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REVIEW ARTICLE

Reporting and management of incidental lung findings on computed tomography: beyond lung nodules

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ABSTRACT

Non-nodular incidental lung findings can broadly be categorised as airway- or airspace-related abnormalities and diffuse parenchymal abnormalities. Airway-related abnormalities include bronchial dilatation and thickening, foci of low attenuation, emphysema, and congenital variants. Diffuse parenchymal abnormalities relate to the spectrum of diffuse parenchymal lung diseases cover a spectrum from interstitial lung abnormalities (ILAs) and pulmonary cysts to established diffuse parenchymal lung abnormalities such as the idiopathic interstitial pneumonias and cystic lung diseases. In this review, we discuss the main manifestations of these incidental findings, paying attention to their prevalence and importance, descriptors to use when reporting, the limits of what can be considered "normal", and conclude each section with some pragmatic reporting recommendations. We also highlight technical and patient factors which can lead to spurious abnormalities.

INTRODUCTION

While pulmonary nodules tend to dominate conversations around incidental findings in the lung parenchyma, there are many non-nodule findings that can, for the most part, incur unnecessary investigation, anxiety or even harm, but may occasionally provide an opportunity for early diagnosis. Deeming a thoracic finding as "incidental" should, on the face of it, be simple enough: the finding should be considered incidental if it is unrelated to the clinical indication, using the definition of 'imaging findings serendipitously diagnosed in an asymptomatic patient or symptomatic patient undergoing imaging for an unrelated reason'.[1] However, as thoracic imaging is increasingly undertaken for more general indications, the definition and context of such "symptoms" becomes increasingly ambiguous, and the reporting radiologist can frequently—and probably reluctantly—be thrust into the role of arbitering whether the findings in question could, on balance of probability, be causing generalised symptoms. For example, bronchiectasis detected on a CT chest, abdomen and pelvis done for weight loss could easily be construed incidental, but just as easily could be leading to repeat infections and immunosuppression in turn causing weight loss, if it is considered sufficiently extensive or severe.

Conversely, certain findings are more common in older, healthy individuals, *e.g.* cysts. Do these findings merit mention, even if considered "incidental", since they are to be expected with age? As postulated by Vikgren et al,[2] a patient living in an urban or polluted area for some time will undeniably be exposed to infectious and noxious agents which will no doubt affect the lungs and airways, however slight. Given the proportion of individuals that will undergo thoracic imaging from this background environment, do we perhaps alter our threshold for what befits an incidental lung finding? These ambiguities around the relevance of an incidental finding are made all the more stark because a clear definition of "normality" in the thorax remains stubbornly elusive.

In this review, we discuss the main categories of these incidental findings, paying attention to their prevalence and importance, descriptors to use when reporting, the limits of what can be considered "normal", and some pragmatic reporting recommendations. In parallel with the current definition of interstitial lung abnormalities (ILAs),[3] we have termed these findings "abnormalities" rather than "diseases", to underscore the fact that their clinical importance rests on clinical and functional corroboration. It goes without saying that this list of incidental findings is not

exhaustive; comprehensive textbooks on thoracic radiology fulfil that function. For a wider pattern-based discussion on the range of normal lung and airway appearances, David Hansell's excellent review on the subject is essential reading.[4]

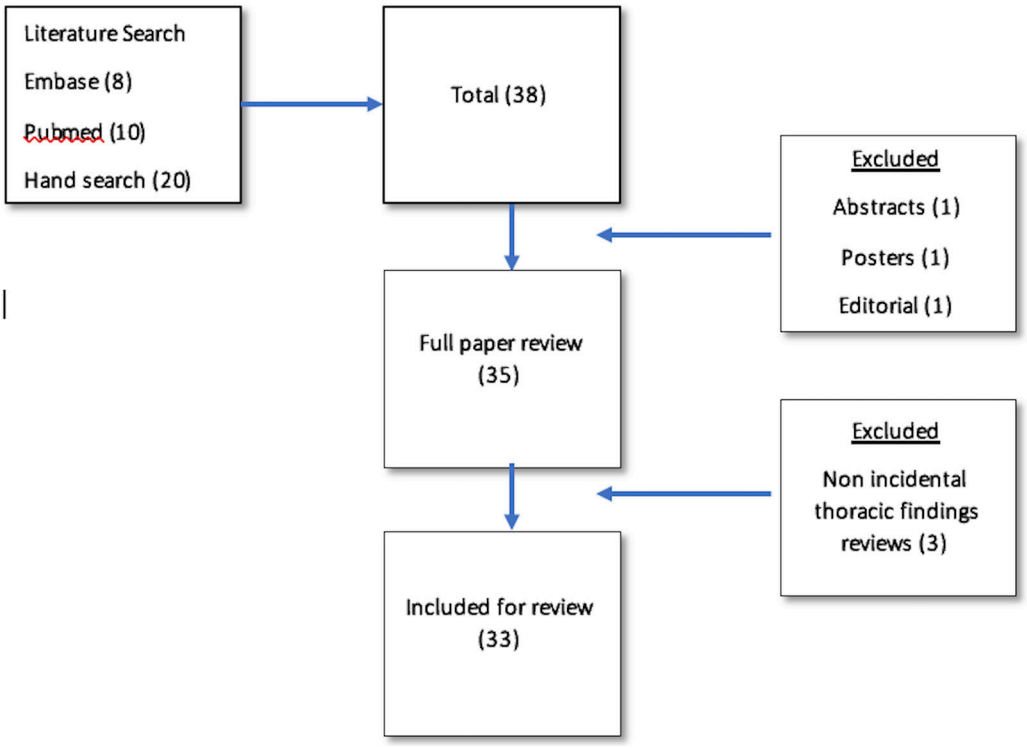
LITERATURE SEARCH

Lung cancer screening programmes have provided large data sets which can be mined for relevant thoracic incidental findings, and as such inform many of the expected rates of various findings. These data sets are certainly valuable, but they are likely to overestimate the presence of certain types of incidental findings (emphysema or other smoking-related disease, for example) and underestimate others, *e.g.* those in younger people, who are less likely to be imaged. For the purposes of this review, we have conducted a literature review of Embase and PubMed (Figure 1). We also performed a hand search for less common but generally accepted incidental lung parenchymal findings, including congenital findings, and combined this with our preceding

Figure 1. Table and diagram detailing literature search for this manuscript.

EMBASE and PUBMED Search Strategy
1 Thoracic/Lung
2 Incidental findings
3 Computed tomography/CT
4 1, 2 and 3
5 Limit 4 to year "1990 – 2022"

Inclusion criteria	Exclusion criteria
Observational studies	Papers not available in the English language
Retrospective cohort studies	Papers only available in abstract form
Review articles	Papers not reporting on original human data
Position papers	Papers primarily referencing the use of PET-CT/radionuclide imaging
Lung cancer screening outcome papers	Papers primarily referencing the use of radiomics, artificial intelligence or other software not routinely available
Outcome measures relating to the detection of incidental findings	Papers primarily reporting on non-thoracic incidental findings



knowledge of the literature. Publications were excluded if they were abstracts or published in a language other than English.

AIRWAY-ASSOCIATED ABNORMALITIES

Bronchiectasis

The prevalence of bronchiectasis ranges from less than 1% to almost 10%, depending on the studied cohort and radiologic definition used.[5–10] "Bronchiectasis" can broadly be defined as the presence of airways with an overall diameter greater than the homologous pulmonary artery—*i.e.* an increased bronchoarterial ratio (BAR)—and is usually associated with other signs such as bronchi being peripherally visible (either within 1 cm of the costal pleural surface or contacting the mediastinal pleura)[11] and a lack of airway tapering.[12] Further characterisation into cylindrical, cystic and varicose subtypes is also described[13] but, while visually evocative, does not generally offer discriminatory value with respect to aetiology. Rather, it is more helpful to distinguish such "freestanding" bronchiectasis—where accompanying architectural distortion is absent—from the distortion and dilatation of airways by fibrosis, termed traction bronchiectasis. Bronchiectasis is also described as mild, moderate or severe in research studies, depending on whether the BAR is 1–2, < 2 but <3, or >3, respectively.[14]

Although bronchiectasis is most frequently idiopathic or the consequence of previous infection, multiple other well-known associations and causes such as primary ciliary dyskinesia, cystic fibrosis, and chronic immunocompromise are recognised.

