ILAs: State of the Art

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Critical Literature

Interstitial lung abnormalities detected incidentally on CT: a Position Paper from the Fleischner Society

Lancet Respir Med 2020; 8: 726–37

Hiroto Hatabu, Gary M Hunninghake, Luca Richeldi, Kevin K Brown, Athol U Wells, Martine Remy-Jardin, Johny Verschakelen, Andrew G Nicholson, Mary B Beasley, David C Christiani, Raúl San José Estépar, Joon Beom Seo, Takeshi Johkoh, Nicola Sverzellati, Christopher J Ryerson, R Graham Barr, Jin Mo Goo, John H M Austin, Charles A Powell, Kyung Soo Lee, Yoshikazu Inoue, David A Lynch†*

Interstitial Lung Abnormalities: State of the Art

Radiology 2021; 301:19–34

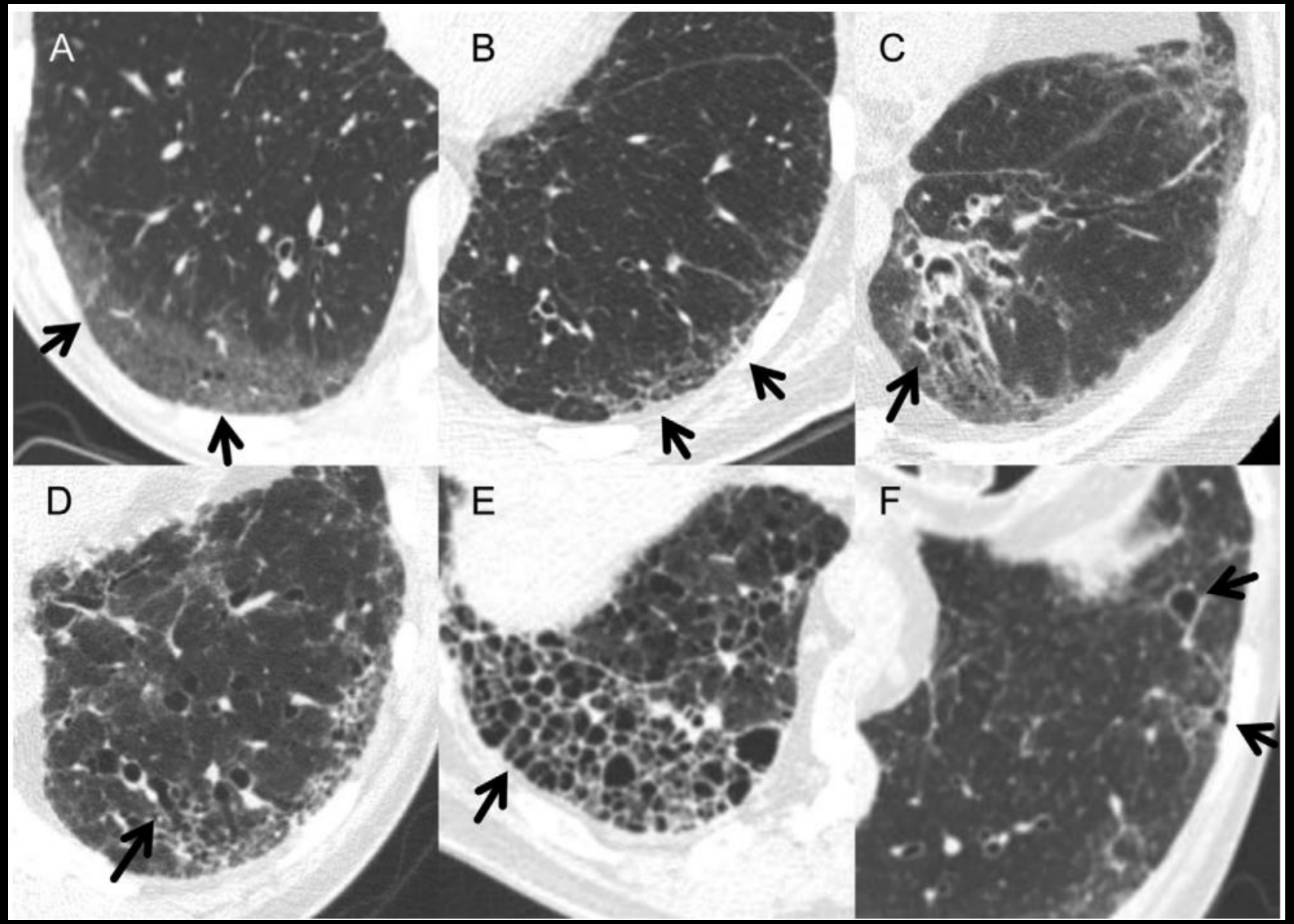
Akinori Hata, MD, PhD • Mark L. Schiebler, MD • David A. Lynch, MB, BCh • Hiroto Hatabu, MD, PhD

What is an ILA?

Incidental CT findings of

- Ground glass,
- Reticulation,
- Architectural distortion,
- Traction bronchiectasis,
- Honeycombing, and/or
- Nonemphysematous cysts

Radiology 2021; 301:19–34



What is *not* an ILA?

Same CT findings, but there is clinical reason to suspect ILD…

regardless of symptoms.

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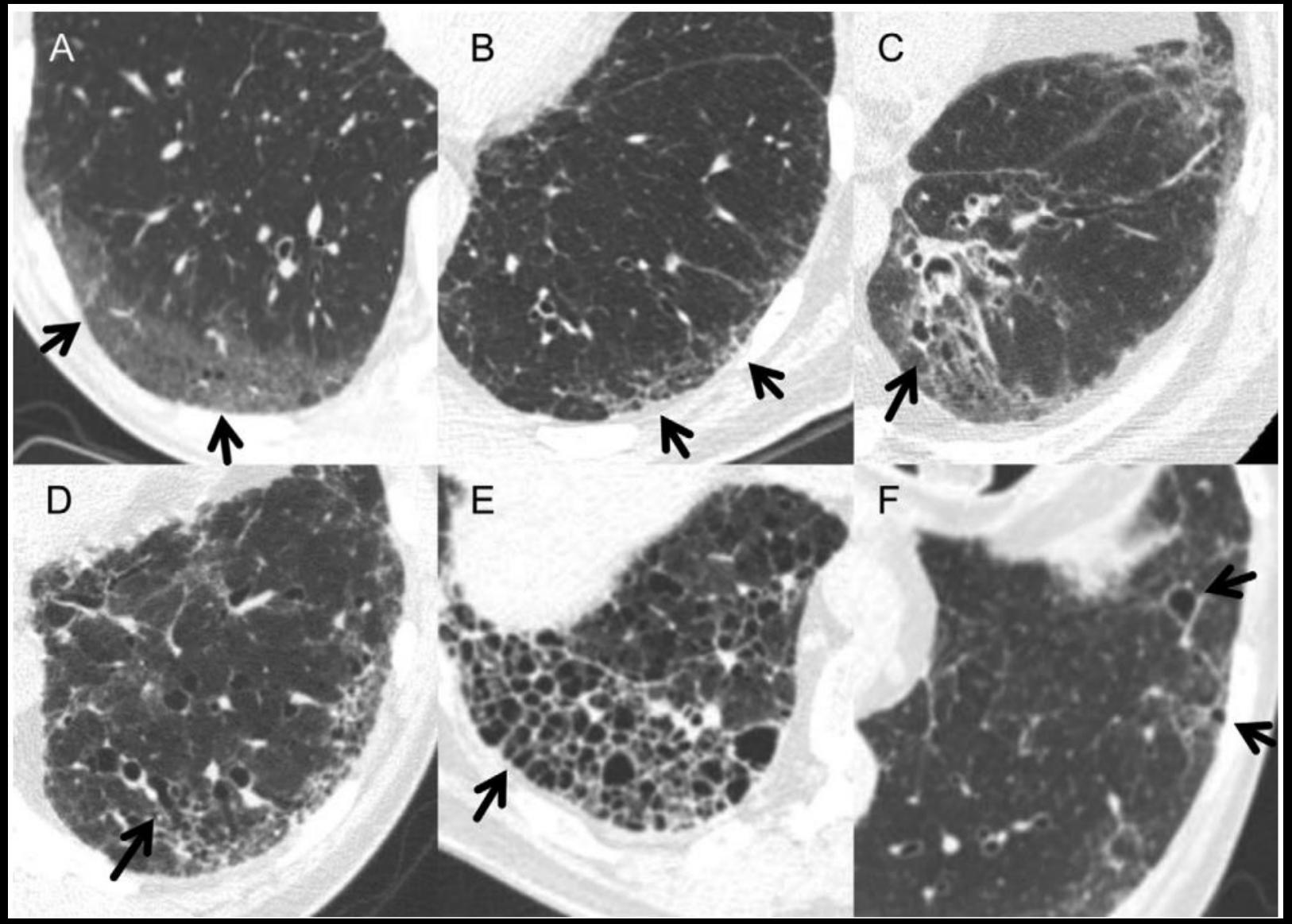

