



Lung Changes in Oncologic Treatment

Gina Al-Farra, senior consultant in oncoradiology at
Herlev –Gentofte University Hospital

Agenda

- 1 Introduction: Why Lung Changes Matter in Cancer Care
- 2 Treatment Modalities That Affect the Lung
- 3 Mechanisms of Lung Injury During Oncologic Treatment
- 4 Typical Imaging and Clinical Patterns
- 5 Differential Diagnosis: Treatment Effect vs Disease Progression
- 6 Risk Factors and Monitoring Strategies
- 7 Management of Suspected Lung Toxicity
- 8 Conclusion: Key Takeaways for Clinical Practice

Clinical significance and treatment continuity

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Treatment-related lung injury often begins subtly, yet it can reshape prognosis, symptom burden, and the feasibility of continuing cancer therapy.

Treatment Modalities That Affect the Lung

Therapeutic exposures and pulmonary risk



Radiation therapy

Thoracic irradiation can damage alveolar epithelium, capillaries, and bronchiolar structures, with risk shaped by dose, fractionation, and irradiated volume.



Chemotherapy

Cytotoxic agents may provoke diffuse inflammatory or fibrotic toxicity, particularly when combined with other lung-stressing therapies.



Targeted therapy

Molecular inhibitors can disrupt epithelial repair, endothelial integrity, or signaling pathways, occasionally producing pneumonitis-like syndromes.



Immunotherapy

Checkpoint blockade may unleash immune-mediated lung inflammation, often with radiographic patterns that mimic infection or organizing pneumonia.



Surgery and combined-modality care

Resection, perioperative stress, and sequential treatment can reduce pulmonary reserve and amplify susceptibility to treatment-related injury.

Different oncologic modalities injure the lung through different biologic pathways, making treatment history central to interpretation.

Mechanisms of Lung Injury During Oncologic Treatment

Biologic cascade from acute damage to chronic remodeling



Lung toxicity evolves through a sequence of epithelial injury, immune activation, and architectural remodeling that may culminate in fibrosis.



Direct epithelial damage

Initial injury to type I and type II pneumocytes impairs barrier function, surfactant homeostasis, and reparative capacity.



Inflammatory cascade

Cytokine and chemokine signaling recruits inflammatory cells, amplifying edema, capillary leak, and tissue dysfunction.



Immune-mediated pneumonitis

Dysregulated antitumor immunity can target normal lung tissue, producing symptomatic pneumonitis with variable tempo and severity.



Fibrotic remodeling

Persistent injury drives fibroblast activation and extracellular matrix deposition, leading to reduced compliance and restrictive physiology.

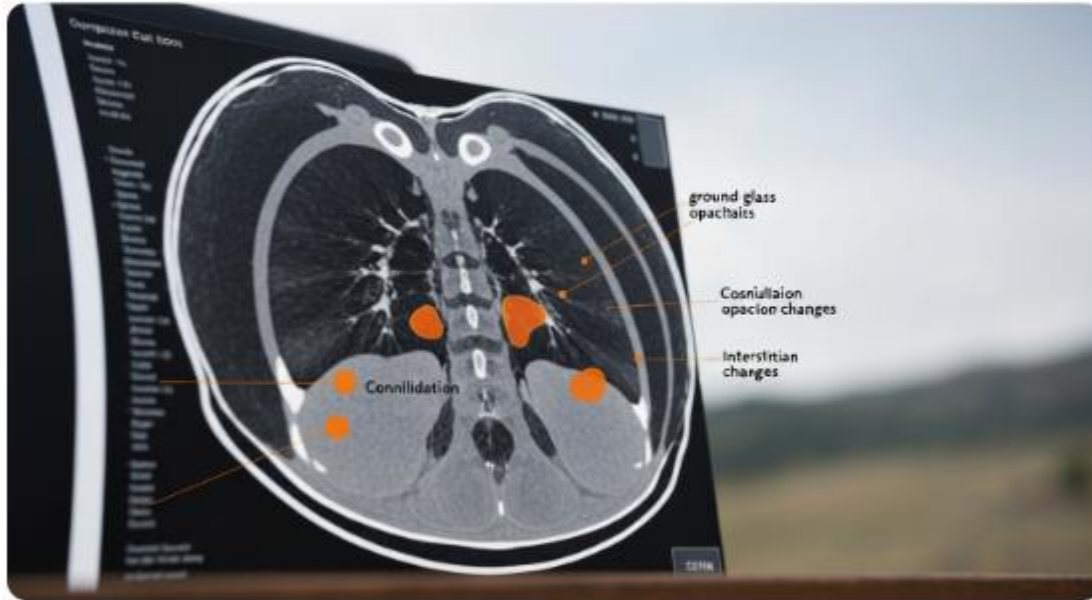


Vascular and alveolar disruption

Microvascular injury and alveolar-capillary damage impair gas exchange, worsening diffusion limitation and hypoxemia.