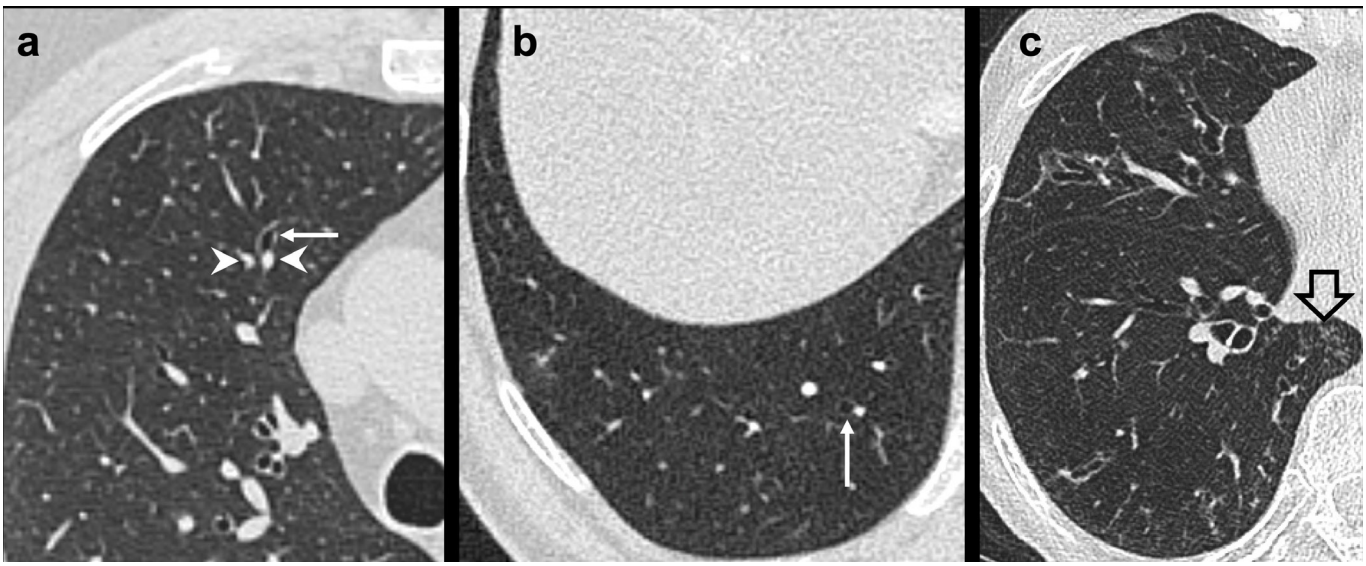
Using a BAR >1 alone to define bronchiectasis is problematic for a few reasons. First, a physiologic increase in the BAR is to be expected in certain situations, especially increasing age and high altitude (the latter due to relative hypoxic vasoconstriction of the adjacent pulmonary artery).[11] Furthermore, the overall diameter of the bronchus may be increased in acute inflammation of the airway not because the airway itself is dilated, but due to thickening of its wall, rendering the use of BAR fallible. To mitigate against the latter, more recent definitions have relied on the use of *internal* diameter rather than *overall* diameter in determining the ratio[11]; however in an inflamed bronchiectatic airway, the internal diameter could be decreased, conversely resulting in a spuriously low BAR. Cylindrical bronchial dilatation, without a physiologic deficit, is also recognised as an ancillary finding in chronic pulmonary embolism.[15,16] Lastly, there is a wide range of normal ratios. Kim et al demonstrated that the artery-to-outer bronchus ratio in healthy subjects had a mean of 0.98 ± 0.14, and suggested that, if normal was defined as mean ± 2 standard deviations, then a normal such ratio would vary from 0.70 to 1.26[17] ; in other words, the BAR (its inverse) in healthy subjects could be between 0.79 and 1.43 (Figure 2).

Finally, it is important to recognise that dilated airways within areas of consolidation should not be considered bronchiectatic; such transient dilatation in acute inflammation is well-recognised and described in the earliest CT descriptions of bronchiectasis by Naidich and colleagues.[12]

Bearing in mind that clinical labels of "bronchiectasis" can be assigned as soon as they appear in a radiological report, and once assigned are often perpetuated indefinitely, we suggest the following pragmatic approach to reporting airway calibre:

(1) Ignore airway dilatation which is mild (*e.g.* a BAR <1.5) (Figure 2b) especially in older participants or scenarios such as high altitude or CT for lung cancer screening only[18] ; if one feels compelled to mention so as to dismiss it, use a

Figure 2. Bronchial calibre assessment. (a) Spuriously increased BAR in the anterior right upper lobe, as the subsegmental bronchus (arrow) is obliquely oriented relative to the homologous pulmonary arteriole, which has also just bifurcated (arrowheads). (b) A single subsegmental bronchus (arrow) which is slightly larger than the accompanying artery within 2 cm of the pleura can be ignored. (c) Established cylindrical bronchiectasis in a 79-year-old female with subsequently proven non-tuberculous mycobacterial infection. Note the tree-in-bud nodularity in the medial right lower lobe (block arrow). BAR, bronchoarterial ratio.



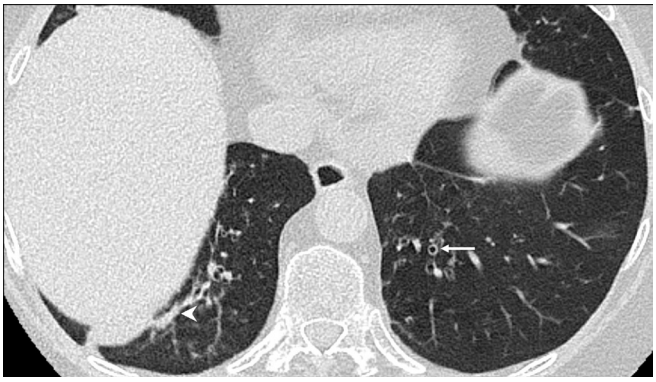
phrase such as "airway calibre considered the upper limit of normal and not significant".
(2) If airway dilatation is present in only one or two segments, ignore it or consider stating that there is "certainly no diffuse objective bronchiectasis."
(3) If airway dilatation is definitely present and diffuse
    (a) Consider whether the ancillary signs of airway dilatation and non-tapering are present, before labelling it bronchiectasis (Figure 2c);
    (b) Assess whether it is "freestanding" rather than "tractional" by looking for distortion and other signs of fibrosis
    (c) Assign a grade of mild, moderate or severe using the BAR as suggested above.[14]

Bronchial wall thickening

Bronchial wall thickening (BWT) is a descriptive term denoting the end result of irritation to the airways from a variety of causative factors with or without the association of endobronchial mucus plugging. BWT, whilst encountered not infrequently on routine reporting of thoracic CT, has not been specifically addressed in the large thoracic data sets generated from LCS.

Although LCS data thus far have shed relatively little light on its current prevalence as an incidental finding amongst the smoking population, more historical literature has cited a strong causative relationship between the two. In 1993, Remy-Jardin et al[19] demonstrated an 33% incidence of BWT in smokers *vs* 16% in non-smokers in a prospective analysis of 175 patients. Following on from this, in 2003 Matsuoka et al[20] and soon after in 2004, Vikgren[2] et al both demonstrated an increased frequency of BWT in smokers compared to non-smokers in their respective prospective analyses. Interestingly, both groups also observed a positive correlation between the frequency of detection of BWT and increasing patient age, thus postulating that BWT should be considered along the spectrum of normal in patients over the age of 65 regardless of their smoking status (Figure 3). Copley et al[21] also re-affirmed the synergistic relationship of BWT and increasing age when describing the continuum of normal in the senescent lung. Interestingly, however, a recent study of 99 individuals (48 never smoked and 51 currently smoking) with a median age of 39 years re-affirmed the positive association of current smoking status with increased BWT, but conversely found decreasing BWT with age[22]—this difference perhaps being somewhat attributable to their use of three-dimensional and automated, rather than visual, airway thickness measurement.

Figure 3. Single thickened subsegmental bronchus in the medial left lower lobe (arrow). There is at most trivial bronchial wall thickening and some atelectasis in the basal right lower lobe, both of which can probably be discounted.