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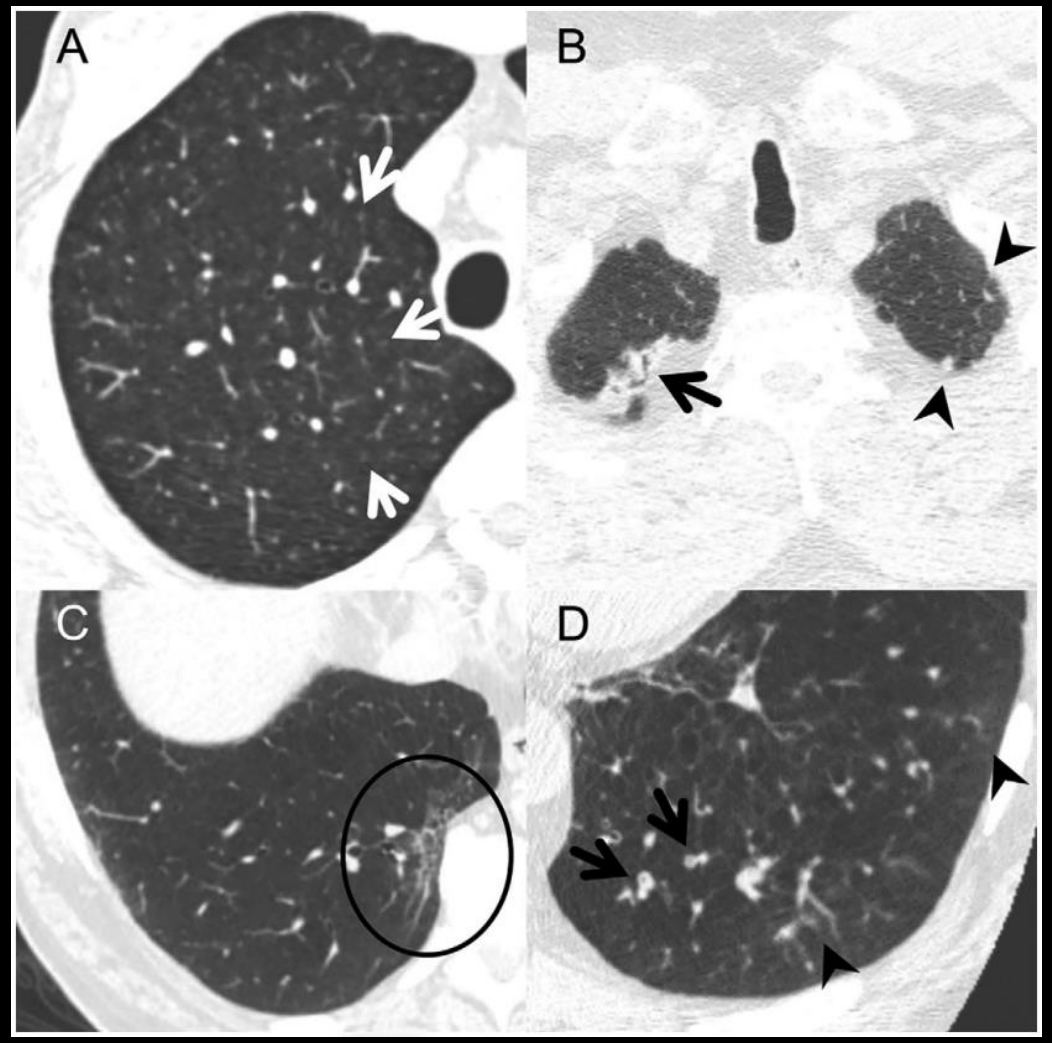

- Centrilobular nodules
- Paraspinal or apical fibrosis
- Sequelae of infection or aspiration
- Trace or unilateral findings
- Dependent atelectasis

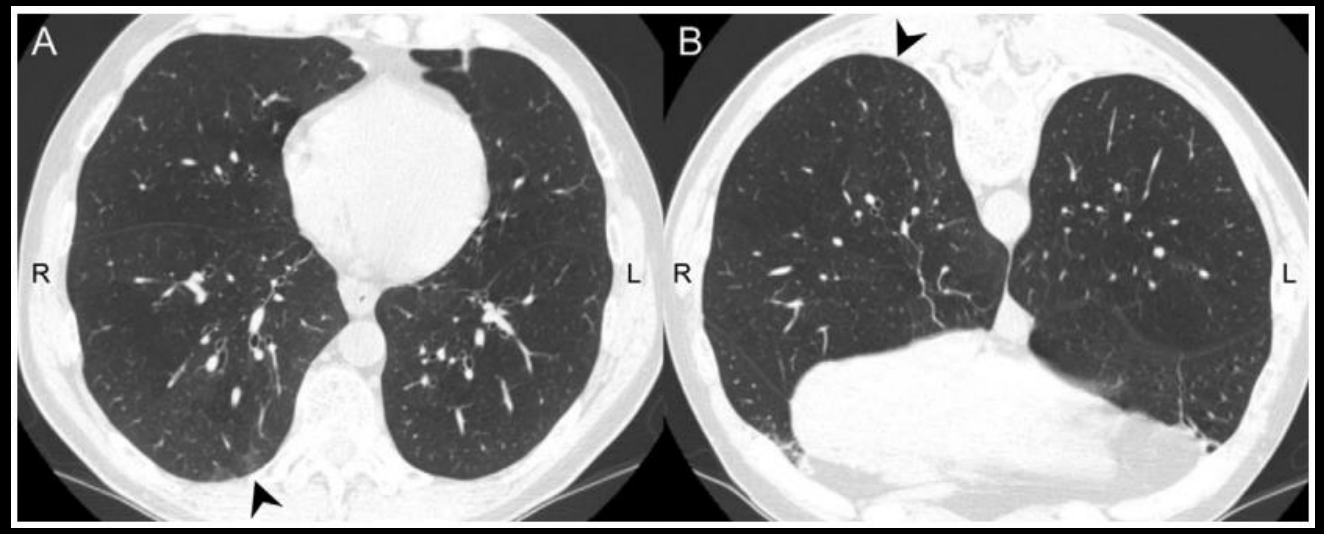

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Table 3: Incidentally Detected CT Findings and Entities That May Be Distinguished from ILA, including Non-ILA Findings