Typical Imaging and Clinical Patterns

Radiographic phenotypes that recur across therapies

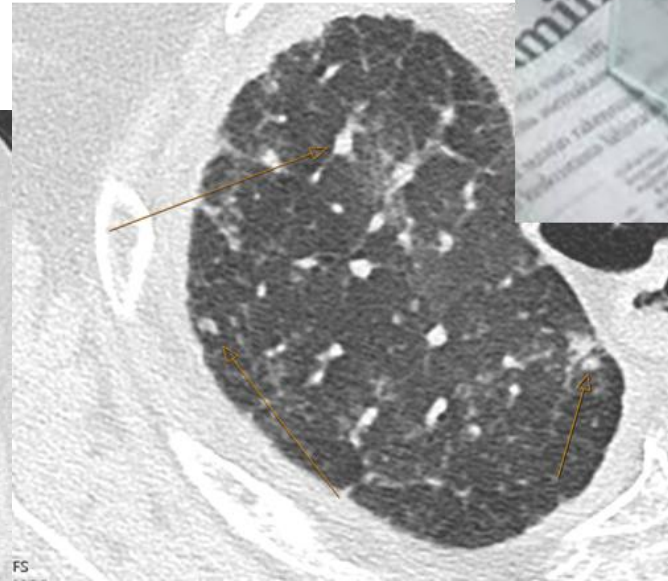


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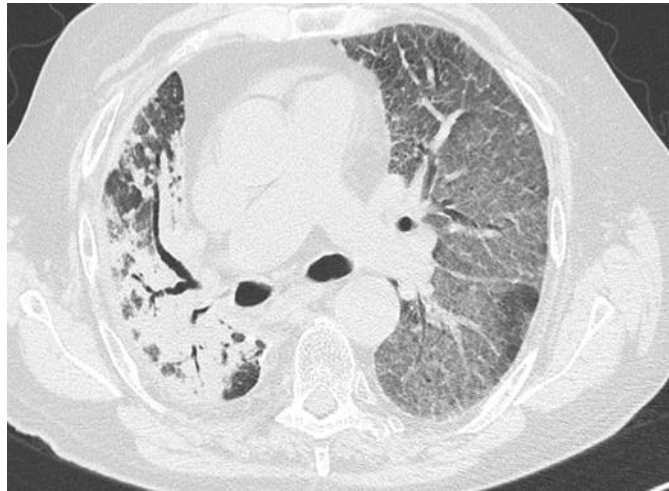
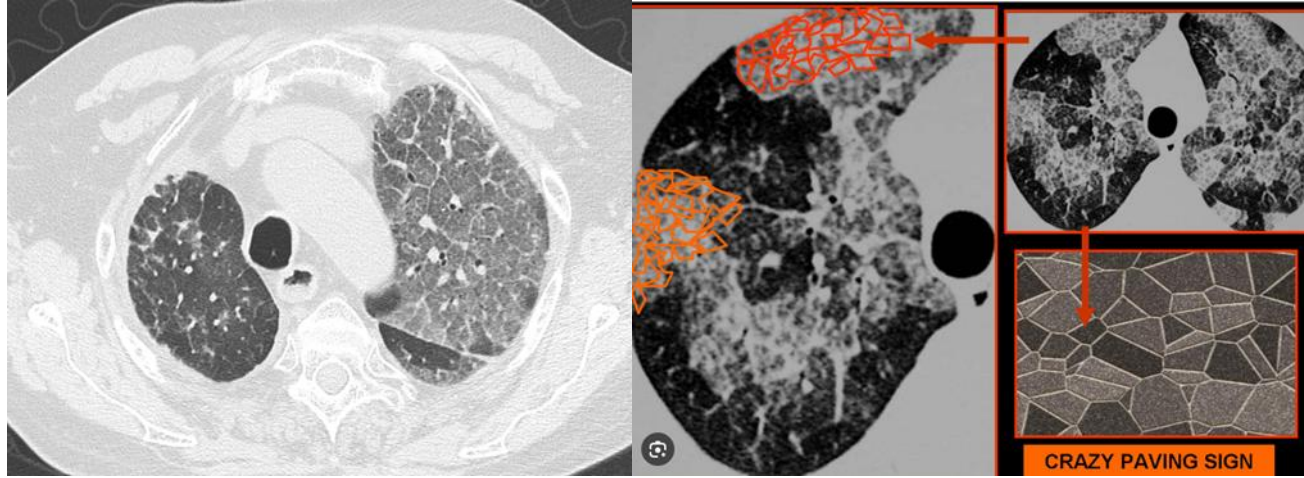
Imaging is pattern-based: distribution, density, and timing together distinguish expected treatment effects from alternative diagnoses.

- **Ground-glass opacities:** This common early pattern reflects partial airspace filling or interstitial inflammation and may be patchy, bilateral, or treatment-field limited.
- **Consolidation:** Denser alveolar filling suggests more advanced inflammatory injury, infection, or organizing response depending on context and timing.
- **Interstitial changes:** Reticulation, septal thickening, and architectural distortion raise concern for evolving interstitial injury or fibrotic remodeling.
- **Organizing pneumonia pattern:** Peripheral or peribronchovascular opacities can migrate over time, often mimicking infection but responding to anti-inflammatory treatment.
- **Radiation-field distribution:** Conformity to the radiation portal strongly supports treatment effect, especially when changes sharply respect geometric treatment boundaries.

