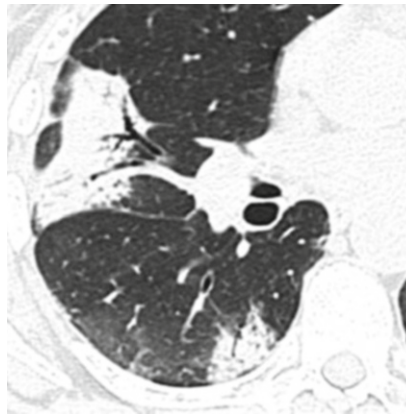
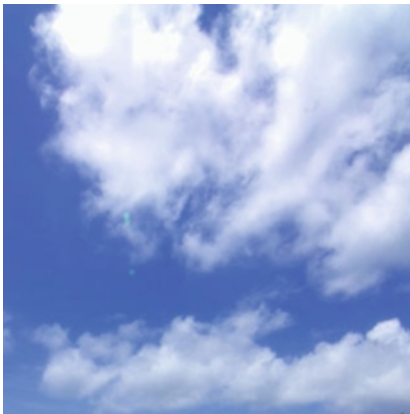


# Alveolar Pattern

Radiology  
Pathology

**Giorgia Dalpiaz**  
**Alessandra Cancellieri**



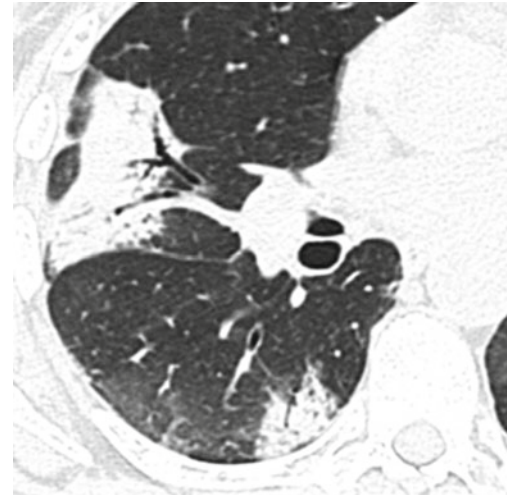
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**Definition****ALVEOLAR PATTERN**

An alveolar pattern is defined by the existence of more or less broad portions of the lung more opaque than normal due to partial or complete alveolar filling. With a few exceptions, the pulmonary architecture is overall preserved, and, if signs of interstitial involvement are present, they are not prevalent. On HRCT the different opacity of the alveolar pattern reminds the variable density of the clouds.



Air-space-filling pattern, cloudy opacities



Why are some clouds white and others gray? The color depends on a physical characteristic called reflectance, which indicates the percentage of light that is reflected from the cloud. In general, denser clouds have a reflectance of 90% and are thus light in color.



In alveolar diseases, this pattern is predominant; however, there are other diseases in which alveolar opacities may be found, albeit less important or sporadic. They are therefore described in the relevant chapters.

The HRCT key signs are:

- Ground-glass opacity
- Consolidation

The ancillary signs are:

- Hypodense consolidation
- Hyperdense consolidation
- Cystic consolidation
- Crazy paving
- Head-cheese sign
- Reversed halo sign (atoll sign)
- Perilobular pattern
- Lobular/sublobular consolidation and GGO
- Tree-in-bud sign, bronchiolar

The prevalent distribution of the signs, together with the presence of non-parenchymal signs, may be helpful for the diagnosis of a specific disease (please see the tables at the end of this chapter).