Defining the normal range of BWT is vexatious not least because, unlike bronchial calibre, there is limited value in comparing BWT relative to the homologous pulmonary artery outside of research studies. A lower internal to external bronchial diameter ratio could hypothetically indicate increased BWT, but the wide range of such ratios in normal subjects (0.51–0.86 as reported by Kim et al[17]), as well as fluctuation with technical factors such as end-expiration, increased image noise, motion, mucus plugging and narrow window widths (especially<1000 HU)[23]—all of which also cause overestimation of BWT—preclude its pragmatic use. Quantitative definitions of wall thickness, such as airway wall area percentage and the airway wall thickness for a theoretical airway with an internal perimeter of 10 mm (AWT–Pi10),[24] have existed for almost two decades, and but are still not part of routine clinical imaging software, despite showing good physiologic correlation.

As such, we should probably only mention incidentally discovered BWT if (subjectively defined) severe narrowing of the internal bronchial diameter by circumferential thickening is accompanied by at least one of the following[1]: mucus impaction (being careful not to mistake this mucus impaction for circumferential wall thickening)[2]; bronchiectasis (as discussed above); and[3] diffuse patchy low attenuation areas that could indicate coexistent small airways obstruction (see later section). Even when worthy of mention, it is still probably helpful to state categorically that while this finding indicates airways inflammation, it can be disregarded if the patient has reported no respiratory symptoms, rather than a reflex statement asking the referrer to simply "correlate clinically".

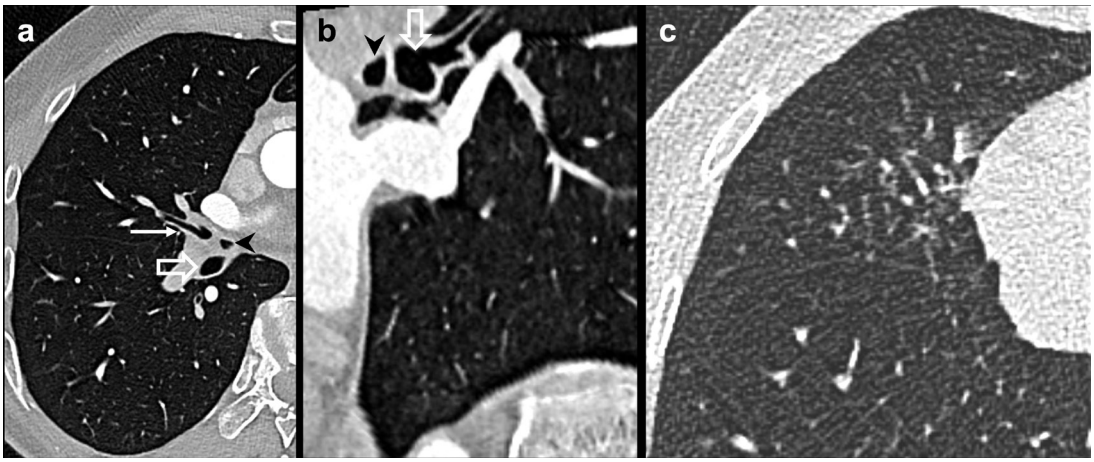
It is also worth bearing in mind that, although both BWT and bronchiectasis are frequent features of connective tissue diseases, they are usually clinically silent even in these populations[25] ; thus, exhorting referrers to search for such underlying conditions when these incidental findings are uncovered is probably futile at best and harmful at worst. In lung cancer screening, reporting of BWT is not recommended.[18]

Congenital variation

Congenital airway variants are relatively rare and typically comprise tracheal bronchus, accessory cardiac bronchus and bronchial agenesis/aplasia/hypoplasia, with the latter technically falling into the bracket of a pulmonary airway malformation and will not be explored further here.

A tracheal bronchus describes an accessory or aberrant bronchus which originates from the lateral wall of the trachea and supplies the upper lobe, with a prevalence of 0.1–2% for the right and 0.3–1% for the left having been reported on bronchoscopic evaluation.[26] There are two subtypes; supernumerary, which exists

Figure 4. Accessory cardiac bronchus in a 35-year-old male undergoing a CT coronary angiogram for unexplained dyspnoea and a family history of premature coronary artery disease. Coronary arteries were normal; however axial (a)and sagittal (b) 1mm lung reconstructions demonstrate a blind-ending cardiac bronchus (black arrowhead) arising opposite the middle lobe bronchus (arrow), and anteromedial to the right lower lobe bronchus (block arrow). (c) Wide field of view 1mm lung reconstructions show tree-in-bud nodularity in the middle lobe, indicating that in this case the cardiac bronchus may be acting as a sump for mucus accumulation and reaspiration in the context of the patient's symptoms; however, the vast majority of patients with this variant are completely asymptomatic.



in addition to an anatomically normal upper lobe bronchus, and displaced, which exists in place of an absent segmental upper lobe bronchus and usually arises directly from the trachea. A 'pig bronchus' or 'bronchus suis' can be used to describe an origin of the entire right upper lobe bronchial tree directly from the trachea and has a reported incidence of 0.2%.[26,27]

Accessory cardiac bronchus (ACB) is the rare entity of a true supernumerary bronchus arising from the medial wall of the right or left main bronchus or the bronchus intermedius[27] which courses caudally towards the pericardium (Figure 4). ACB is stereotypically blind-ending but can occasionally branch and supply a small lobule of normal lung parenchyma, hypoplastic lung or a ventilated lobule within the azygo-oesophageal recess demarcated by an accessory fissure (cardiac lobe). Unlu et al[28] reported a prevalence of 0.2% from a retrospective analysis of ACB in 5790 patients undergoing CT chest with interestingly all positive cases being asymptomatic.

Indeed both a tracheal bronchus and ACB are usually asymptomatic as reported in the literature and whilst they do not necessarily pose a diagnostic reporting dilemma in the way other incidental findings can, it is worth remembering that they can be associated with cough, haemoptysis, recurrent infection (especially ACB, which can act as a "sump" for accumulation of secretions), and rarely malignancy.[28] Thus, it is useful as a matter of record to describe these anomalies if encountered, even if no clinical significance needs to be attached to them.

Low attenuation areas

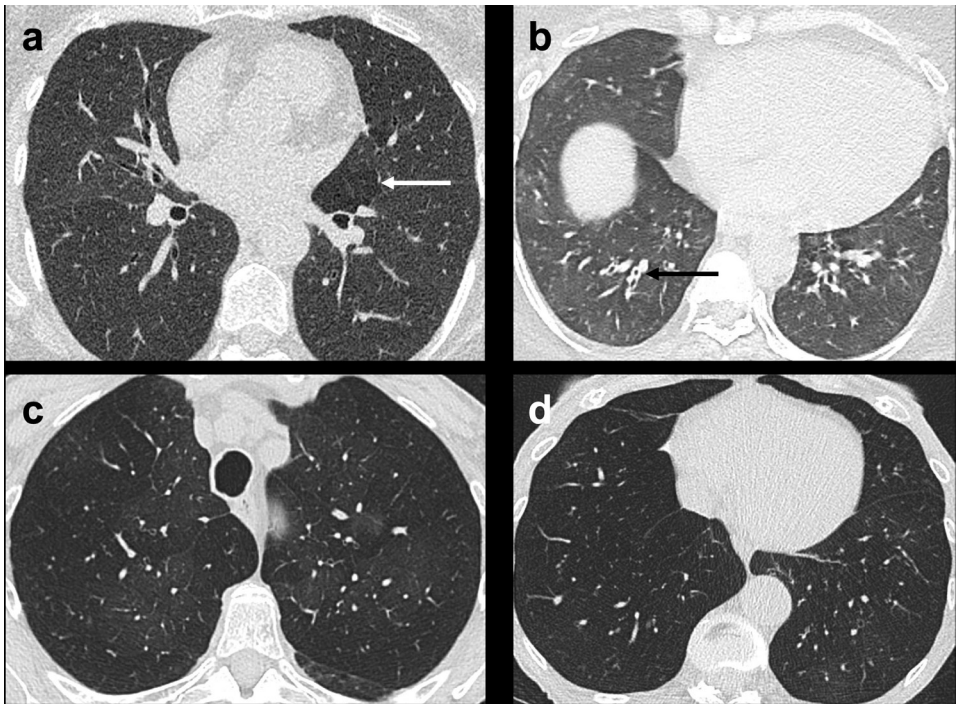
Foci of decreased attenuation, which may be lobular, segmental or even non-segmental, can be incidentally encountered on thoracic CT, and exaggerated by the presence of iodinated contrast and an (commonly unintended) expiratory phase of imaging. In fact, 50–80% of healthy asymptomatic and physiologically normal individuals may have expiratory decreased attenuation.[29–33] Such decreased attenuation usually reflects small airways dysfunction that has resulted in a combination of localised hypoxic vasoconstriction and decreased perfusion, as well as a degree of "air-trapping". It can involve as much as 25% of the lung in healthy asymptomatic individuals, and increase with age. It is important to note that chronic pulmonary thromboembolic disease also causes foci of decreased attenuation, and attenuation of pulmonary arteriolar calibre in the regions of decreased attenuation is common to both small airway- and chronic thromboembolic-mediated decreased attenuation.