CT Finding or Disease	Non-ILA or Overlap*	Feature or Method Used to Determine ILA	Cause
Basilar linear opacities	Non-ILA	Lack of other findings of fibrosis, often resolve at prone imaging	Atelectasis or scarring
Interlobular septal thickening because of interstitial edema	Non-ILA	Clinical evaluation	Edema in lymphatics surrounding secondary lobule (eg, heart failure, volume overload, or pulmonary veno-occlusive disease)
Osteophyte-related fibrosis	Non-ILA	Close contact with osteophytes, usually craniocaudally aligned	Mechanical irritation from osteophytes
Aspiration	Non-ILA	Gravity-dependent distribution, patchy ground-glass and tree-in-bud opacities, plugged central airways	Dysphagia, gastroesophageal reflux
Post-infection reticulation	Overlap	Usually focal or multifocal and often linear	Organizing pneumonia
Scarring	Overlap	Usually linear or band-like	Healing of previous trauma or infection
UIP or IPF	Overlap	Extensive abnormalities and clinical findings(ILA may sometimes be an early phase of UIP or IPF)	Cause is unknown, and genetic predisposition, familial factor, and short-telomere syndrome may be present
Desquamative interstitial pneumonia	Overlap	Smoking history, homogenous ground-glass abnormality, often with cysts	Macrophage-induced inflammation with fibrosis
Respiratory bronchiolitis	Non-ILA	Centrilobular nodularity, smoking history	Macrophage-induced inflammation with fibrosis
PPFE	Non-ILA	Dense apical subpleural fibrotic abnormality	Unknown; associated with post–bone marrow transplant, lung transplant, autoimmune or connective tissue disease, or acute lung injury
CTD-ILD	Overlap	Clinical evidence of CTD	Autoimmune disorder
Sarcoidosis	Overlap	Perilymphatic nodularity, lymphadenopathy	Immune-mediated multisystem disorders with granulomas
PLCH	Overlap	Nodules with cavitation, cysts	Formation of Langerhans cell nodules related to cigarette smoking
Occupational dust inhalation (eg, asbestosis, silicosis, and aluminosis)	Overlap	Worker history of exposure to dusts, pleural plaques	Immune response to dusts and accumulation in macrophages with occasional granulomatous response
Fibrotic hypersensitivity pneumonitis	Overlap	Mosaic attenuation, air trapping, three-density pattern (eg, ground-glass opacity, lobules of decreased attenuation and vascularity, and normal-appearing lung)	Exposure to antigens eliciting an immune response (eg, farmer's lung and bird fancier's lung)
Drug-related pneumonitis	Overlap	History of medication (eg, bleomycin, methotrexate, or amiodarone)	Drug-induced lung injury

Note.—CTD = connective tissue disease, ILA = interstitial lung abnormality, ILD = interstitial lung disease, IPF = idiopathic pulmonary fibrosis, PLCH = pulmonary Langerhans cell histiocytosis, PPFE = pleuroparenchymal fibroelastosis, UIP = usual interstitial pneumonia.

* "Overlap" means that CT findings of the disease are possibly similar to those of ILA, although the disease may be differentiated from ILA according to clinical background and symptoms.

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What is interstitial lung abnormality (ILA)?

"fixed"

- Incidental identification of ~~non-dependent~~ abnormalities, including ground-glass or reticular abnormalities, lung distortion, traction bronchiectasis, honeycombing, and non-emphysematous cysts

- Involving at least 5% of a lung zone (upper, middle, and lower lung zones are demarcated by the levels of the inferior aortic arch and right inferior pulmonary vein)

- In individuals in whom interstitial lung disease is not suspected

Subcategories of ILA

Non-subpleural
ILA without predominant subpleural localization

Subpleural non-fibrotic
ILA with a predominant subpleural localization and without evidence of pulmonary fibrosis*

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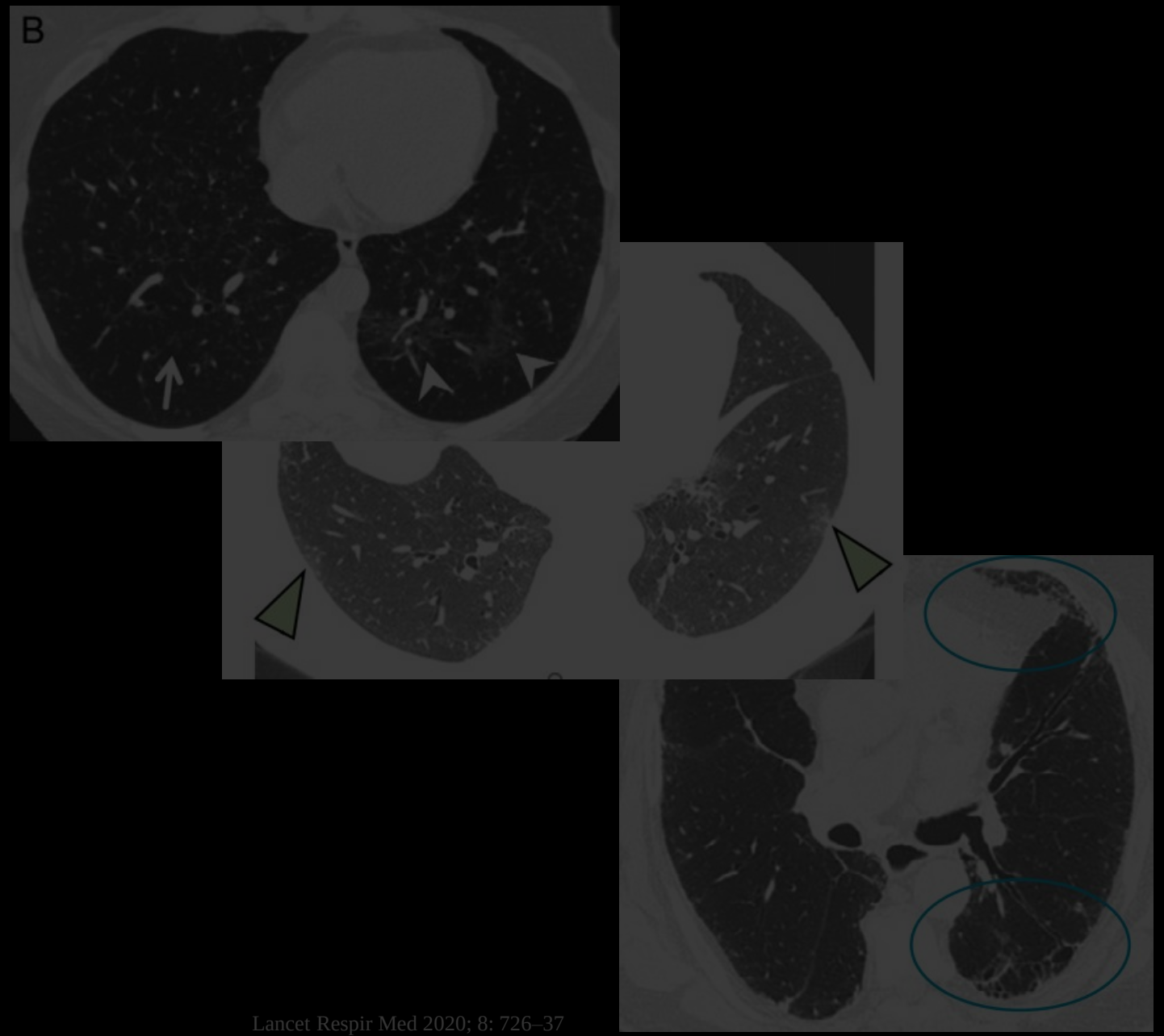