## Ground-Glass Opacity (GGO)

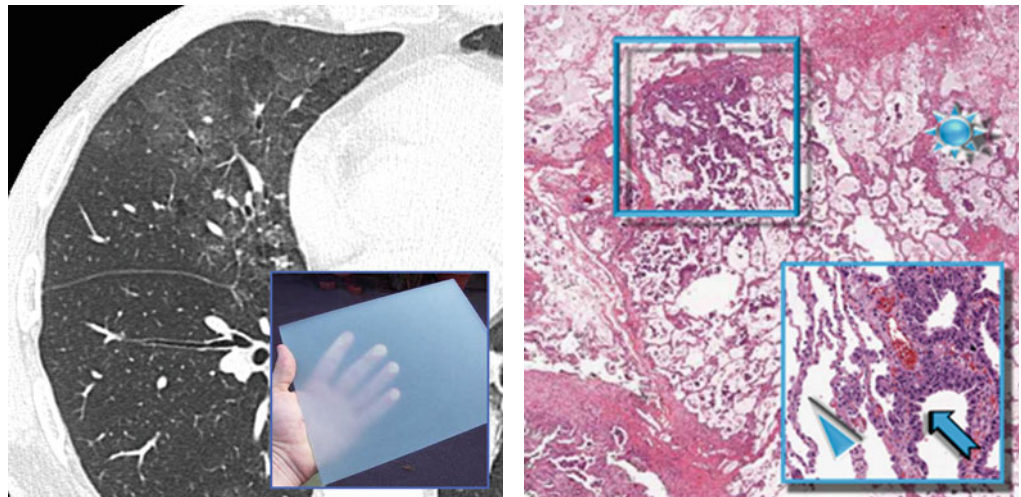


### ALVEOLAR KEY SIGNS

On CT scans, ground-glass opacity (GGO) appears as hazy increased opacity of the lung, with preservation of bronchial and vascular margins. Ground-glass opacity is less opaque than consolidation, in which bronchovascular details are obscured. The final effect is similar to the ground glass inserted in windows and doors (please see the left image below).

Pathologically, it may be caused by partial filling of air spaces and/or interstitial (mainly, alveolar septal) thickening due to fluid and/or cells. An example, illustrated in the figures below, is mucus in pneumonia-like pattern of mucinous adenocarcinoma. There are both a diffuse partial filling of air spaces due to mucus (★) and the lepidic growth of the neoplasm along alveolar septa (□). In the close-up, note the comparison between the different thickening of the normal alveolar septa (▶) and the pathological ones (➡). Please also refer to the chapter entitled “Thinking Through Pathology”.

GGO



GGO may also be due to partial collapse of alveoli, increased capillary blood volume, or a combination of these, the common factor being the partial displacement of air.

GGO may also be due to interstitial fibrosis. It is variously associated with traction bronchiectases and bronchiolectases, fibrotic reticular abnormalities, and volume loss (please also refer to chapter “[Fibrosing Pattern](#)”).



Dark bronchogram sign (“overly good” visualization of bronchial structures within areas of ground-glass opacity) is a helpful mark to recognize minimal diffuse GGO.



GGO can be seen in all patients with an underlying alveolar lung disease, so its diagnostic value in isolation is limited; however, the clinical context (*acute* or *chronic*), together with the distribution and in combination with associated signs, may be helpful (please see the tables at the end of this chapter).



Engeler CE (1993) Ground-glass opacity of the lung parenchyma: a guide to analysis with high-resolution CT. *AJR Am J Roentgenol* 160(2):249

Hansell D (2008) Fleischner Society: glossary of terms for thoracic imaging. *Radiology* 246(3):697

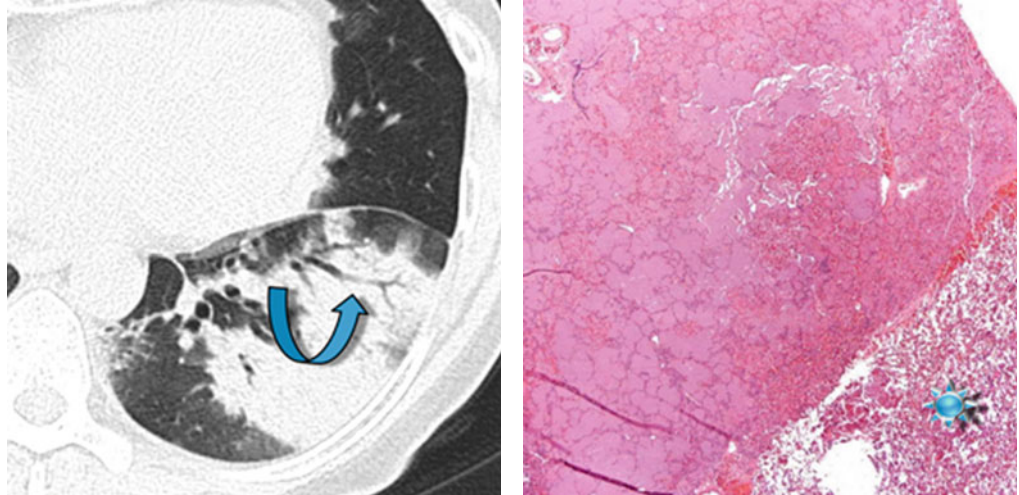
Miller WT Jr (2005) Isolated diffuse ground-glass opacity in thoracic CT: causes and clinical presentations. *AJR Am J Roentgenol* 184(2):613