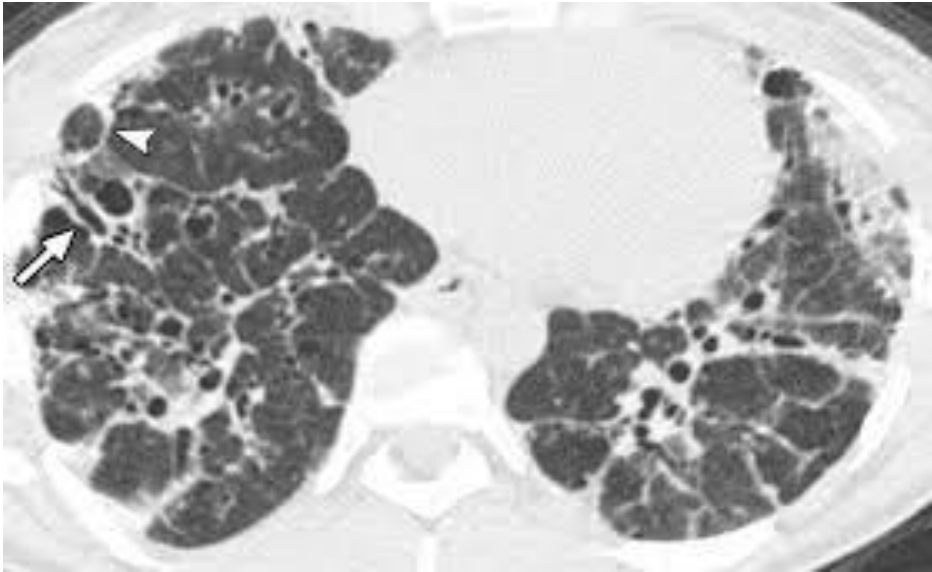
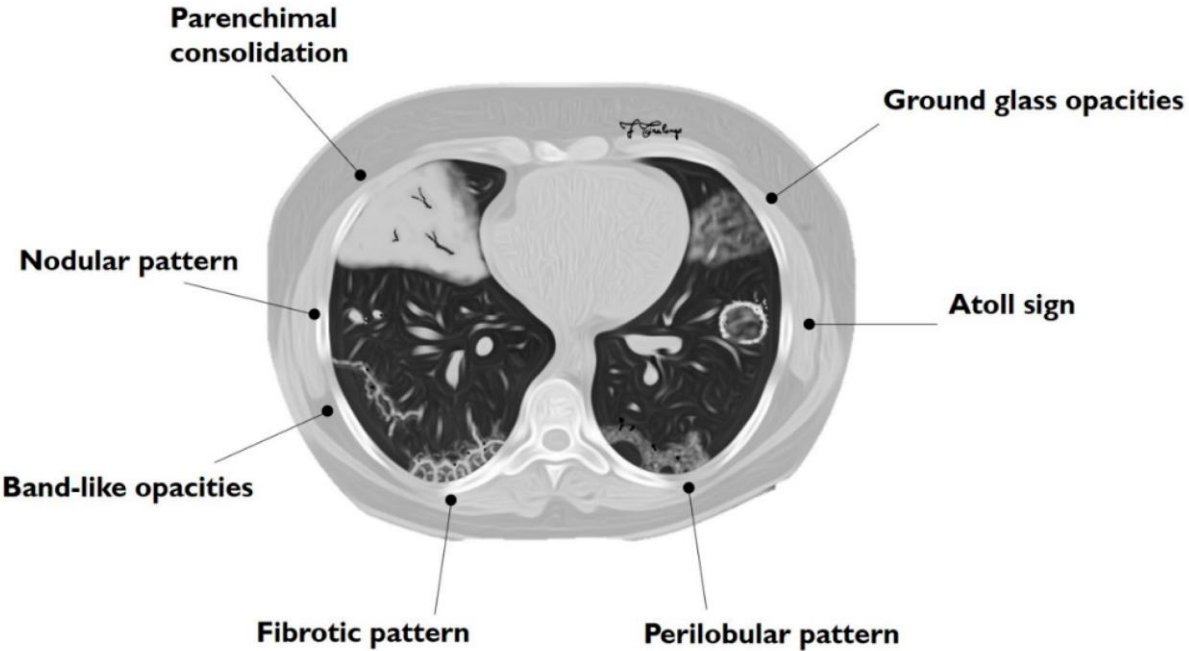
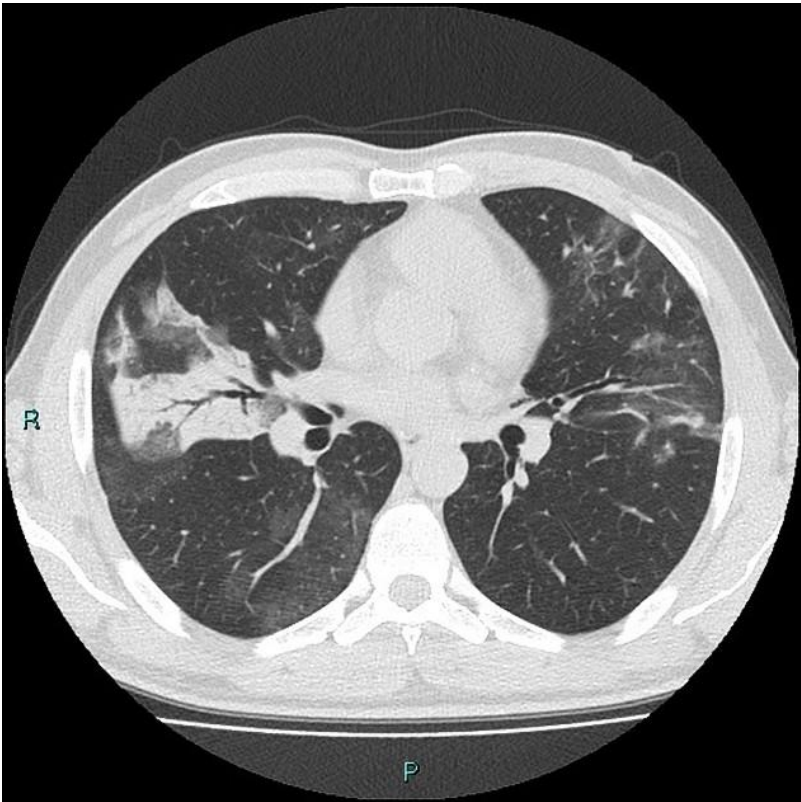
Ground-glass opacities