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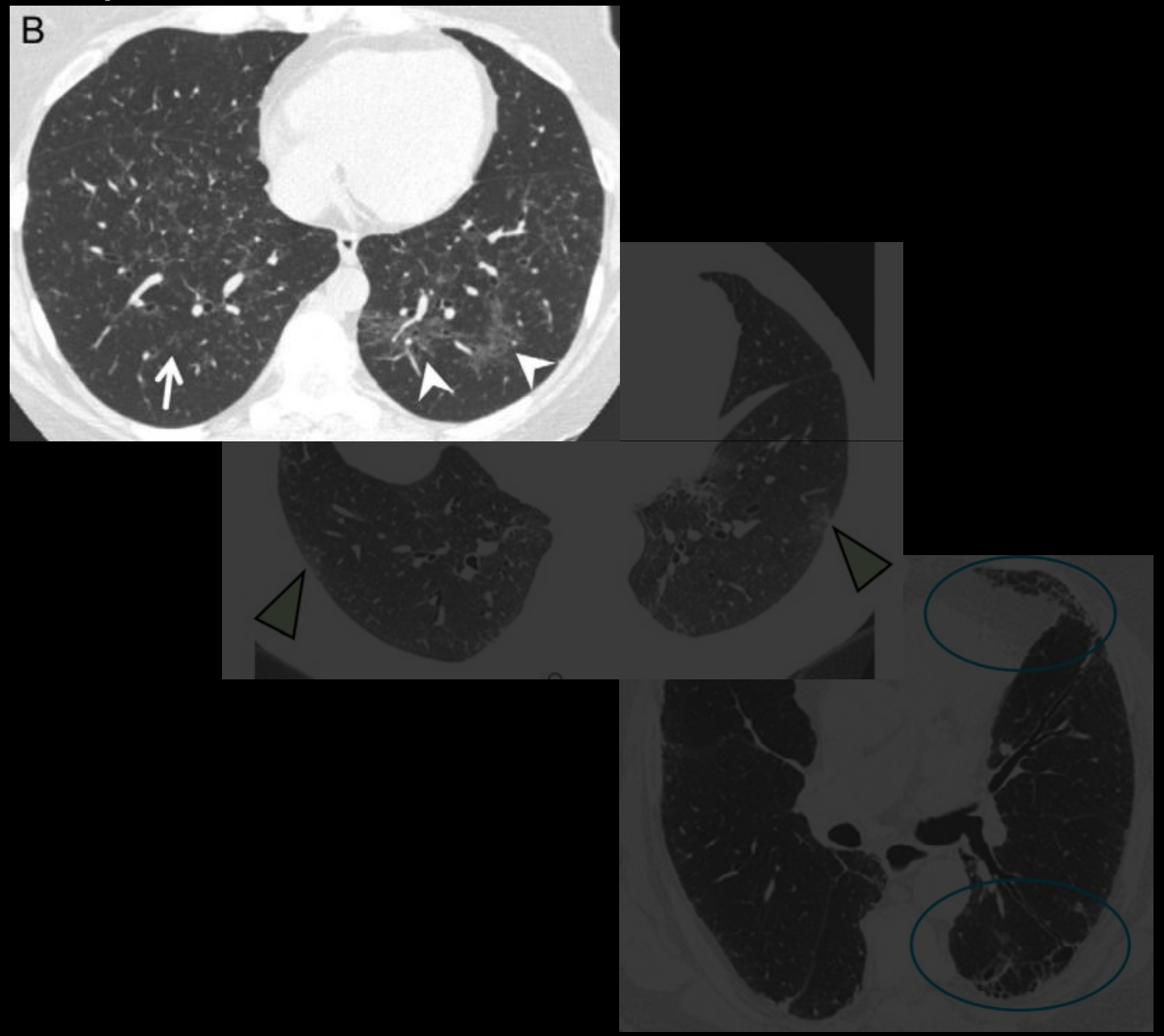

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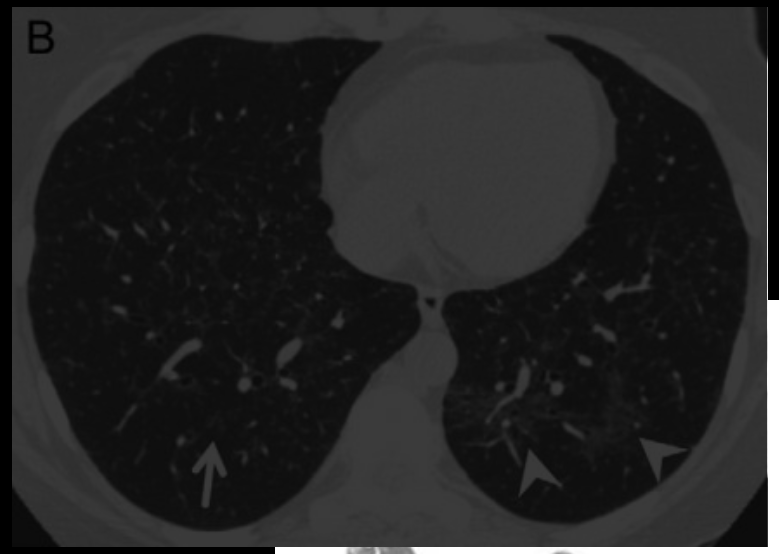