## Consolidation

Consolidation appears as a homogeneous increase in pulmonary parenchymal attenuation that obscures the margins of vessels and airway walls. An air bronchogram may be present (↘).

Pathologically, consolidation is due to a complete filling of alveoli by any material (exudate, cells, or other disease product can cause a similar radiological aspect) – the common factor being the full displacement of air from alveoli. An example, illustrated in the figure below, is pulmonary alveolar proteinosis (PAP).

Note the abrupt transition between the area in which alveoli are filled by proteinaceous material and the normal lung (★). Please also refer to the chapter entitled “Thinking Through Pathology >Pattern >Alveolar Filling.”



Consolidation can be seen in all patients with an underlying alveolar lung disease, so its diagnostic value by itself is limited; however, the clinical context (*acute* or *chronic*) and the distribution may be helpful together with the associated signs (please see the tables at the end of this chapter).



Hansell D (2008) Fleischner Society: glossary of terms for thoracic imaging. *Radiology* 246(3):697

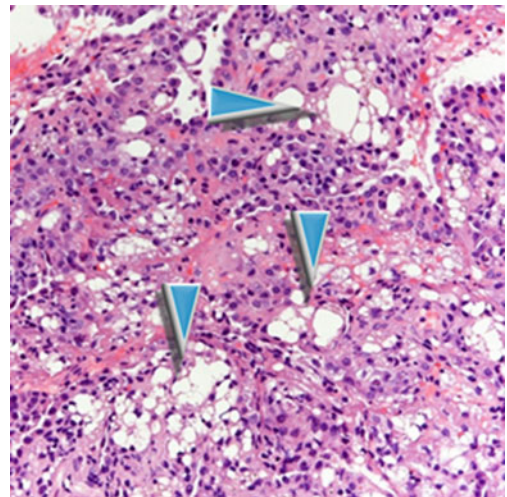
## Hypodense Consolidation

### ANCILLARY SIGNS

Using mediastinal window, an attenuation of consolidation is defined hypodense when it is lower than the muscle. It may be visible on CT after intravenous contrast material administration (please see CT image below). Sometimes the hypodense consolidation may be visible on unenhanced CT (HRCT) due to very low-density material (e.g., fat ► in lipoid pneumonia). Hypodense consolidations can result from ischemia (lung infarction) or from the presence in the air space of mucinous neoplasms (primary or metastatic mucinous adenocarcinoma), mucus (obstructive pneumonia with abundant accumulation of secretions), necrosis (necrotizing pneumonia), or fat (lipoid pneumonia, see the images below). With the exception of infarct, on enhanced CT you can also find the so-called angiogram sign, i.e., the visualization of pulmonary vessels within an airless, low-attenuation consolidation (please see CT image below). Please also refer to angiogram sign in “Case-Based Glossary with Tips and Tricks”.



Low-attenuation consolidation, low-density consolidation



Fat-containing hypodense consolidation shows CT-negative Hounsfield units (values between  $-150$  and  $-30$  HU).

Hypodense consolidation is recognizable only with CT mediastinal window, and it is more visible on contrast enhancement CT (see the figure above).