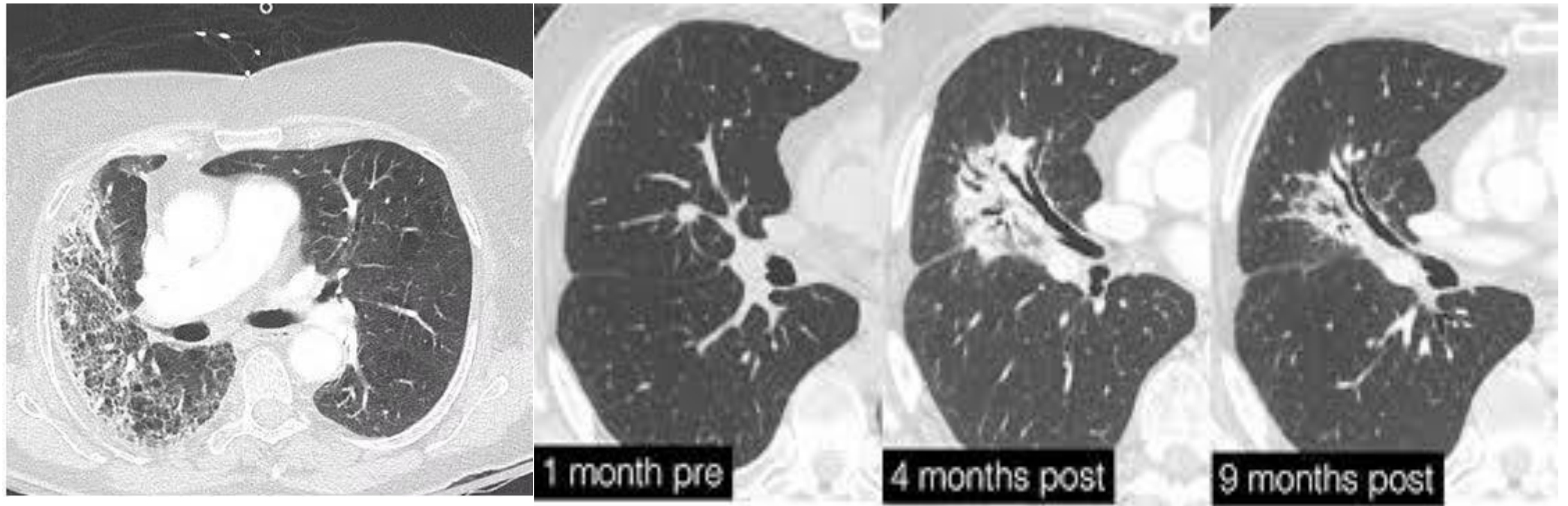
Interstitial changes-crazy paving,
consolidation ,

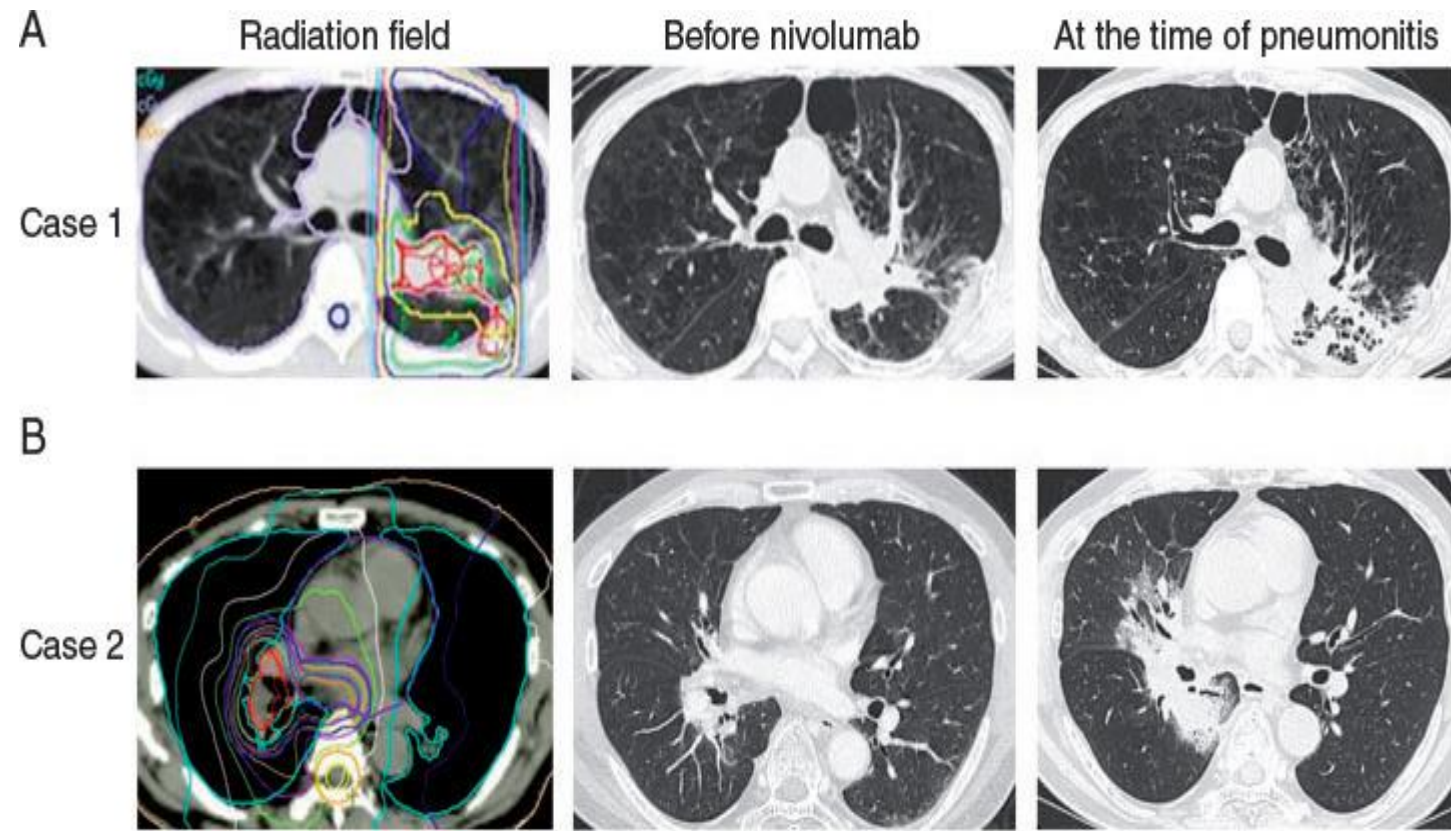
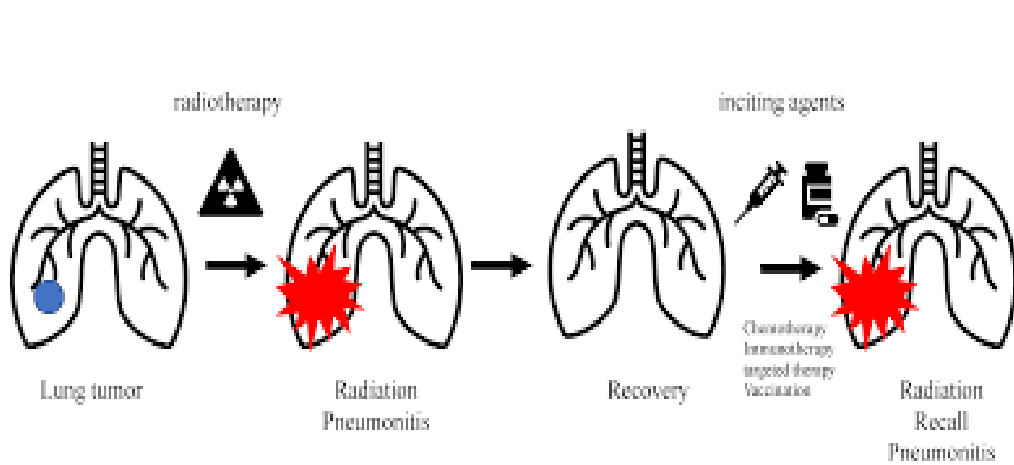