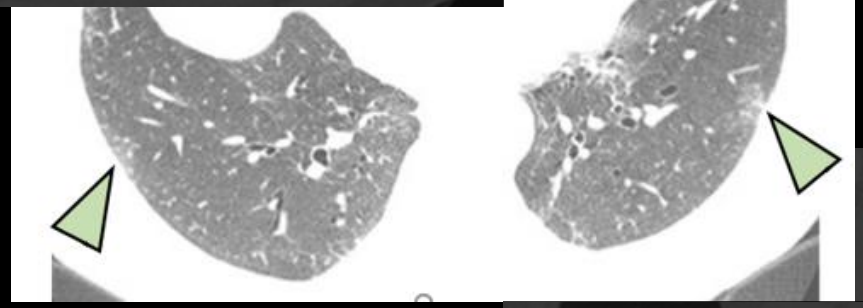

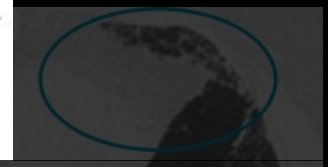

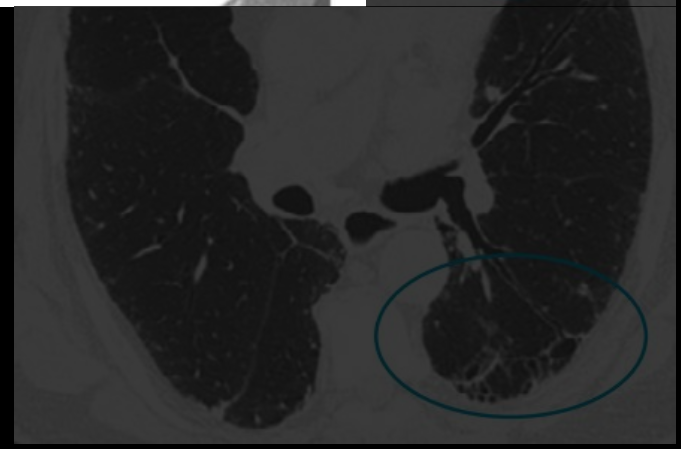

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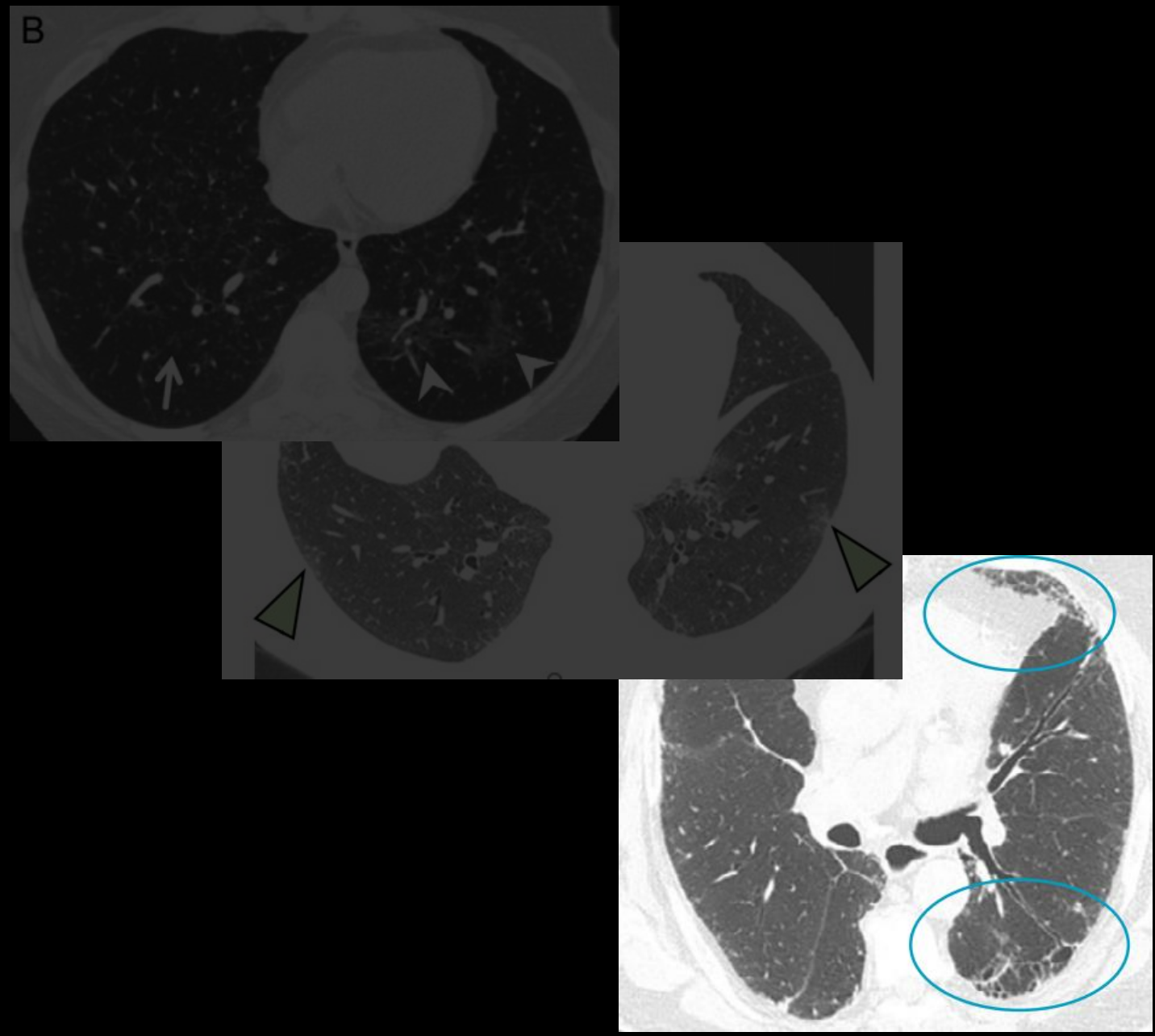
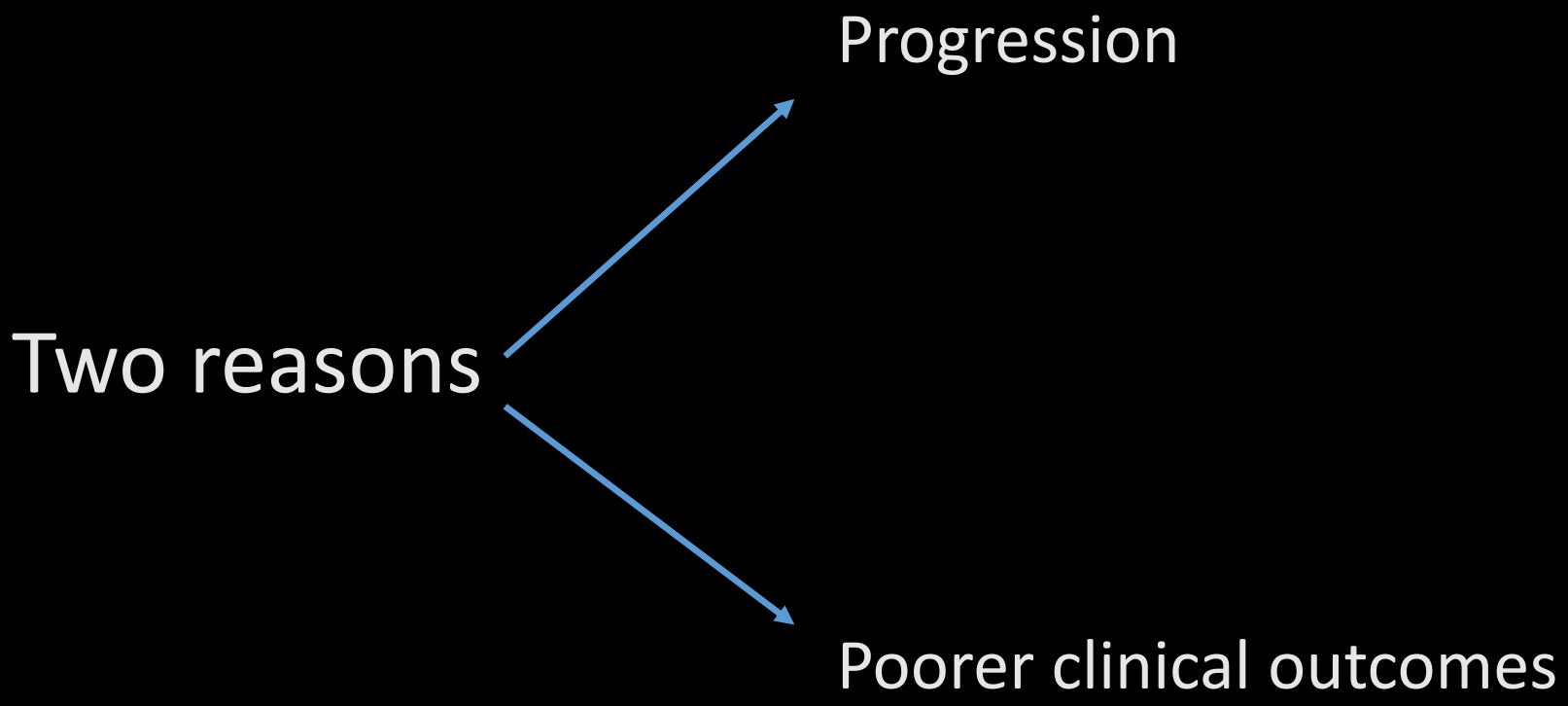


