Immunotherapy-related interstitial lung disease: a call to improve risk stratification and diagnostic consistency

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A standardised risk assessment before initiating ICI treatment may improve early detection and management of IR-ILD. Multidisciplinary collaboration is crucial for enhancing patient safety, improving treatment outcomes and advancing research. https://bit.ly/4qAthl6

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Immune checkpoint inhibitors (ICIs) are immunomodulatory monoclonal antibodies that enhance T-cell-mediated cytotoxicity and have significantly transformed the treatment and outcomes of many cancers [1]. The primary targets of checkpoint inhibition are programmed cell death receptor 1 (PD-1), its ligand programmed cell death ligand 1 (PD-L1) and cytotoxic T-lymphocyte associated antigen 4 (CTLA-4) [2]. However, the success of ramping up the body's immune response comes at a price; the increasingly recognised spectrum of immune-related adverse events that can affect almost any organ, including the lung parenchyma, and cause severe dysfunction. Immunotherapy-related interstitial lung disease (IR-ILD) represents one of the most severe complications, affecting 2–4% of patients treated with

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ICIs, often leading to permanent discontinuation of ICI treatment and, rarely, fatal events [3]. This complication can in some rare conditions unmask an underlying ILD that can be considered as a risk factor of developing IR-ILD [4].

Several other systemic anticancer agents (for example trastuzumab deruxtecan (T-DXd), everolimus, bleomycin, methotrexate, as well as epidermal growth factor receptor tyrosine kinase inhibitors such as erlotinib, gefitinib and osimertinib) have also been implicated in the development of drug-induced ILD [5]. Although less frequent than ICI-related pneumonitis, drug-induced ILD from these agents can present with similar clinical and radiographic features, particularly in patients undergoing combination or sequential therapies. Such overlaps complicate the attribution of causality and highlight the need for broader awareness of drug-induced lung injury in oncologic care [6].

There is a current lack of evidence and structured recommendations that needs to be addressed to provide guidance on the best practice for identifying, diagnosing and monitoring IR-ILD. In this editorial, we highlight the objectives set forth by a European Respiratory Society task force to strengthen evidence-based recommendations.

Pathogenic mechanisms of drug-induced lung injury

The underlying mechanisms of drug-induced ILD are heterogeneous, reflecting the diverse nature of anticancer therapies. ICIs drive cytotoxic T-cell activation, alveolar inflammation, and cytokine release [7]. Epidermal growth factor receptor inhibitors impair alveolar epithelial repair by blocking pathways crucial for lung regeneration, leading to an increased risk of inflammation and fibrosis. Bleomycin induces lung injury through direct oxidative damage and free radical formation, while methotrexate is associated with an acute hypersensitivity pneumonitis. These distinct pathogenic mechanisms frequently converge on similar clinical phenotypes; including cough, dyspnoea, and radiologic patterns like organising pneumonia; making mechanistic insight essential to improve diagnostic precision and therapeutic choice. In this context, proper histopathological evaluation in selected cases should be considered, as it can provide clinicians with complementary insights of critical importance for improving patient care.

Risk factors for IR-ILD

Several well-recognised factors increased the risk of IR-ILD following ICI. These include a smoking history, pre-existing (often undiagnosed) lung disease such as COPD, idiopathic pulmonary fibrosis or other fibrotic lung diseases, interstitial lung abnormalities, lung cancer, prior thoracic radiotherapy, treatment with PD-1 inhibitors (*e.g.* nivolumab, pembrolizumab) or PD-L1 inhibitors (*e.g.* atezolizumab, durvalumab) and the use of PD-(L)1/CTLA-4 combination immunotherapy. The type and site of the primary cancer and metastases may also play a role. Poor performance status (ECOG$\geqslant$2) has also been associated with a greater susceptibility to immune-related adverse events. In rare cases, ICIs may unmask previously undiagnosed autoimmune rheumatic diseases in which ILD may be the dominant or sole manifestation. Despite this, screening for and documentation of known risk factors is inconsistent across clinical trials. In many cases, pre-existing pulmonary disease is either undiagnosed or underestimated, leading to an increased likelihood of complications once ICI therapy has been initiated.

Gaps in pre-therapy evaluation and documentation empower diagnostic ambiguity

The absence of clear guidelines for pre-treatment evaluation results in missed opportunities for early intervention, limits shared decision-making on risk mitigation and hinders the large-scale utilisation of data.

Another significant challenge in the early and accurate diagnosis of IR-ILD is the presentation overlap with other pulmonary conditions. Symptoms such as dyspnoea and cough are non-specific and often attributed to different aetiologies, including infectious pneumonia, radiation pneumonitis, acute exacerbation of airway diseases, heart failure, and cancer progression [3]. High-resolution computed tomography (HRCT) findings can be similarly ambiguous, with radiologic patterns similar to those seen in organising pneumonia, non-specific interstitial pneumonia, and acute interstitial pneumonia, which may have other aetiologies [8–10]. The lack of specificity in both clinical presentation and imaging underscores the need for a standardised diagnostic and risk evaluation framework.