Organizing pneumonia pattern



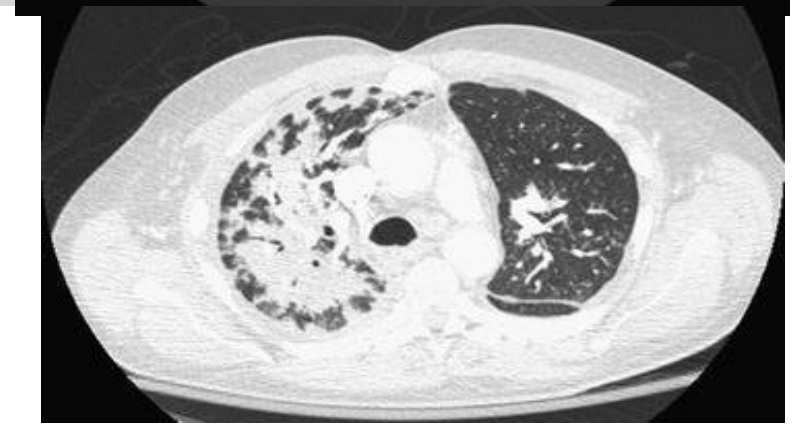
Radiation-field distribution



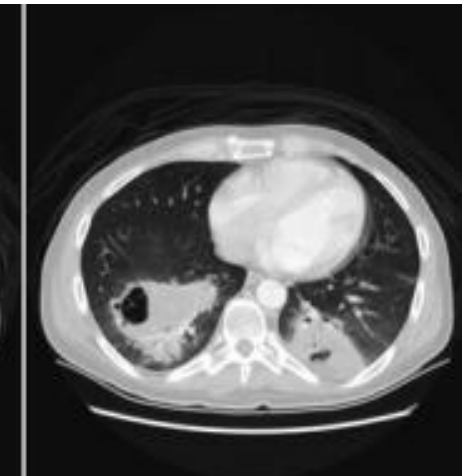
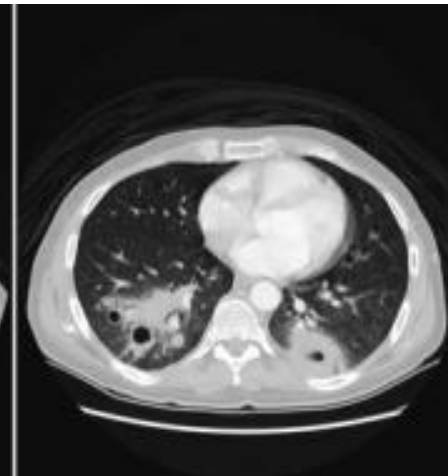


Radiation recall pneumonitis

(RRP) is a rare but severe condition which has been mainly detected in the previously irradiated lung of patients with cancer after using inciting agents, most commonly antineoplastic regimens including chemotherapy, targeted therapy, or immunotherapy.