Thus, if foci of decreased attenuation are seen, we suggest discounting them even in a younger patient if affecting up to a quarter of the lung volume on expiratory imaging (Figure 5a and b). If they are more extensive, or considered prominent on a good quality inspiratory non-contrast CT, the radiologist should first determine whether the lobular pulmonary artery calibre is decreased (relative to areas of normal attenuation) in these areas (Figure 5c and d). A decreased arteriolar calibre should then prompt a search for significant BWT or mucus impaction as signs of unequivocal coexistent airways inflammation (potentially indicating coexisting large and small airways disease) and the size of the main pulmonary artery relative to the ascending aorta (as a surrogate of potential pulmonary hypertension); in the absence of the latter ancillary findings, it would be reasonable to ignore the decreased attenuation as it would in all likelihood be clinically insignificant.

Emphysema

Because of its causative relationship with smoking, emphysema is unsurprisingly found much more commonly in lung cancer screening (LCS) cohorts, where rates range from 11 to 50%[7,9,34–36] and subjects are likely to be older and have significant smoking histories, compared to non-LCS studies, where rates range from

Figure 5. Examples of low-attenuation areas in three different individuals. (a) Solitary focus of low attenuation in the medial lingula (arrow), with relatively decreased pulmonary arterial calibre; this may be the result of localised small airways obstruction but is often encountered and best ignored. (b) 44-year-old obese female. 1mm axial expiratory HRCT slice shows patches of decreased attenuation, but not exceeding the 25% upper limit of LAA which can be seen in normal patients. Note the apparently thickened subsegmental bronchi which is probably spurious due to the expiratory phase. (c) Upper and (d) lower axial HRCT slices from a 78-year-old female who is currently smoking, showing extensive mosaic attenuation—even on this inspiratory phase—with marked lobular pulmonary arterial calibre attenuation, representing extensive small airways obstruction in this context. With compatible obstructive pulmonary function, this would represent obliterative bronchiolitis. HRCT, high-resolution CT.



2 to 12%[6,37–41] and patients are more likely to be younger and healthier. Perhaps what is surprising is the range of prevalence *within* these groups of patients, likely attributable to different ways of scoring emphysema, and moderate inter- and intraobserver agreement for its presence,[32] as well as true intercohort and interindividual variability. Of note, emphysema has been reported in 6–9% of individuals with no smoking history,[33,42] and 11% in non-smoking chronic obstructive pulmonary disease (COPD) populations exposed to biomass fuels or other environmental occupational risk factors.[43]

The classification of emphysema based on its acinar location into centrilobular, paraseptal, and panlobular subtypes is well-drilled into radiologists regardless of subspeciality interest. Foci of centrilobular emphysema (CLE) are characterised by small hypoattenuating areas that are usually poorly defined but may sometimes have a barely perceptible wall (especially if the emphysematous destruction extends to the interlobular septa), making them sometimes difficult to distinguish from pulmonary cysts (Figure 6). The presence of centrilobular arterioles traversing, rather than being displaced by, the hypoattenuating focus is somewhat helpful in characterising the lucency as CLE as opposed to a pulmonary cyst (we discuss pulmonary cysts later). CLE is usually upper lobe predominant. In contrast, paraseptal emphysema (PSE) is characterised by sub- or juxtapleural and peribronchovascular foci of low attenuation separated by intact interlobular septa, almost invariably affecting the lung apices.

The visual grading of emphysema extent involves assessing the overall percentage of lung affected. Typically grading was pragmatically classified as mild (1%–≤25%), moderate (>25–50%),

Figure 6. Pulmonary lucencies. (a) 8mm poorly lucency has poorly defined walls. It is unclear if this is truly a "cyst" and may be a solitary focus of centrilobular emphysema, which can probably be ignored. (b) 7mm slightly better-defined solitary pulmonary cyst in a 47-year-old male with no smoking history; such cysts are not uncommon over the age of 40 and can be ignored.

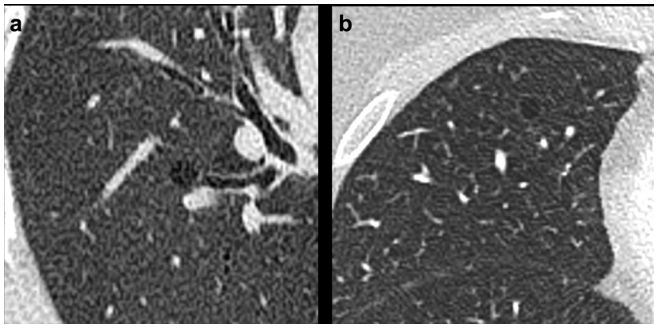
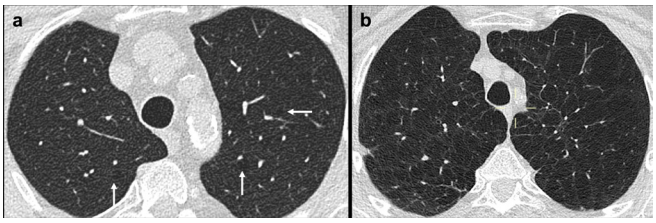


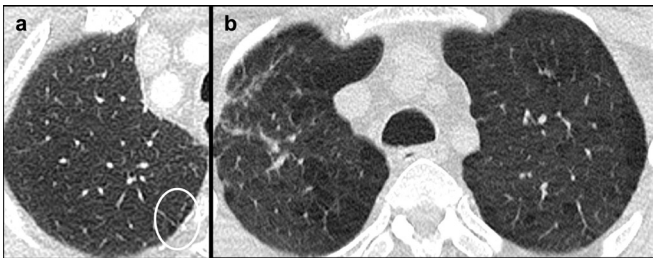
Figure 7. Different grades of CLE according to the Fleischner Society recommendations. (a) Trace CLE—scattered centrilobular lucencies (arrows) that overall occupy less than 0.5% of this upper lung zone. (b) Advanced destructive CLE with coalescent lucencies causing hyperexpansion and architectural distortion. CLE, centrilobular emphysema.



or severe (>50%),[44] and this grading is still recommended for reporting emphysema on lung cancer screening CT in England.[45] However, in 2015 the Fleischner Society recommended a new grading system that is probably less familiar to the general radiology community. This system juxtaposes both extent of involvement and degree of parenchymal destruction,[46] dividing the lungs into three zones demarcated by the carina and inferior pulmonary veins, respectively. CLE is graded as trace (<0.5% of a lung zone); mild (0.5–5% of a lung zone, *i.e.* with largely normal interspersed lung); moderate (>5% of any lung zone); confluent (coalescent foci of CLE that may span several pulmonary lobules but without hyperexpansion or architectural distortion); and advanced destructive (coalescent CLE with hyperexpansion and architectural distortion) (Figure 7). In contrast, paraseptal emphysema is graded as mild (juxtapleural lucencies ≤ 1 cm in diameter) or substantial (mainly >1 cm juxtapleural lucencies and bullae) (Figure 8). This visual grading has been shown to predict emphysema progression in patients with both a current and former smoking habit with and without known COPD, with good interobserver agreement.[47]

Given the relative frequency of emphysema in both ever-smoking and non-smoking populations on CT then, it is worth understanding what, if anything, a radiologist should say about it. COPD diagnosis is based on symptoms and airflow obstruction in accordance with Global Initiative for Obstructive Lung Disease (GOLD) guidelines,[48] and not on imaging characteristics alone. In the USA, a multidisciplinary expert panel convened by the journal "CHEST" did not recommend further follow-up of emphysema (or BWT) found on CT performed for screening.[49] It is also reassuring that the Fleischner Society recommends ignoring the presence of up to five cysts at the lung apices,[46] since minimal subpleural (*i.e.* paraseptal) emphysema is common even in non-smoking populations[32,33] (Figure 8a).