Diseases with hypodense consolidation:

- *Lipoid pneumonia*: fatty consolidation ( $-150$  and  $-30$  HU) with dependent distribution (please see the figures above); patchy GGO and crazy paving often coexist.
- *Mucinous adenocarcinoma, primary or metastatic*: low-density consolidation with possible air-filled cystic spaces (please also refer to bubble-like sign in “Case-Based Glossary with Tips and Tricks”); patchy GGO often coexists.
- *Obstructive pneumonia*: crucial is the visibility of abundant accumulation of secretions inside the airways.
- *Lung infarction*: contrast enhancement CT shows filling defects within the pulmonary vasculature with acute pulmonary emboli.
- *Necrotizing pneumonia*: progression to a necrotizing pneumonia can occur from either virulence factor of the microorganism, predisposing factors of the host, or both. It can result from a large number of pathogens, mostly bacteria. Normal pulmonary parenchymal architecture within the necrotic segments is often lost.



MRI with “water-sensitive” sequences is useful in the diagnosis of patients with pulmonary consolidations suspected to be mucinous adenocarcinoma.



Betancourt SL (2010) Lipoid pneumonia: spectrum of clinical and radiologic manifestations. *AJR Am J Roentgenol* 194(1):103

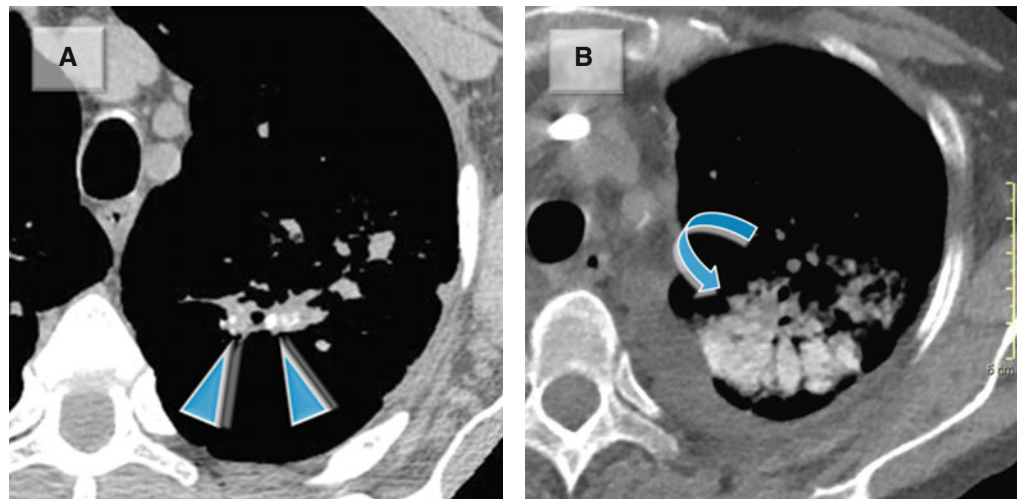
**Hyperdense Consolidation**

Gaeta M (2012) MRI differentiation of pneumonia-like mucinous adenocarcinoma and infectious pneumonia. *Eur J Radiol* 81(11):3587

Attenuation higher than the muscle seen as dense/calcified diffuse or focal pulmonary opacities can result from a variety of different conditions. They can be due to deposition of calcium or, less commonly, other high-attenuation material such as talc, amiodarone, iron, mercury, iodinated substances, and barium sulfate. Small and *focal*, hyperdense opacities inside a consolidation can be secondary to dystrophic calcifications in previously damaged lung parenchyma or, most commonly, as a result of infection diseases (▶).