Lung infections are a leading cause of morbidity and mortality for cancer patients. Because tumors and treatments (like chemotherapy and radiation) compromise the immune system, patients are highly vulnerable to bacterial, viral, and fungal pathogens

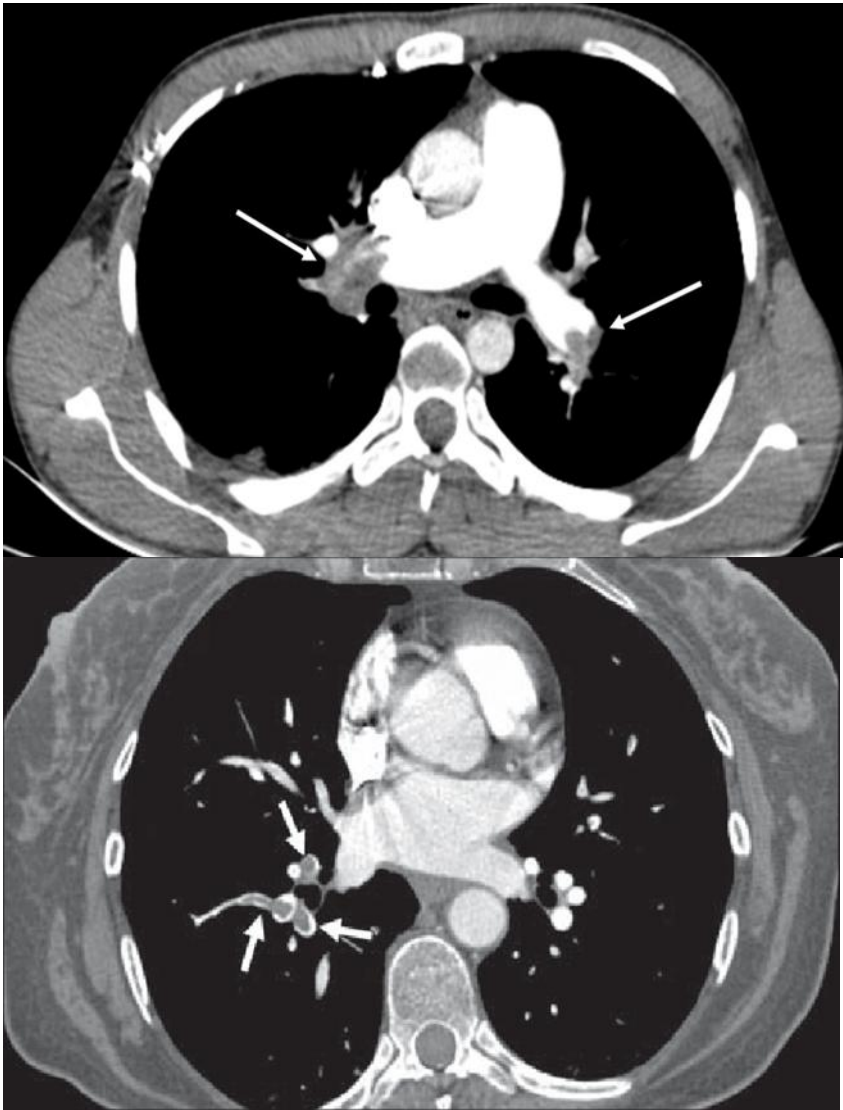
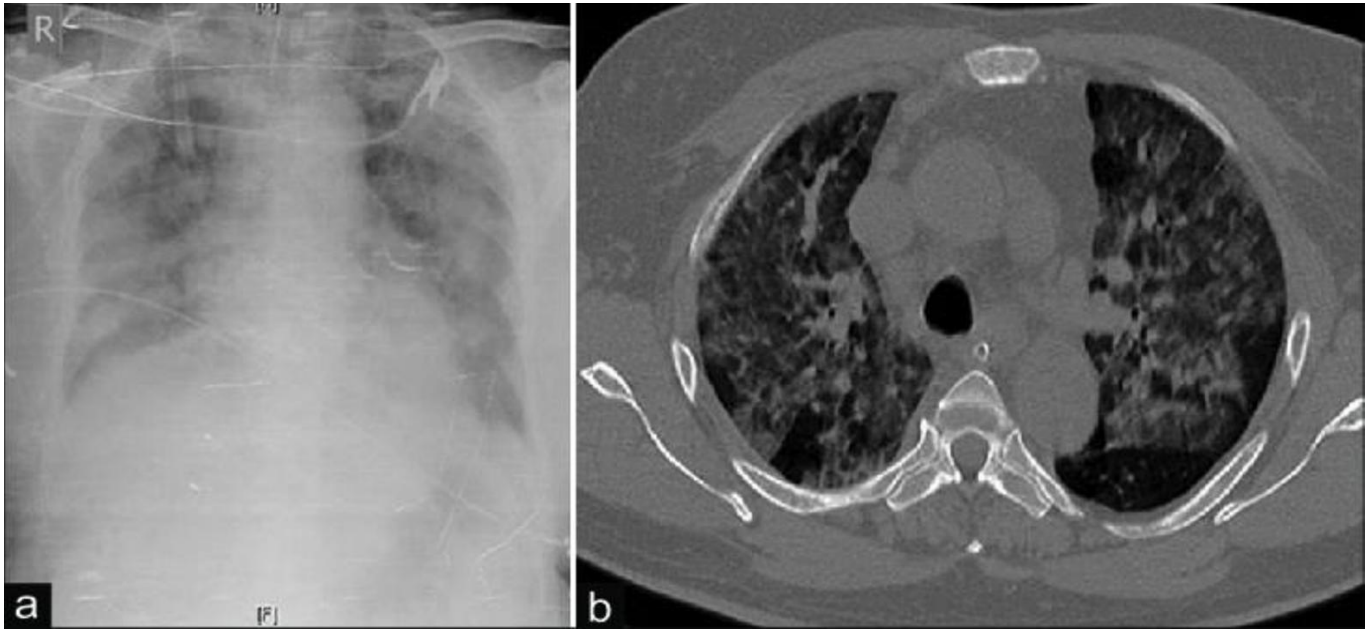