Figure 8. Paraseptal lucencies. (a) Approximately, four subpleural subcentimetre lucencies (circle) in the posteromedial right apex in a 55-year-old male with no smoking history. These can be ignored. (b) Moderate (>5% of the lung zone) centrilobular and substantial (mostly >1cm subpleural blebs) paraseptal emphysema, along with some scarring in the lateral right upper lobe.



We therefore take the view that the presence of emphysema should, at most, be recorded in the body of a report, and perhaps prompt an enquiry about smoking history. If trace in extent and encountered in a younger, non-smoking individual, it is just as reasonable to ignore, or at most mention it in the body of report but state that it is not considered significant. If it is moderate or greater in extent, it seems practical to report it and at most prompt the physician to enquire about both a smoking history and respiratory symptoms, even as we concede that the utility of the latter enquiry is uncertain.

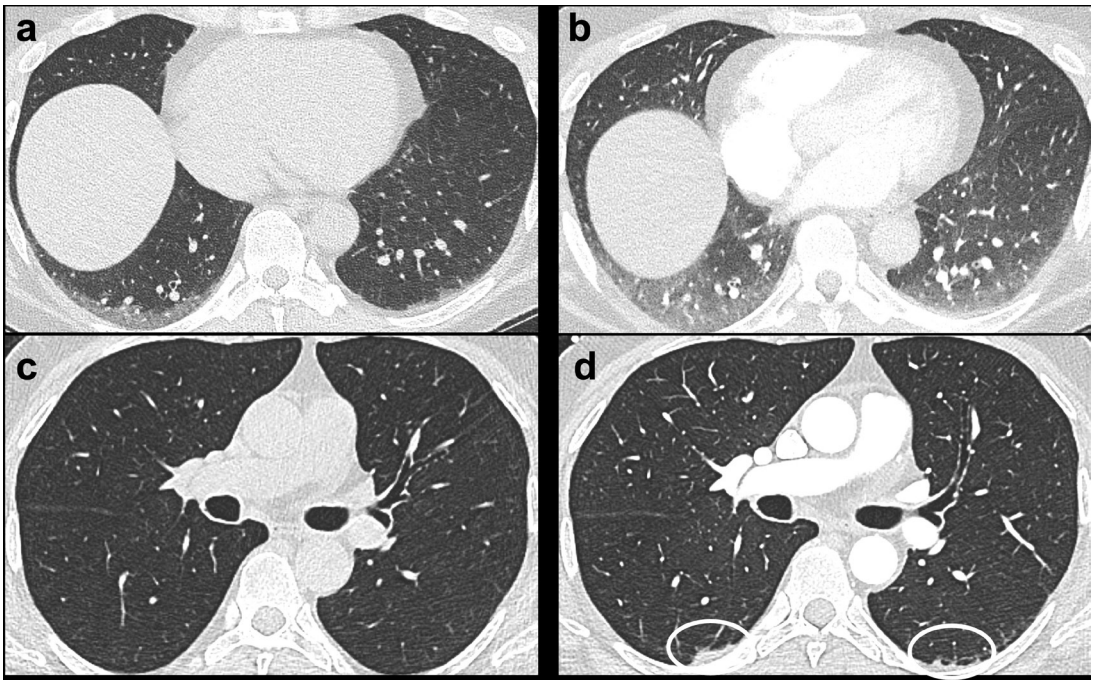
DIFFUSE PARENCHYMAL LUNG ABNORMALITIES

Diffuse lung disease (DLD) is an umbrella term encompassing a multitude of diseases that affect primarily the lung interstitium, interfere with gaseous exchange and may eventually result in some degree of pulmonary fibrosis. DLDs can broadly be classified according to the following: whether they have a known aetiology, *e.g.* connective tissue disease (CTD), the idiopathic interstitial pneumonias (IIPs), granulomatous DLDs and another group which includes cystic lung diseases such as pulmonary Langerhans Cell Histiocytosis (LCH) and lymphangioleiomyomatosis (LAM).

The reporting of diffuse parenchymal lung abnormalities can be challenging to even a thoracic radiologist. Because the diagnosis of DLD requires the integration of clinical, functional, radiological and pathological evaluation, rather than any single one of these—not even pathology—being considered a diagnostic "gold" standard, radiological evaluation is given at least equal weighting in this process, which can both reward and worry the reporting radiologist. This emphasis on radiologic interpretation, coupled with a mystique surrounding parenchymal evaluation that is engendered at the training level, can lead radiologists to overcall interstitial findings; after all, they do not want to miss an opportunity to diagnose such diseases early, in particular the subgroups of interstitial lung diseases that either carry poor prognoses or that may herald a hitherto undiagnosed CTD (so-called CTD-ILD). Such overzealousness may lead radiologists to inadvertently overlook key technical factors that can cause spurious parenchymal increased density, such as the phase of respiration, presence of iodinated contrast, CT acquisition (in particular so-called ultra-low dose) and reconstruction techniques, patient position and habitus[50] (Figure 9).

Here, we briefly review overall prevalence of DLDs, and the main types of IIPs and cystic lung diseases, before discussing how to approach the parenchymal abnormalities that could suggest these diseases when they are encountered incidentally.

Figure 9. Spurious parenchymal appearances due to technical factors. (a) Unenhanced supine end-inspiratory HRCT 1mm slice shows only dependent increased lung density. (b) However, on the CT pulmonary angiogram (CTPA) performed 2 min later, the presence of iodinated contrast, coupled with the gentle inspiration (or even expiration) phase that CTPA examinations are performed with, result in increased overall lung density and dependent high density. (c) In a different patient, unenhanced supine end-inspiratory HRCT 1mm slice shows normal lung density, but a CTPA 9 min later (d) shows dependent atelectasis that was mistaken for pleural nodularity. CTPA, CT pulmonary angiogram



Prevalence

When compared to other incidental lung findings DLDs, by virtue of their diversity and scope, can range from being relatively common in the population, *e.g.* IPF and sarcoidosis which have an annual incidence of 5–10 people per 100,000, to being exceptionally rare.[51] An analysis of the prevalence of lung-related incidental findings from the American National Lung Screening Trial by Pinsky et al demonstrated a prevalence of between 20.1 and 36.8% of DLD amongst over 20,000 participants.[34] Another analysis of a smaller American LCS cohort by Morgan et al demonstrated a prevalence of DLD of 1.6% (5 out of 320 participants).[8] In Europe, the Dutch-Belgian Randomised Controlled Lung Cancer Screening Trial (NELSON study) established an incidence rate of pulmonary fibrosis-related signs in 8% of their cohort (117 out of 1409 participants)[35] and in Italy, Sverzellati et al specifically addressed the rate of ILD in a lung cancer screening cohort which was defined as 9.3% when adjusted for age, sex and smoking status.[52]

It is essential to recognise that the rates of incidental DLD reported across any cohorts including the results from screening programmes reflects an inherent variability in the studies' definition of DLD, *e.g.* the presence of subpleural reticulation and ground-glass opacification in some as opposed to established lung fibrosis in others. Furthermore, in light of the robustly observed relationship between smoking or noxious agent exposure and DLD, it is worth considering the rates of prevalence in screening data sets, which represent a group of current and former smokers, are likely to be artificially high if extrapolated to the general population. It is still too early to tell how, if at all, the Covid-19 pandemic will affect this general prevalence.

It is also worth considering that the term ILA, used with varying definitions over the past 10 years, provides insight into the prevalence of diffuse parenchymal abnormalities. In 2013, Jin et al reported an ILA prevalence of 9.7% (86 of 884 participants) in a National Lung Screening Trial population.[53] Collectively, ILA prevalence across several cohort studies between 2010 and 2016 is around 4–9% in smokers and 2–7% in non-smokers all over the age of 60.[3] However, the definition of ILAs has recently been refined (see later).