High-attenuation consolidation, calcified consolidation



Diseases with multiple small *focal* hyperdense opacities inside the consolidation (Figure A above):

- *Tuberculosis*: parenchymal focal calcifications are frequently seen in tuberculosis (▶). A sequela of dystrophic calcification follows caseation, necrosis, or fibrosis. These nodules are seen as well-circumscribed parenchymal calcifications with fibrosis. Most patients with pulmonary nodular calcifications secondary to tuberculosis are located in the upper lobes and the upper segments of lower lobes. Calcified hilar or mediastinal lymph nodes often coexist.
- *Amyloidosis*: the diffuse parenchymal pattern is mostly nodular and septal, although patchy consolidations may be seen. Basal and peripheral distribution is the dominant aspect. Areas of consolidations could show calcifications, some of them with punctate aspect. Lymph node enlargement, together with unilateral or bilateral pleural effusions, may be associated findings.
- *Calcified atelectasis*: chronic atelectasis can be rarely seen as calcified consolidation, often with gravity-dependent distribution.
- *Silicoproteinosis*: it may have focal calcifications, usually seen as small punctate calcified foci inside the areas of consolidation. This disease can also show hyperdense, often perihilar masses. Conglomerate masses are usually oval and have irregular borders.

Diseases with *diffuse* hyperdense consolidation (Figure B above):

- “*Metastatic*” *pulmonary calcification (MPC)*: HRCT findings are characterized by high-attenuation consolidations (↪) most marked in the upper zones. Nodules which may contain foci of calcification may coexist.
- *Drug toxicity (amiodarone)*: the association of dense lung air-space consolidations with high density of the liver and spleen is characteristic of amiodarone impregnation. Consolidations are usually peripheral in location. High-attenuation nodules or masses sometimes coexist.
- *Pulmonary alveolar microlithiasis*: it is a rare chronic disease characterized by widespread calcific intra-alveolar concretions within alveolar spaces. In patients with long-standing disease, numerous

## Cystic Consolidation




adjacent hyperdense nodules result in areas of high-attenuation consolidation. Clinical symptoms are usually absent and, when present, are characterized by dyspnea on exertion.

- *Talcosis*: in the late stage of the disease, hyperdense consolidations or confluent perihilar masses may be present. These lesions are similar to those seen in progressive massive fibrosis caused by silicosis.

Marchiori E (2005) Diffuse high-attenuation pulmonary abnormalities: a pattern-oriented diagnostic approach on high-resolution CT. *AJR* 184:273

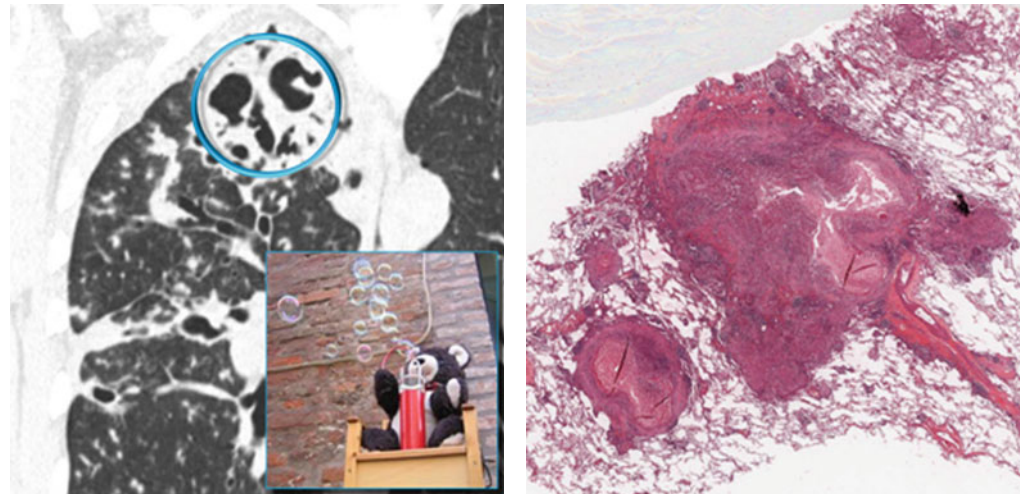
Chan E (2002) Calcium deposition with or without bone formation in the lung. *Am J Respir Crit Care Med* 165:1654

Cysts (black holes) inside the consolidations and the GGO may be large or small, rounded, oval or serpentine, and sometimes confluent.