Why do ILAs matter?



	Population-based cohorts				Smoking and lung cancer screening cohorts				
	MESA[11,12,13,14]	Nagano, Japan*[15]	FHS[6,8,9]	AGES-Reykjavik[9]	ECLIPSE[9]	NLST[7,16]	COPDGene[4,9,17]	MILD[18]	DLCST[19]
Study characteristics									
Total number of chest CT scans evaluated	3137	3061	2633	5320	1670	884	9292	692	1990
Prevalence of ILAs	310 (10%)	80 (3%)	177 (7%)	377 (7%)	157 (9%)	86 (10%)	708 (8%)	28 (4%)	332 (17%)
Mean age of those with ILAs (years)	75	62	70	78	64	62	64	60	60
Radiological progression									
Overall progression, follow-up time	NA	46%, 4 years	43%, 6 years	63%, 5 years	NA	20%, 2 years	NA	20%, 2 years	NA

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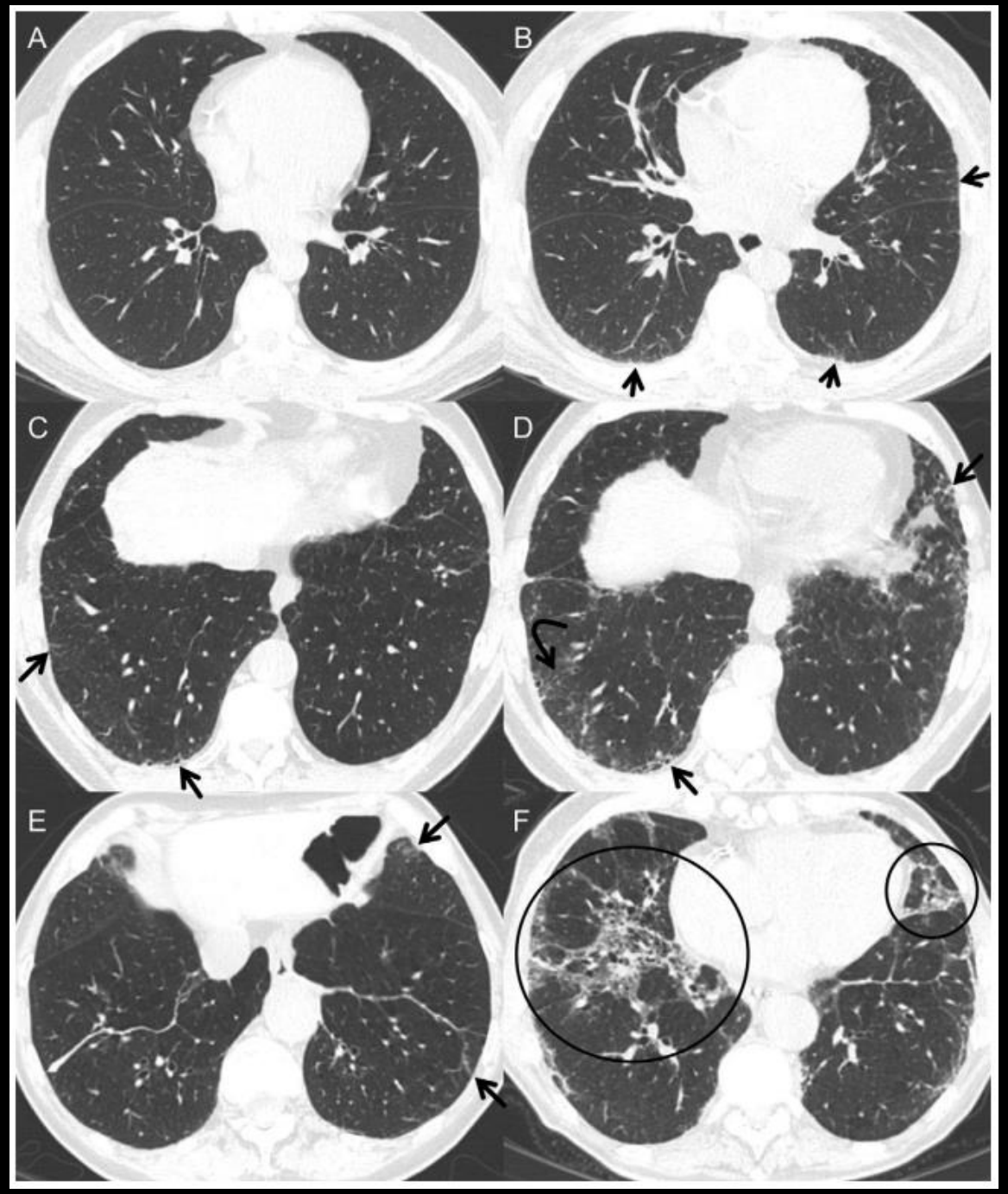
ILAs progress