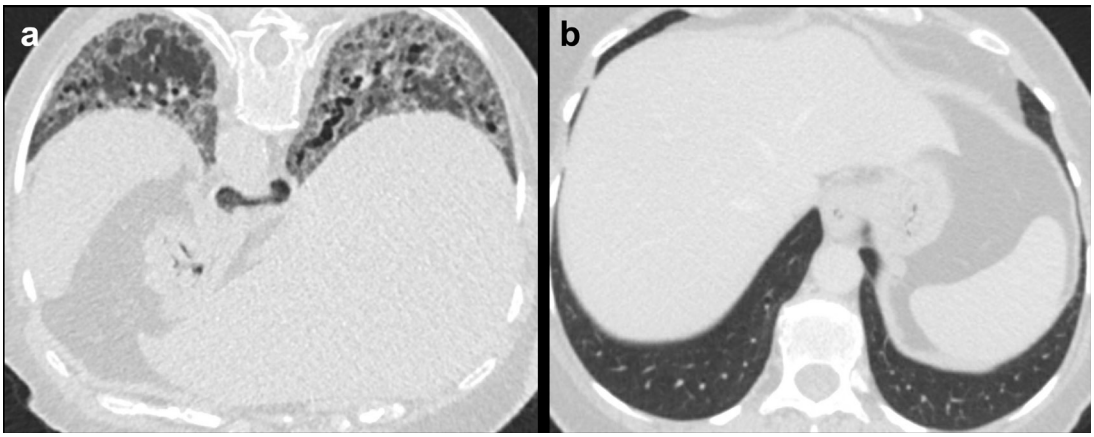
The cystic lung disease subgroup of DLDs are considerably rare, and their reported imaging prevalence is usually based on well-defined cohorts where their diagnoses has been firmly established; the prevalence of such cystic lung diseases in screening or general population imaging cohorts has not been described. That said, the prevalence of lung cysts on thoracic CT is reported as 7.6%, in a study of 2633 asymptomatic Framingham Heart Study participants, with increasing prevalence with age, and none seen below the age of 40.[54] This observation of an increased prevalence with age has been verified in other studies, albeit with different lower age cut-offs of 55[21] and 50[55] years old, respectively.

Types of IIPs and cystic lung diseases

Idiopathic interstitial pneumonias (IIPs)

The idiopathic interstitial pneumonias (IIPs) are divided into six major and two rare multidisciplinary entities, with distinct

Figure 10. Incidentally detected fibrosing lung disease in a 78-year-old female. (a) Soft-tissue 1mm CT slice on lung windows from the prone acquisition of a CT colonography examination demonstrates extensive ground-glass opacity as well as reticulation, with severe traction bronchiectasis. (b) The lung bases were normal on an abdominal CT years 8 years prior, indicating the disease had developed rapidly. A radiologic differential diagnosis of probable UIP or less likely mixed cellular/fibrotic NSIP was made; interestingly the patient also had joint stiffness, sicca symptoms and dysphagia, and a high antinuclear antibody titre of 1 in 320 but no other specific antibodies. Hence, the multidisciplinary team labelled this IPAFs, which is a designation for individuals with interstitial pneumonia and features suggestive of, but not definitive for, a defined connective tissue disease. IPAF, interstitial pneumonitis with autoimmune features; NSIP, non-specific interstitial pneumonia; UIP, usual interstitial pneumonia.



but potentially overlapping clinical, radiological and pathological characteristics. Major IIPs comprise idiopathic pulmonary fibrosis—most commonly manifest radiopathologically as usual interstitial pneumonia (UIP); idiopathic non-specific interstitial pneumonia (NSIP); cryptogenic organising pneumonia (COP), desquamative interstitial pneumonia (DIP), respiratory bronchiolitis-associated interstitial lung disease (RB-ILD) and acute interstitial pneumonia (AIP) (characterised radiologically by diffuse alveolar damage, DAD).[56] Rare IIPs comprise idiopathic lymphoid interstitial pneumonia (LIP) and idiopathic pleuroparenchymal fibroelastosis (IPPFE).[56] The major IIPs are further categorised into chronic fibrosing (UIP and NSIP), smoking-related (DIP and RB-ILD) and acute/subacute (COP, AIP) entities. However, following a radiologic differential diagnosis, a rigorous search for an underlying association, in particular for autoimmune features that may indicate a potential underlying disease that can be treated or at least controlled, will be undertaken by the multidisciplinary team (Figure 10).

Cystic lung diseases

LAM is characterised by thin-walled, regularly-shaped cysts surrounded by normal pulmonary tissue; the cystic lesions are usually uniformly distributed and can lead to pneumothorax or chylothorax, most often in young females. A well-recognised association with tuberous sclerosis exists because of a shared genetic locus. In contrast, patients with pulmonary Langerhans Cell Histiocytosis (LHC) are more likely to have irregular and heterogeneously thick- and thin-walled cysts, often associated with nodules (typically less than 10 mm in diameter) with an upper and mid-zone predominance, sparing of the costophrenic angles. In later stages, fibrosis and architectural distortion supervene. It is a smoking-related disease, but its frequency is currently undocumented in the LCS literature.[57] Other cystic disease, including Lymphocytic Interstitial Pneumonia, Birt-Hogg-Dubé syndrome (Figure 11a and b) and amyloidosis, are very rare, and may have ancillary features, such as ground-glass opacity, a lower lobe predominance, and wall calcification, respectively.

Interestingly, Rowan et al recently described the presence of diffuse pulmonary cyst with coexisting small airways disease in five patients with no other defined cause for cystic lung disease. They hypothesised that, in a minority of patients, chronic damage to small airways may lead to pulmonary cyst formation, perhaps at least in part due to distal air-trapping causing overinflation[58] (Figure 11c and d).

Though not strictly a cystic lung disease *per se*, it is worth mentioning pericystic lung cancers here (Figure 12) in order to highlight some of the challenges around their identification, particularly in the LCS population, where the pretest probability of malignancy is already high. Sheard et al demonstrated that in multiple LCS cohorts, pericystic lung cancers were often initially missed, as they presented not as standalone pulmonary nodules, but as pericystic thickening or nodularity, on a background of architecturally abnormal lungs.[59]

Reporting incidental diffuse parenchymal abnormalities

In 2007, the NELSON study stated that following up even potentially clinically relevant incidental pulmonary findings in the context of lung cancer screening provided no benefit.[35] Similarly, an American CHEST Guideline consensus panel for implementation of lung cancer screening concluded that follow-up for incidentally detected pulmonary fibrosis was not required.[49] More recently, the SUMMIT Study, the largest lung cancer screening trial in Europe to date, opted for a more pragmatic protocol which reports back findings only if there is an evidence-based clinical response that will be activated and lead to an overall patient benefit.[18] In contrast, the American College

Figure 11. Diffuse pulmonary cysts. 54-year-old male with incidentally detected well-defined cysts in the upper zone (a), with a lower zone and slightly peripheral predominance (b). A diagnosis of Birt-Hogge-Dubé syndrome was suggested. (c) Subcentimetre cysts (circled) in a 45-year-old male who does not smoke. (d) Both the cysts and an area of peripheral decreased attenuation in the lateral left lower lobe are accentuated on a minimum intensity projection reconstruction (10 mm thickness, window width 500 HU, centre −856 HU). It transpired that the patient had a diagnosis of well-controlled asthma and normal lung function; the cysts were thus attributed to the recognised phenomenon of cysts associated with small airways disease.