The case for building a framework: standardised risk stratification, diagnostic criteria and evaluation algorithm

Despite increasing awareness of IR-ILD post-ICIs, good clinical practice is hampered by the lack of agreed pre-therapy risk stratification and clear diagnostic criteria for IR-ILD. We identify an urgent need for a straightforward, systematic approach to pre-treatment risk assessment and standardised diagnostic criteria to

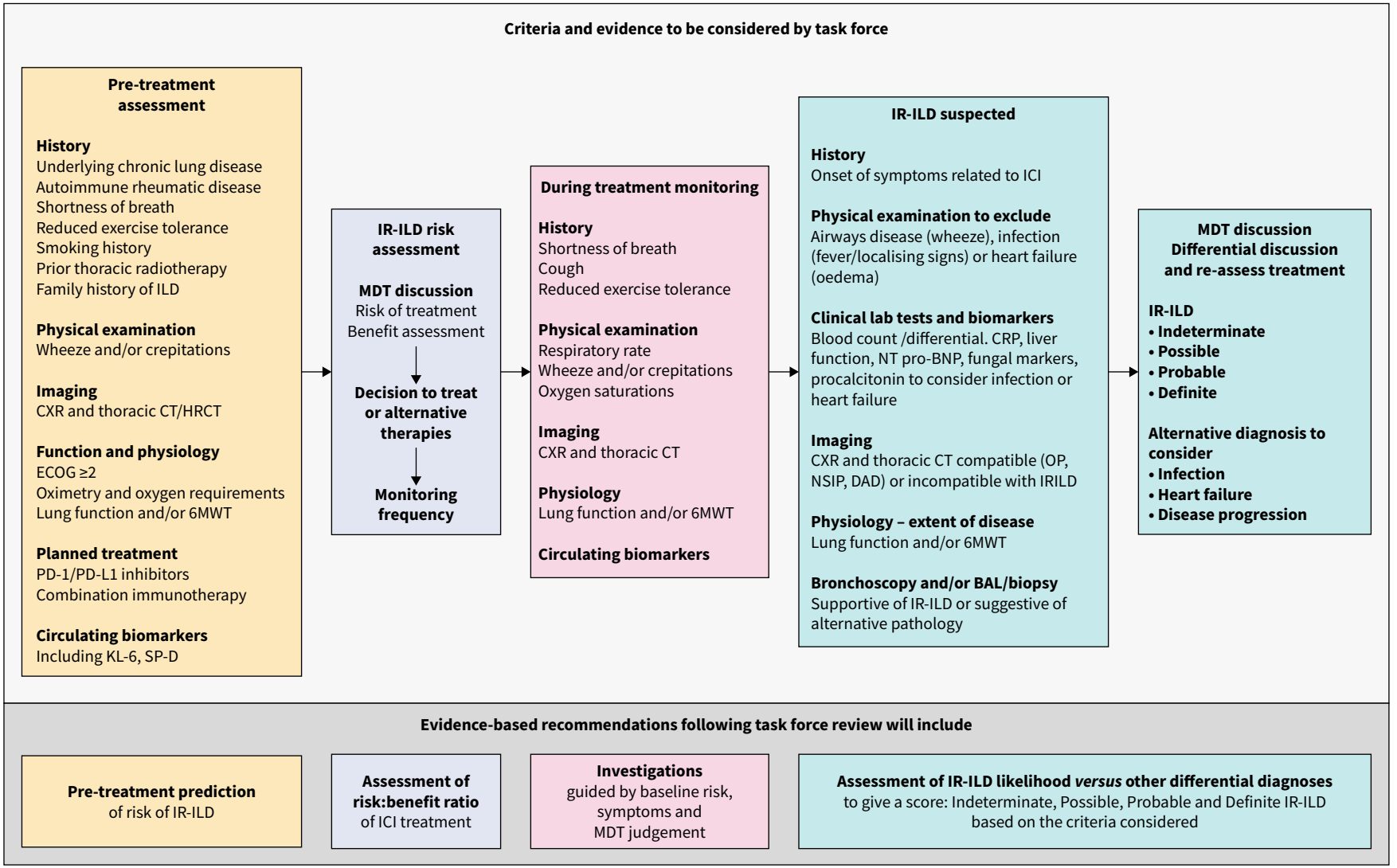

FIGURE 1 Flow diagram on the evidence-based recommendations that this task force will focus on. 6MWT: 6-min walk test; BAL: bronchoalveolar lavage; CRP: C-reactive protein; CT: computed tomography; CXR: chest radiograph; DAD: diffuse alveolar damage; ECOG: Eastern Cooperative Oncology Group; HRCT: high-resolution CT; ICI: immune checkpoint inhibitors; ILD: interstitial lung disease; IR-ILD: immunotherapy-related ILD; MDT: multidisciplinary team; NSIP: non-specific interstitial pneumonia; NT pro-BNP: N-terminal pro-brain natriuretic peptide type B; OP: organising pneumonia; PD-1: programmed cell death receptor 1; PD-L1: programmed cell death ligand 1.