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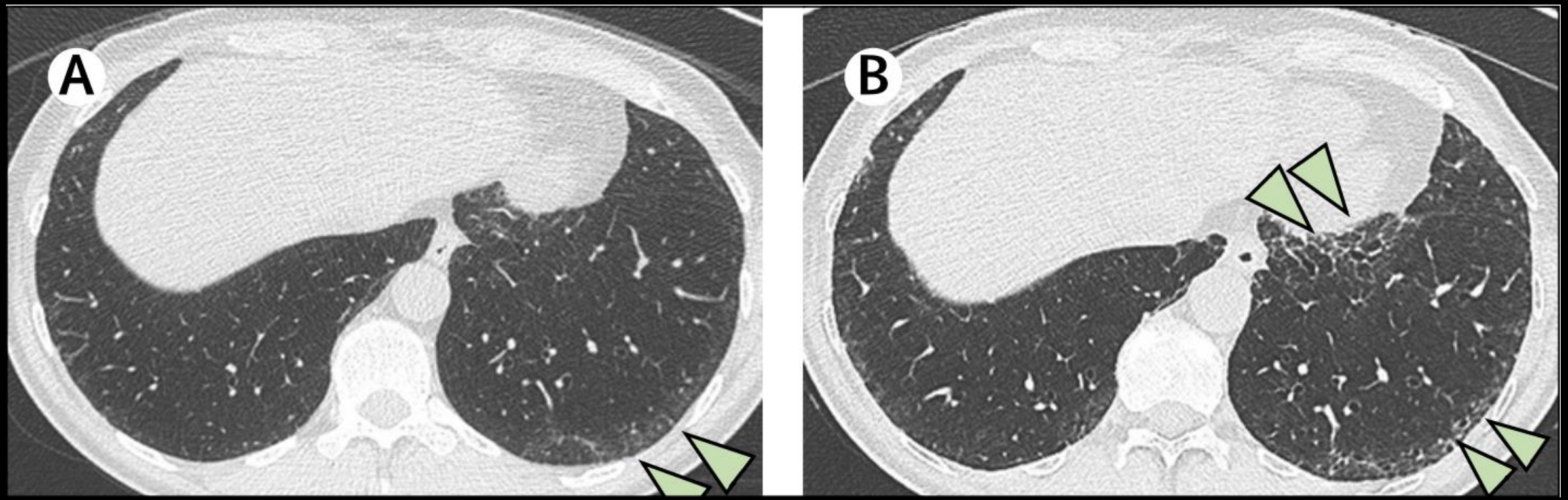

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Table 4: Risk Factors for ILA Progression

Risk Factor Type	Risk Factor
Clinical	Cigarette smoking; other inhalational exposures; medications (eg, chemotherapy or immune checkpoint inhibitors); radiation therapy; thoracic surgery; physiologic or gas-exchange findings at lower limits of normal range
Radiologic	Nonfibrotic ILA with basal and peripheral predominance; fibrotic ILA with basal and peripheral predominance but without honeycombing (ILA with probable UIP pattern); fibrotic ILA with basal and peripheral predominance and honeycombing (ILA with UIP pattern)

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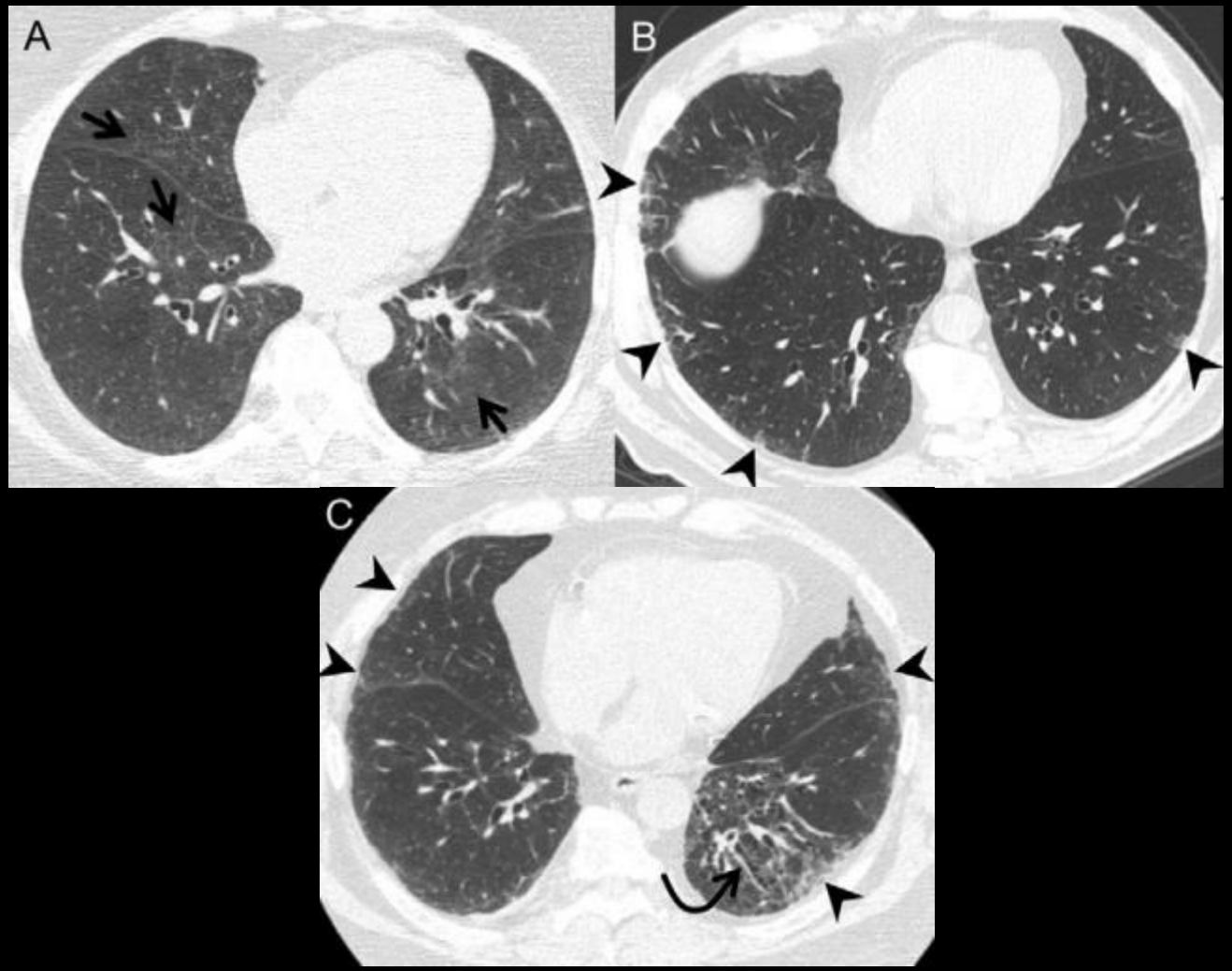

Table 2: Associations between Imaging Features and ILA Progression and between Imaging Features and Mortality in AGES-Reykjavik Study

A: Associations between Imaging Features and ILA Progression

Imaging Feature	Adjusted OR*	*P* Value
~~Centrilobular nodules~~	0.2 (0.1, 0.5)	.0002†
Subpleural reticular markings	6.6 (2.3, 19)	.0004†
Nonemphysematous cysts	2.5 (1.3, 5.1)	.009†
Lower lobe predominant changes	6.7 (1.8, 25)	.004†
Traction bronchiectasis	6.6 (2.3, 19)	.0004†

ILAs are associated with poorer outcomes

- Symptoms
- Lung function
- Lung cancer incidence
- Lung injury risk during cancer therapy
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	ILA patients	Non-ILA patients	P value
chronic cough	12%	6%	.006
SOB	18%	9%	< .001
↓ exercise capacity	-19 m	even	.008

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- ↓ TLC by 0.444 L ($P < .001$)
- ↓ DLCO 86% (vs 98%; $P < 0.001$)
- Some studies report differences in FEV1 and FEV1/FVC