Pathogenesis: true cavitation (more often round or oval, e.g., TB – please see the HRCT  and histologic images below), pseudo-cavitation (bubble-like lucencies following bronchiolar obstruction due to a check-valve mechanism), and air bronchogram (serpentine or linear in shape due to ectatic phenomena)



Bubble-like lucencies



*Nonneoplastic* diseases with cystic consolidation:

- *Infection* (e.g., *TB*): the presence of cavitated consolidations or nodules in the apical and posterior segments of the upper lobes and/or the superior segments of lower lobes (please see the images above) is suggestive of TB. Another hallmark is hilar/mediastinal lymphadenopathy with possible central necrosis, more visible on contrast enhancement CT.
- *Pulmonary infarct*: cystic features are rarely present, often single unilateral consolidation with basal-peripheral distribution.
- *OP*: although cavitory infiltrates are not usually included in textbook descriptions of the disease, OP presenting with cavitating infiltrates has indeed been described, albeit rather rarely. They often present as multiple bilateral consolidations with basal-peripheral distribution.

*Neoplastic* diseases with cystic consolidation:

- *Adenocarcinoma*: patchy areas of non-resolving consolidation with possible halo sign and often with air bronchogram or air-filled cystic spaces. Possible lower lung predominance (please also refer to bubble-like lucencies in “Case-Based Glossary with Tips and Tricks”).
- *Lymphomas*: cystic aspects are rarely present, possible mass-like aspect.



“Cystic lucencies” can be found in patients with BPCO. This pattern is caused by the coexistence of consolidations and underlying severe centrilobular/paraseptal emphysema.



## Crazy Paving

Gaeta M (1999) Radiolucencies in bronchioloalveolar carcinoma: CT-pathologic correlation. *Eur Radiol* 9:55–9

He H (2006) Pulmonary infarction: spectrum of findings on multidetector helical CT. *J Thorac Imaging* 21(1):1

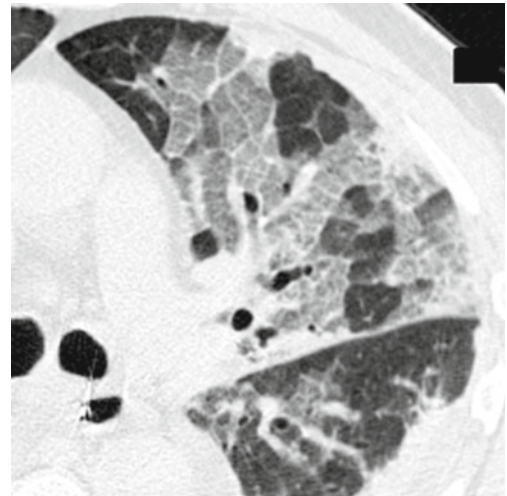
Crazy paving appears as ground-glass attenuation with superimposed interlobular septal thickening and intralobular lines. The crazy-paving pattern is often sharply demarcated from normal lung and may have a geographic outline, often with lobular or geographic sparing. The term crazy paving refers to the resemblance of this sign to paths made with broken pieces of stone or concrete.

In the crazy-paving sign, ground-glass opacity reflects the presence of air space or interstitial abnormalities; the lines of reticular opacities may represent interlobular septal thickening, thickening of the intralobular interstitium, irregular areas of fibrosis, or a preponderance of an air-space-filling process at the periphery of lobules or acini.

Please also refer to crazy paving in “Case-Based Glossary with Tips and Tricks”.



Colonial-era pavement, palladian sign



Acute diseases with crazy paving listed in alphabetic order:

- *Acute interstitial pneumonia (AIP)*
- *Acute respiratory distress syndrome (ARDS)*
- *Acute exacerbation of IPF*
- *Diffuse alveolar hemorrhage (DAH)*
- *Drug-induced pneumonitis*
- *Infection, acute*
- *Pulmonary edema (PE)*

Subacute/chronic diseases with crazy paving listed in alphabetic order:

- *Adenocarcinoma*
- *Chronic eosinophilic pneumonia (CEP)*
- *Lipoid pneumonia (LP)*
- *MALT lymphoma*
- *Nonspecific interstitial pneumonia (NSIP)*
- *Organizing pneumonia (OP)*
- *Pulmonary alveolar proteinosis (PAP)*
- *Radiation pneumonitis*
- *Sarcoidosis, alveolar*
- *Tuberculosis*





A crazy-paving sign can be seen as the dominant pattern only in some of the diseases listed above (e.g., PAP and lipid pneumonia). It often represents an ancillary or uncommon finding.