enhance early detection, improve patient care, and strengthen research quality. Of utmost importance, the systematic approach to address the differential diagnosis excluding other conditions like heart failure or lung infection must be carefully proposed to avoid misdiagnosis. For this purpose, bronchoscopy evaluation with specific viral and bacterial testing needs to be considered as well as a specific cardiac evaluation in dedicated cases. Moreover, the lack of standardised pre-treatment evaluation and diagnostic criteria for IR-ILD impacts patient care and research. It causes variability in diagnosis, making it difficult to determine the true incidence, assess treatments, or validate biomarkers. Unnecessary discontinuation of ICI can also harm patients. Clear, harmonised guidelines would improve research reliability and enable better patient selection in clinical trials and improve outcomes.

To address these points, we have established a multidisciplinary task force, endorsed by the European Respiratory Society, to systematically review the evidence and develop a consensus-based, evidence-informed algorithm for pre-therapeutic evaluation. This approach is likely to include elements of clinical risk assessment, pulmonary function testing, HRCT, and biomarker/autoantibody profiling. Clinical evaluation could involve a thorough oncologic and respiratory history, with an assessment of smoking and other relevant exposures, including therapeutic drugs that may impact lung function. Baseline pulmonary function tests, including carbon monoxide transfer factor, might identify the presence and severity of pre-existing COPD or ILD, estimate respiratory reserve, and assess an individual's ability to tolerate IR-ILD episodes. HRCT remains the gold standard for evaluating ILD; dedicated thin-slice chest CT scans of at least 1 mm in lung cancer patients may facilitate the early detection of pre-existing ILDs prior to ICI treatment. Ongoing research suggests that serological markers, such as Krebs von den Lungen-6 (KL-6), surfactant proteins (SP-A, SP-D), and inflammatory cytokines like interleukin-1, may help identify early subclinical pulmonary injury, although specificity may be limited. If available from diagnostic biopsies, a review of resected lung specimens may show early fibrosis. There are, for now, no routine genetic tests predicting the risk of IR-ILD, but this might change since genetic background is a significant risk factor for both cancer and ILD.

In figure 1, we propose a four-group classification of IR-ILD. "Definite" refers to patients with a diagnostic probability $\geqslant$90% (proven by biopsy, with a timely presentation and no confounding factors). "Probable" corresponds to a diagnostic probability of 70–90%, characterised by a timely and typical HRCT presentation without confounding factors. "Possible" denotes a diagnostic probability of 50–70%, defined by a timely or non-timely atypical presentation without confounding factors. "Indeterminate" applies to cases with a diagnostic probability <50%, characterised by atypical presentations (timely or not) in the presence of one or more confounding factors.

Classification models and biomarker challenges towards safer and smarter immunotherapy

Regarding the challenge of the non-specificity of symptoms at early manifestations, we propose a classification system for IR-ILD, to be redefined after the evidence review. Such a framework may well define IR-ILD cases as "definite", "probable", "possible", or "indeterminate", based on clinical, radiological, and (when available) histopathological findings. The absence of universally accepted biomarkers further complicates diagnosis. Although markers like KL-6 and SP-D show promise in detecting lung epithelial injury, their ability to distinguish IR-ILD from other ILDs, pulmonary toxicities, or lung cancer remains unclear. Bronchoalveolar lavage analysis, especially for CD8+ lymphocytosis and cytokine profiles, may help differentiate IR-ILD from other conditions. However, bronchoalveolar lavage is not always feasible, particularly in critically ill, acutely deteriorating or frail patients. Given these limitations, there is an urgent need for continued biomarker research to establish reliable, non-invasive tools for the early detection of IR-ILD. Machine-learning algorithms applied to HRCT imaging and multi-omics approaches integrating proteomics and transcriptomics may provide additional avenues to improve diagnostic precision and optimise the benefit-to-risk ratio of proposed treatments. FDG-positron emission tomography/computed tomography might have a role in the risk stratification of IR-ILD development, though it requires further studies [11].

Addressing these gaps will enhance patient safety, reduce unnecessary treatment interruptions, and advance understanding of IR-ILD. While health economics analysis is needed to balance costs and risks, a structured risk stratification and diagnostic approach is vital to optimise outcomes and support research. Standardisation in IR-ILD assessment is crucial for the future of cancer immunotherapy and patient safety.

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