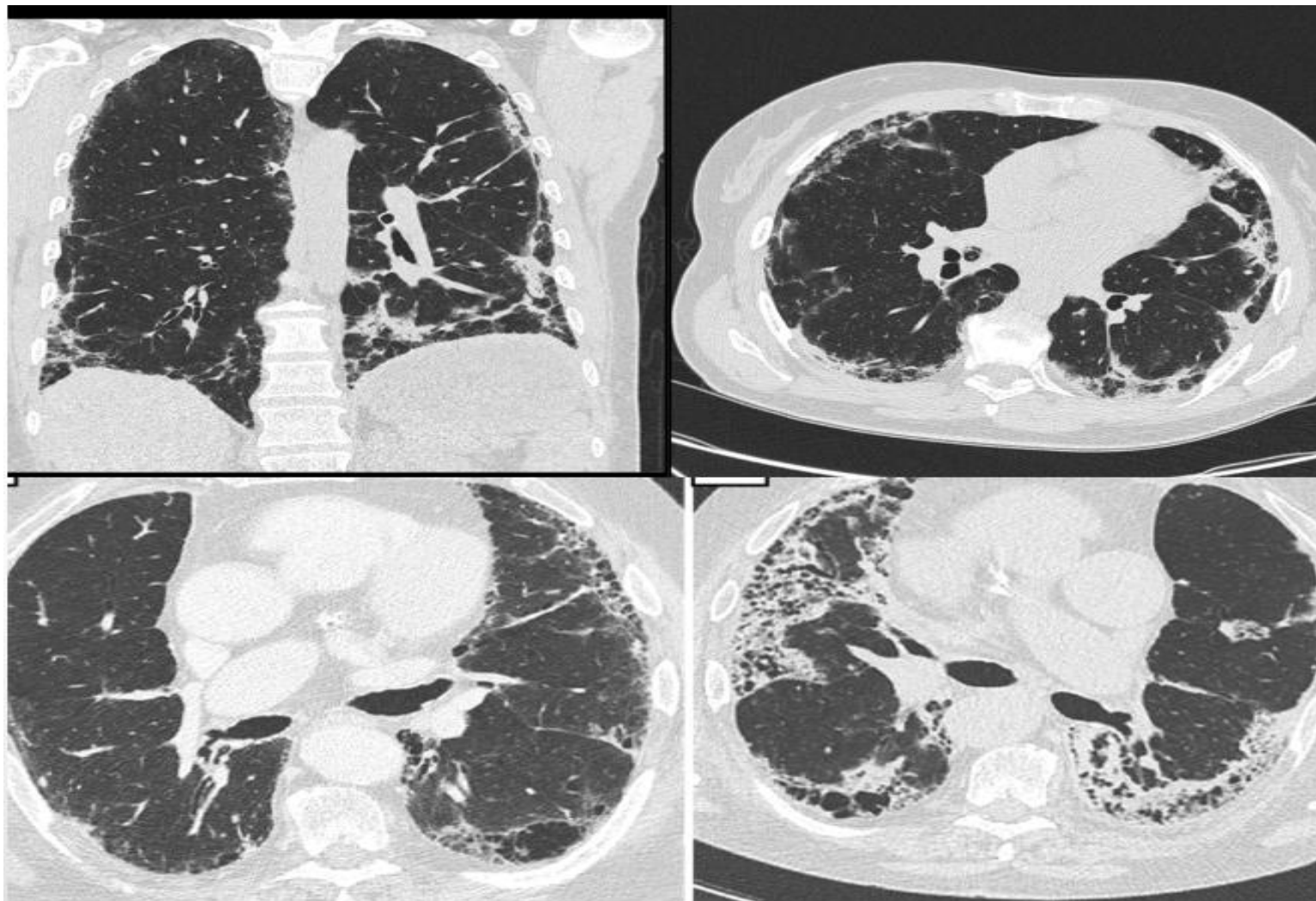
COPENHAGEN



Granulomatous activation in cancer patients -formation of granulomas—clusters of activated immune cells—either within a tumor, in draining lymph nodes, or systemically. It typically represents either a robust anti-tumor immune response or a reaction to specific cancer treatments, though it must be carefully distinguished from infections or metastasis

Vascular complications in cancer patients arise from a dangerous triad: pro-thrombotic tumor states, vascular-damaging therapies (e.g., VEGF inhibitors), and baseline risk factors. They commonly manifest as venous thromboembolism (VTE), acute arterial bleeding, accelerated atherosclerosis, and endothelial dysfunction





Differential Diagnosis: Treatment Effect vs Disease Progression

Concise clinical differentiation matrix

Condition	Typical timing	Imaging clues	Key clinical clues
Infection	Days to weeks; often abrupt	Focal or multifocal consolidation, air bronchograms, tree-in-bud, cavitation in selected settings	Fever, leukocytosis, sputum, neutropenia, microbiologic exposure
Tumor progression	Subacute to progressive; often after prior response	Enlarging mass, new nodules, lymphangitic spread, pleural or mediastinal extension	Rising tumor markers, worsening cancer burden, poor response to prior therapy
Pulmonary edema	Acute or peri-infusion; can follow fluid overload	Bilateral perihilar opacities, septal thickening, pleural effusions, cardiomegaly	Volume overload, cardiac dysfunction, weight gain, elevated BNP
Thromboembolism	Sudden onset; often unexplained	Segmental perfusion defects on CT angiography, infarct-related wedge opacity	Pleuritic pain, tachycardia, hypoxemia, VTE risk factors
Drug toxicity	Variable, often after cumulative exposure or new agent	Bilateral ground-glass change, organizing pneumonia, diffuse interstitial pattern, field-restricted change	Temporal link to therapy, lack of infection source, improvement with drug hold or steroids

Clinical differentiation depends on timing, imaging distribution, and bedside clues, because treatment toxicity often overlaps with common oncologic complications.