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- AGES-Reykjavik Hazard ratio = 2.77 ($P < .0001$)
- NLST Incidence rate ratio = 1.33

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- ↑ Risk of postoperative pulmonary compliations in early stage lung cancer

  - Pneumonia
  - ARDS
  - Respiratory failure
  - BPF
  - Empyema
  - Prolonged air leak
  - Pneumothorax

  OR = 1.91; P = .004

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- ↑ Risk of grade 3 or higher radiation pneumonitis
  - 36% vs. 9%; P = .005
- ↑ Risk of extensive radiation pneumonitis in early-stage lung cancer treated with stereotactic radiation
  - 19% vs. 0%; P = .0035
- ↑ Risk of immunotherapy-associated pneumonitis
  - 43% vs. 10%; P = .007

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Mortality									
Relative risk of death, (hazard ratio [95% CI])	NA	NA	2·7 (1·1–6·5)	1·3 (1·2–1·4)	1·4 (1·1–2·0)	NA	1·8 (1·1–2·8)	NA	2·0 (1·4–2·7)

ILAs=interstitial lung abnormalities. NA=not available. *Patients participating in a health screening programme from Nagano prefecture, Japan.

Table: **Interstitial lung abnormalities across study populations**

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- Hazard ratio for death = 1.5 (P < .0001) for patients with ILA with fibrosis vs. no ILA
- Odds ratio for death from a respiratory cause = 2.4 (P < .001) for ILA vs no ILA
- Hazard ratio for lung cancer mortality ranges from 1.51 to 4.66 across studies for ILA vs no ILA
- Hazard ratio for death from ARDS within 28 days after sepsis or SIRS = 2.3 (P = .01)
- Hazard ratio for mortality during or after TAVR = 3.29 (P = .009)

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- HR of mortality in patients with progression of ILA ranges from 1.4 to 3.9

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B: Associations between Imaging Features and Mortality[‡]

Imaging Feature	Adjusted HR[§]	*P* Value
Reticular markings	1.6 (1.0, 2.5)	.049[†]
Centrilobular nodules	0.9 (0.7, 1.1)	.3
Nonemphysematous cysts	1.4 (1.1, 1.8)	.02[†]
Traction bronchiectasis	1.6 (1.3, 2.1)	.0001[†]
Lower lobe predominant changes	1.1 (0.6, 1.7)	.8
Subpleural location	1.6 (1.0, 2.7)	.05
ILA without fibrosis	1.2 (1.1, 1.3)	.0004[†]
ILA with fibrosis	1.5 (1.3, 1.6)	<.0001[†]
Indeterminate for UIP	1.2 (0.98, 1.5)	.07
Probable UIP pattern	1.9 (1.5, 2.5)	<.0001[†]
UIP pattern	4.5 (2.8, 7.2)	<.0001[†]

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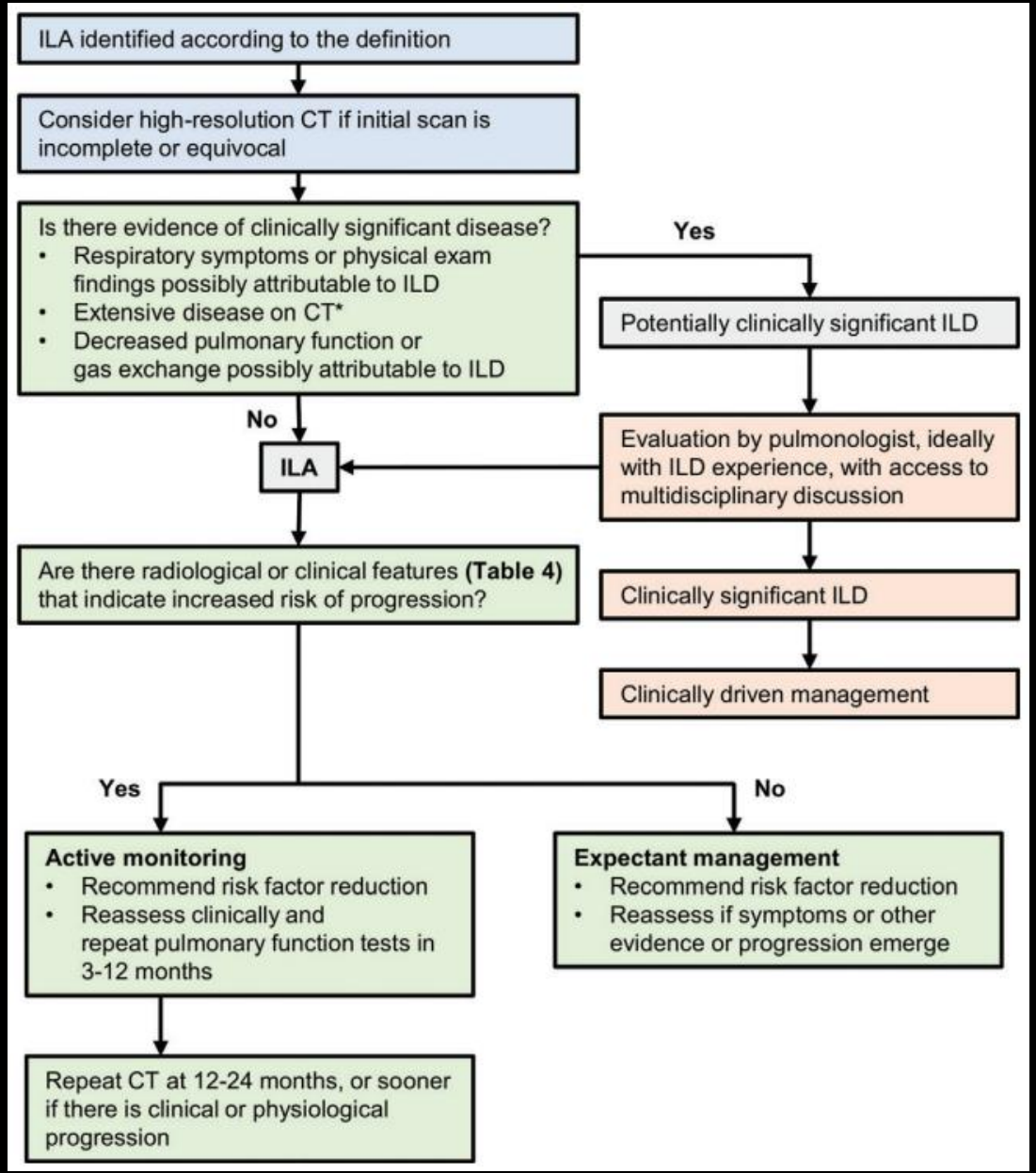

Lancet Respir Med 2020; 8: 726–37

Key messages

Background

- Early interstitial lung abnormalities (ILAs) are common incidental findings on CT, particularly in older individuals
- The presence of ILAs is an independent predictor of mortality
- About 20% of ILAs progress over 2 years, and more than 40% progress over 5 years
- Individuals with subpleural predominant fibrotic ILAs are most likely to progress

Management of ILAs

- Identify potential risk factors for interstitial lung disease
- Identify clinical or functional impairment
- Establish whether there is current evidence of clinically significant interstitial lung disease
- Undertake clinical and imaging follow-up as appropriate

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ILAs: State of the Art

Thank you!

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