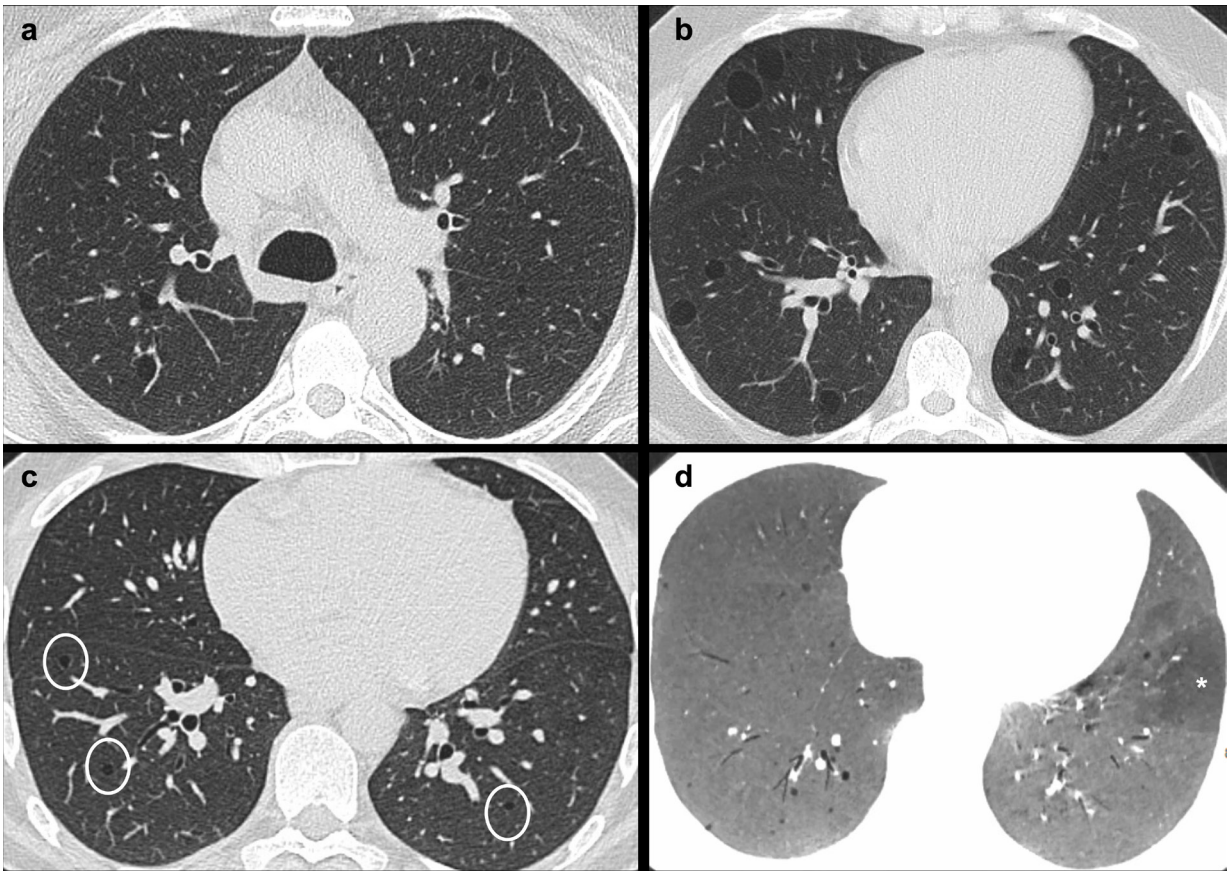
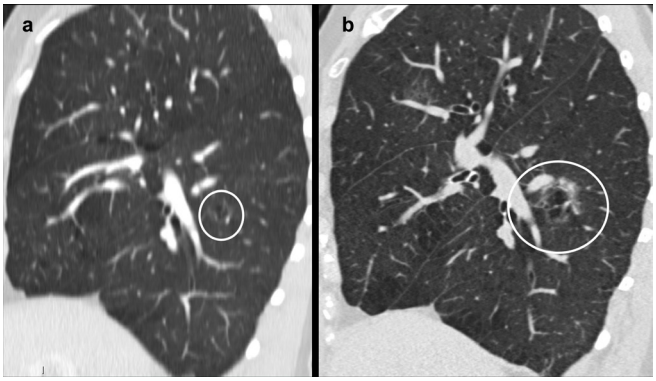


Figure 12. Lung cancer associated with a cystic airspace in a 75-year-old female. (a) Sagittal reconstruction of a 3-mm-thick post-contrast CT chest demonstrates a thin walled multiseptated cyst in the right lower lobe (circle) (no higher-resolution reconstructions were performed). (b) Over 8 years, the cyst eventually developed a peripheral solid component and ground-glass nodularity, concerning for a lung cancer associated with a cystic airspace. Surgical resection proved a T1aN0M0 invasive pulmonary adenocarcinoma.

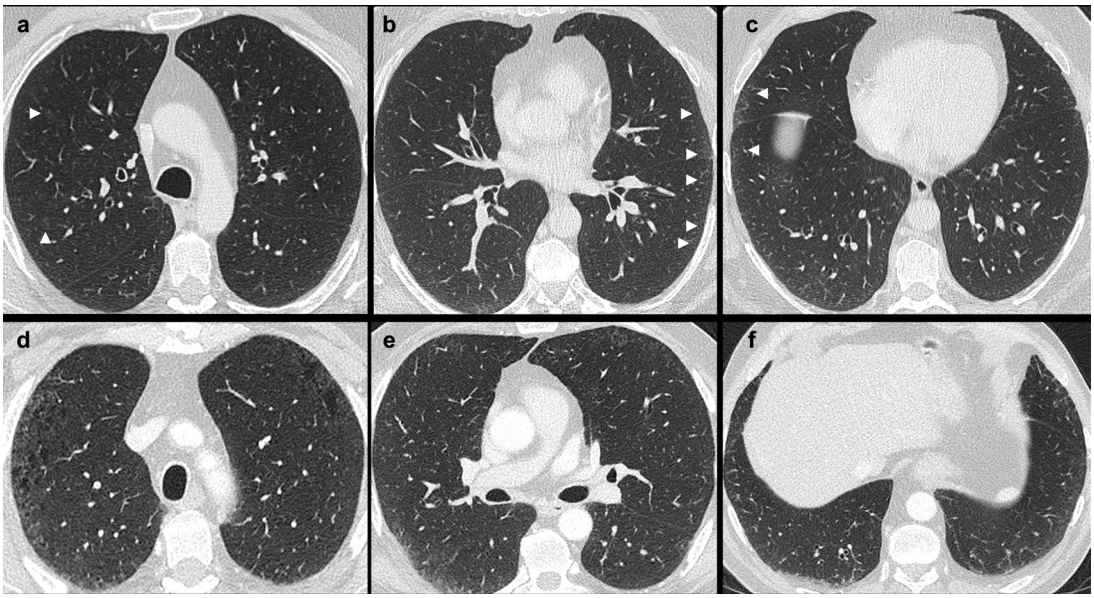


of Radiologist guidelines on reporting thoracic incidental findings offer ample guidance on *how* (but not always *when*) to report these findings, considering their distribution and profusion, with the overarching take-home being that these are best managed in a shared decision-making process between the radiologists, referrer, and patient.[60]

Where does this leave the radiologist in their verdict on when to report, and whether to label these incidental pulmonary abnormalities as significant or not? Radiologists are no doubt left to set our own thresholds based on the likelihood of clinical significance, since it would be impractical to report *every* degree of these findings but then abnegate responsibility for them by invoking shared decision-making.

Recently published guidance on ILAs offers some direction. The Fleischner position paper on ILA in 2020 describes it is as a primarily radiological descriptor for incidental imaging abnormalities on thoracic CT encountered in patients in whom interstitial lung disease is not suspected; *i.e.* to say, the term should not be used in patients with a recognised predisposition to ILD (*e.g.* CTDs), nor is it synonymous with subclinical ILD, because

Figure 13. Defining ILAs in two different patients. Axial 1mm HRCT slices from a 58-year-old male with no smoking history demonstrates barely perceptible ground-glass nodularity (arrowheads) in the upper zone (a), and minimal subpleural ground-glass opacity in the mid (b) and lower (c) zones (arrowheads in representative areas shown). The overall extent in any one zone is less than 5% of the lung area, so this would not qualify as an ILA. Axial 1mm lung reconstructions from an arterially enhanced CT in a 60-year-old male who currently smokes demonstrates centrilobular emphysema with admixed peripheral ground-glass opacity, involving more than 5% of the the upper zone (d), with minimal subpleural ground-glass opacity in the mid (e) and lower (f) zones. This can be classified as a smoking-related ILA, but whether it represents subclinical *vs* early smoking-related interstitial lung *disease* depends on the respective absence or presence of symptoms and a physiologic deficit. (*Images d, e and f courtesy of Dr Asia Ahmed, Consultant Thoracic Radiologist, University College London Hospital.*)HRCT, high-resolution CT; ILA, interstitial lung abnormality.



patients may have respiratory symptoms and impaired lung function that may represent a mild or early ILD.[3] Further, they refine the definition of ILA as the presence of non-dependent changes affecting at least 5% of any lung zone and include reticulation, ground-glass opacification, diffuse centrilobular nodularity, non-emphysematous cysts, honeycombing and even tractional airways dilatation.[61] (Figure 13). The term does not include insignificant abnormalities such as paraosteophyte localised fibrosis. Evidently, ILA is a relatively common finding with established overlap in the smoking and ageing population due to previously aforementioned commonality in pathological processes that arise as a consequence of both "conditions". ILA can also be further classified into fibrotic and non-fibrotic types but the intricacies of this is beyond the scope of this paper.