Risk Factors and Monitoring Strategies

Identifying patients who need closer surveillance



Baseline lung disease

COPD, interstitial abnormalities, prior smoking injury, or reduced reserve lower the threshold for clinically meaningful toxicity.



Cumulative dose and field size

Higher delivered dose and larger irradiated volume increase the likelihood of tissue injury and late fibrosis.



Concurrent therapies

Overlapping pulmonary toxicity from combination regimens can magnify risk through additive inflammatory or cytotoxic mechanisms.



Prior thoracic irradiation

Re-irradiation or previously injured lung tissue may respond unpredictably, with greater susceptibility to decompensation.



Symptoms and pulmonary function trends

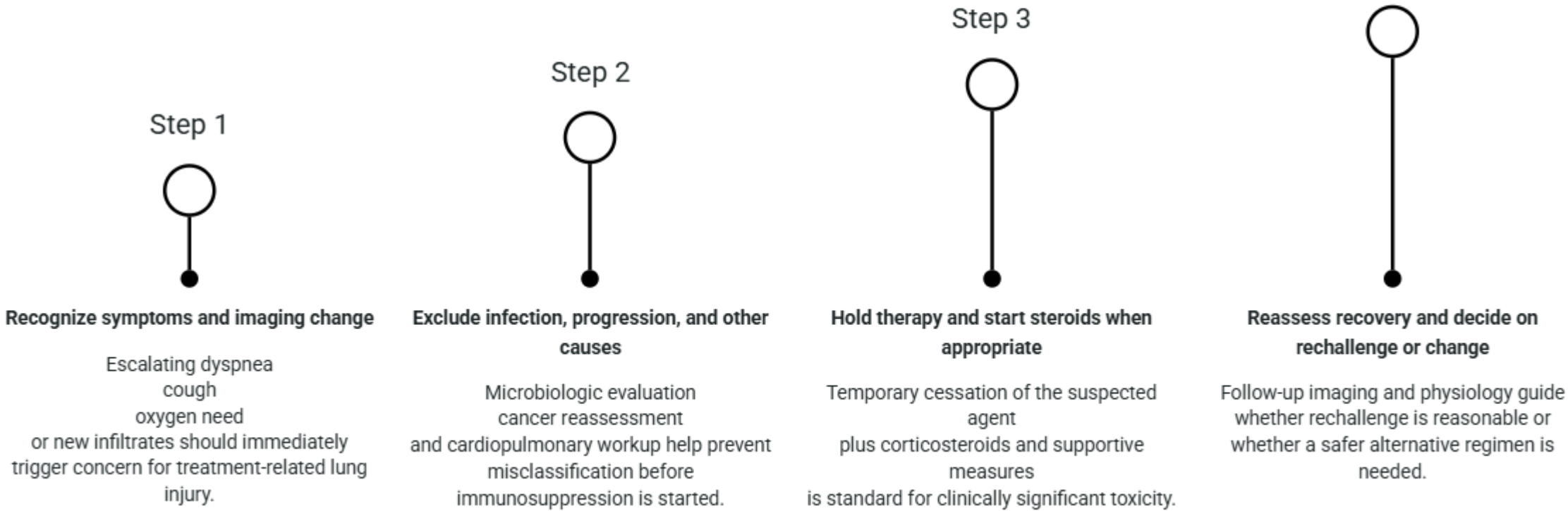
Serial dyspnea assessment, exertional tolerance, diffusion capacity, and spirometry often detect decline before severe hypoxemia develops.

Risk assessment is strongest when baseline vulnerability, treatment intensity, and dynamic physiologic trends are interpreted together.

Management of Suspected Lung Toxicity

Stepwise response to emerging pulmonary adverse events

Management succeeds when the culprit therapy is paused early, alternative causes are excluded, and anti-inflammatory treatment is targeted to severity.



Conclusion: Key Takeaways for Clinical and Radiology Practice

High-yield principles for frontline care

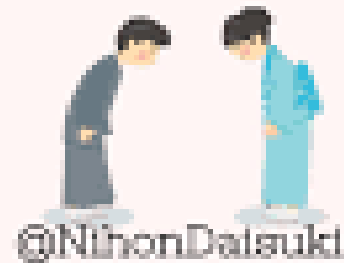
- **Early recognition is critical:** Small changes in symptoms or imaging can precede major respiratory compromise, so clinicians should react before deterioration accelerates.
- **Patterns are treatment-specific:** The most likely diagnosis depends heavily on the therapeutic exposure, elapsed time, and distribution of radiographic abnormalities.
- **Differential diagnosis is essential:** Infection, edema, progression, embolism, and drug toxicity must be separated systematically because treatment implications differ profoundly.
- **Management requires multidisciplinary care:** Optimal decisions often require oncology, pulmonology, radiology, infectious disease, and radiation oncology collaboration.

The practical message is simple: timely recognition, careful exclusion of alternatives, and multidisciplinary management preserve both safety and oncologic intent.

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Kiite kurete arigatou

THANK YOU FOR LISTENING.



THANK YOU
FOR YOUR
ATTENTION