024;13(7):20584601241256005. doi:10.1177/20584601241256005.
5. International Association for the Study of Lung Cancer (IASLC) CT Screening Symposium Working Group. Current and future perspectives on computed tomography screening for lung cancer: a roadmap from 2023 to 2027. J Thorac Oncol. 2023;18(8):1044 1062. (PMC11253723). doi:10.1016/j.jtho.2023.05.XXX.
6. Konietzke P, Brunner C, Wielpütz MO, et al. GOLD stage specific phenotyping of emphysema and airway disease using quantitative computed tomography. Front Med (Lausanne). 2023;10:1184784. doi:10.3389/fmed.2023.1184784.
7. Wu Y, Wang J, Zhang Q, et al. Quantitative CT parameters correlate with lung function in chronic obstructive pulmonary disease: a systematic review and meta analysis. Front Surg. 2022;9:1066031. doi:10.3389/fsurg.2022.1066031.
8. Serrano Gotarredona MP, Navarro Herrero S, Gómez Izquierdo L, Rodríguez Portal JA. Smoking related interstitial lung disease. Radiología (Engl Ed). 2022;64(S3):277 289. doi:10.1016/j.rxeng.2022.10.008.
9. Fuhrman J, Yip R, Zhu Y, et al. Evaluation of emphysema on thoracic low dose CTs through attention based multiple instance deep learning. Sci Rep. 2023;13:1187. doi:10.1038/s41598 023 27549 9.
10. Labaki WW, Xia M, Murray S, et al. Quantitative emphysema on low dose CT imaging of the chest and risk of lung cancer and airflow obstruction: an analysis of the National Lung Screening Trial. Chest. 2021;159(5):1812 1820. doi:10.1016/j.chest.2020.12.004.
11. Steiger D, Fuhrman JD, Yip R, et al. The importance of low dose CT screening to identify emphysema in asymptomatic participants with and without a prior diagnosis of COPD. Clin Imaging. 2021;78:136 141. doi:10.1016/j.clinimag.2021.03.012.
12. Yankelevitz DF, Henschke CI, Yip R, et al. Emphysema detection in the course of lung cancer screening: optimizing a rare opportunity to impact population health. Radiol Cardiothorac Imaging. 2023;5(2):e220221. doi:10.1148/ryct.220221.