De Wever W (2011) The crazy-paving pattern: a radiological-pathological correlation. *Insights Imaging* 2(2):117

Rossi SE (2003) "Crazy-paving" pattern at thin-section CT of the lungs: radiologic-pathologic overview. *Radiographics* 23(6):1509

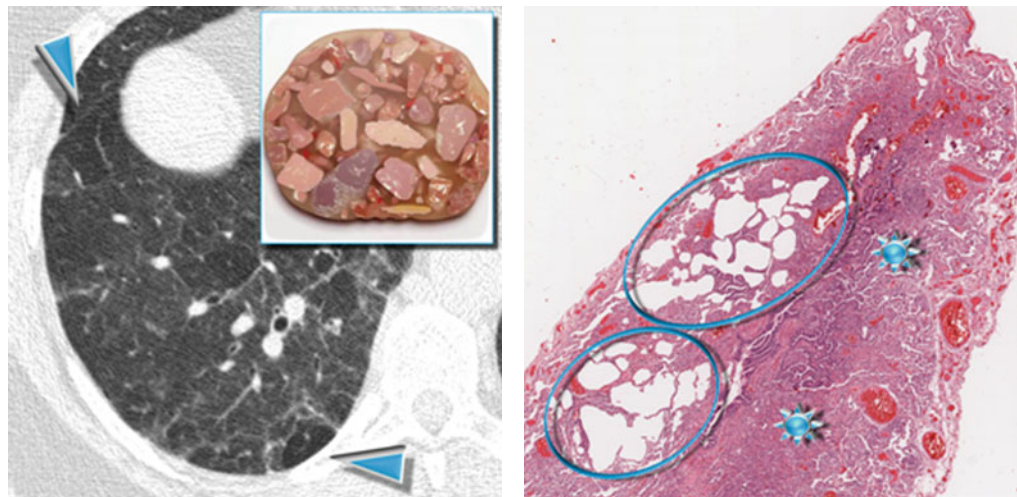
## Head-Cheese Sign

On HRCT scan, head-cheese pattern is characterized by a combination of patchy lobular areas of normal parenchyma, ground-glass opacity, and mosaic oligemia with lobular air trapping (▶). This definition is due to its resemblance to the variegated appearance of a sausage made of parts of the head of a hog. Pathologically head-cheese sign is indicative of a mixed infiltrative and obstructive process. The ground-glass opacity (GGO) component represents the infiltrative portion of the underlying disease (★). Low-attenuation lobules reflect obstructive small airways disease with resultant air trapping (⊙) and vasoconstriction from localized hypoxia.

Please also refer to head-cheese sign in "Case-Based Glossary with Tips and Tricks".



Hog's head-cheese sign, mixed (infiltrative and obstructive) disease



Common diseases with head-cheese sign:

- *Hypersensitivity pneumonia (HP), subacute*: it is the prototype disease of head-cheese sign (see figures above). The coexistence of low-density centrilobular nodules (snowflake nodules) is helpful for the diagnosis.

Rare diseases with head-cheese sign:

- *Atypical infection with bronchiolitis* (e.g., *mycoplasma pneumoniae*): frequent bilateral, lobular areas of GGO and consolidations with patchy distribution.
- *Respiratory bronchiolitis-interstitial lung disease (RB-ILD)*: although reports have cited mild emphysema in the upper lobes and small foci of GGO as distinct features of RB-ILD, it can still be difficult to distinguish RB-ILD from HP.
- *Sarcoidosis*: the presence of perilymphatic solid nodules (well-defined bronchovascular nodules and nodules along the pleural surface) helps to distinguish sarcoidosis from HP.



Integrating clinical and laboratory findings may indicate the most likely diagnosis in the setting of the head-cheese sign.