We thus suggest the following approach in reporting diffuse parenchymal lung abnormalities:

(1) Consider whether the apparent abnormality could be the result of technical factors (phase of respiration, presence of iodinated contrast, CT acquisition and reconstruction technique, patient position and habitus) (Figure 9).
(2) Ensure comparison with any imaging that allows visualisation of the lung bases (such as a CT abdomen), particularly if recent, as that imaging may show that the abnormality was not present recently and is spurious—or it may show that it is progressive (Figure 10).
(3) When considering whether the abnormality is genuine, assess whether it is solely dependent (and thus the result of atelectasis) or whether it also extends into non-dependent regions. Do not consider para-osteophyte fibrosis as significant.
(4) If there is a high degree of concern that the abnormality is genuine rather than spurious, consider recalling the patient for additional manoeuvres such as prone positioning, respiratory coaching[62] or (in cases where only post-contrast CT is available) non-contrast CT- but with a high threshold for doing so.
(5) Use the radiological descriptors according to their rigorous definitions, and be aware of their pathophysiological correlates, to ensure accuracy (Table 1).[65]
(6) Be aware of what is and is not an ILA. Assess extent based on lung zones or overall lung involvement, using the 5% extent threshold for defining an abnormality ILA. In particular, be sensitive to findings that indicate fibrosis (traction bronchiectasis, honeycombing, and volume loss) away from osteophytes (Figure 13).
(7) With respect to cysts, we suggest:
    (a) ignoring one or two pulmonary cysts seen in isolation, even in a younger patient (Figure 6b);
    (b) with more profuse cysts, evaluate their morphology, distribution, and presence of ancillary findings, according to the schema suggested by the ACR[60] (Figure 14) (to

Table 1. Common radiological descriptors of ILA/DLD and their pathological correlates

Radiological finding	Radiopathological description	Cause
Ground-glass opacification	Hazy increased attenuation of lung but with preserved bronchovascular markings[63]	Partial filling of airspaces (with blood, fluid), interstitial thickening, partial alveolar collapse, normal expiration or increased capillary blood volume
Reticulation	Innumerable, interlacing line shadows that suggest a mesh. Can be fine, intermediate or coarse[63]	Thickening of the interlobular or intralobular septa, or development of intralobular (non-septal) lines, usually representing interstitial lung disease
Tractional bronchiectasis	Abnormally dilated airways with an irregular or corrugated contour	Mechanical traction on the airways secondary to fibrosis of the surrounding lung
Honeycombing	Clustered, cystic airspaces with diameters ranging from 0.3 to 2.5 cm usually subpleural with well-defined walls, which are often thick[63]	Destroyed, fibrotic and cystic lung characterised by complete loss of the normal acinar and bronchiolar lung architecture[63]
Centrilobular nodularity	Small, rounded usually ill-defined opacities typically ranging in size from a few millimeters to a centimeter, centred along the central bronchovascular structures in a secondary pulmonary lobule[63]	Usually disease in which the original lesion develops near the bronchioles including hypersensitivity pneumonitis, small airways disease and infections, *e.g.* tuberculosis
Non-emphysematous cysts	A round, parenchymal space with a well-defined wall; usually air containing but without evidence of pulmonary emphysema[63]	Sustained, unresolved insults to the pulmonary airways and parenchyma[64]

DLD, diffuse lung disease; ILA, interstitial lung abnormality.

determine if they suggest a particular diffuse cystic lung disease) (Figure 11);

(c) assess if any cysts demonstrate irregular wall nodularity or a solid component (especially if there is prior imaging to demonstrate that this is evolving); if there is, consider recommending follow-up of these regions. In the absence of formal guidance, we think it practical to base follow-up on the nodular component, using current nodule management recommendations as a guide (Figure 12).

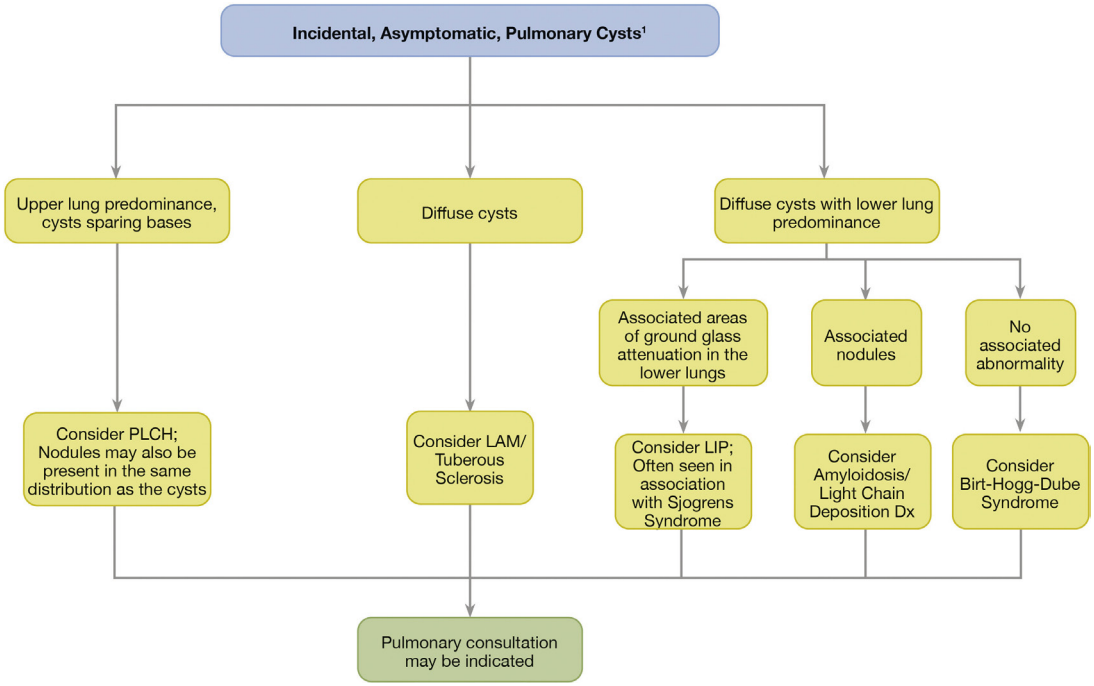
(d) If there are no features of a defined diffuse cystic lung disease, the need for any further testing is questionable.

As with all such abnormalities, please consult a subspecialty thoracic radiologist if in doubt, especially before committing a referrer and patient to further investigation.

CONCLUSION

The gamut of non-nodular thoracic incidental findings will always cause a certain degree of consternation to the reporting radiologist; as David Hansell pointed out in his seminal "Thin-Section CT of the Lungs: The Hinterland of Normal", this predicament is a result of '*..the frequent problem of the "oversensitivity" of thin-section CT and the discrimination between the very earliest*

Figure 14. Evaluation schema for incidentally detected pulmonary cysts, as recommended by the American College of Radiology. Reprinted from reference[60] with kind permission of Elsevier. LAM, lymphangioleiomyomatosis; LIP, lymphoid interstitial pneumonia.



signs of disease and unimportant findings that lie within the normal range'.[4] In this article, we have sought to highlight characteristics of the main airways-based and diffuse parenchymal abnormalities that discriminate between normality and disease states, and then empower the radiologist with pragmatic reporting recommendations. Nevertheless, radiologists will always have different thresholds for deciding when and how to action these findings. Since these thresholds depend on judgement and experience, they are subject to cognitive bias, in particular availability (or recency) bias—*i.e.*, judging diagnostic probability based on the most easily recalled, or recent, examples.[66] In deciding where to set their own thresholds, we can only ask that radiologists are always cognizant of these biases and maintain the guiding principle that in the absence of concrete recommendations, it is better to be specific rather than sensitive when it comes to incidental findings, doing our part to avoid "too much medicine".

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