## Reversed Halo Sign



Chong BJ (2014) Headcheese sign. J Thorac Imaging 29(1):W13

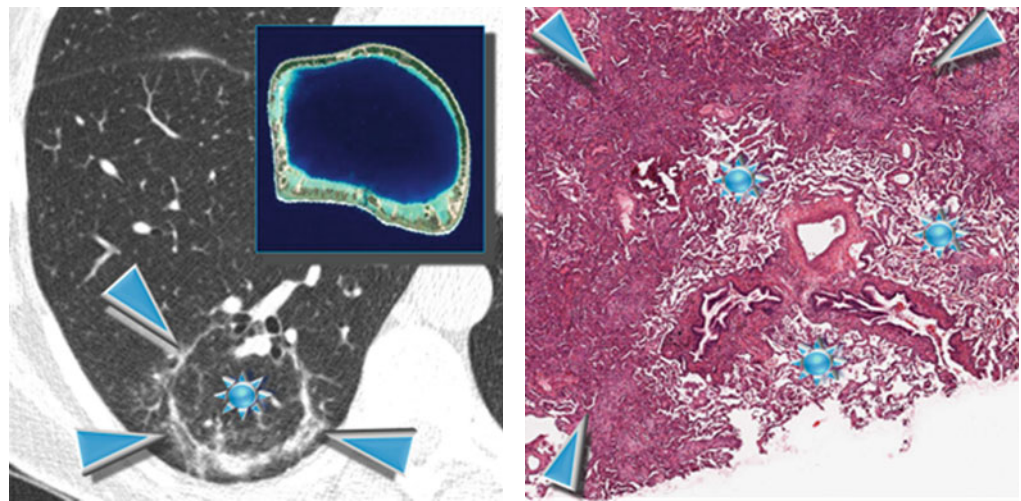
Chung MH (2001) Mixed infiltrative and obstructive disease on high-resolution CT: differential diagnosis and functional correlates in a consecutive series. J Thorac Imaging 16:69

Reversed halo sign (RHS) is a rare sign. On HRCT images, it appears as a focal rounded area of ground-glass opacity (★) surrounded by a more or less complete ring of consolidation (▶). In organizing pneumonia (OP), the central ground-glass opacity corresponds histopathologically to an area of alveolar septal inflammation and cellular debris (★), while the ring-shaped or crescentic, peripheral, air-space consolidation corresponds to the area of organizing pneumonia (▶).

Please also refer to reversed halo sign in “Case-Based Glossary with Tips and Tricks”.



Atoll sign, RHS



*Noninfectious – nonneoplastic* diseases with reversed halo sign:

- *Cryptogenic organizing pneumonia (COP) and secondary OP* (the most frequent)
- *Acute fibrinous organizing pneumonia (AFOP)*
- *Chronic eosinophilic pneumonia (CEP)*
- *Granulomatosis with polyangiitis (Wegener's granulomatosis)*
- *Hypersensitivity pneumonia (HP)*
- *Lipoid pneumonia (LP)*
- *Lymphoid interstitial pneumonia (LIP)*
- *Nonspecific interstitial pneumonia (NSIP)*
- *Post-embolic infarction*
- *Radiotherapy and percutaneous RF ablation*
- *Sarcoidosis*

*Infection* diseases with reversed halo sign:

- *Fungal pneumonia: angioinvasive aspergillosis, pneumocystosis, paracoccidioidomycosis, histoplasmosis, and mucormycosis*
- *Bacterial infections: TB and bacterial pneumonia*
- *Virus infection: H1N1*



*Neoplastic* diseases with reversed halo sign:

- *Adenocarcinoma*
- *Lymphoid granulomatosis*
- *Metastases*



Sarcoidosis and tuberculosis may present the so-called “nodular” reversed halo sign. The nodular aspect of the RHS usually corresponds to the presence of granulomatous inflammation and most likely represents active pulmonary sarcoidosis or the granulomatous infiltrate of tuberculosis. Please refer to chapter “[Nodular Pattern](#).”



Despite being no longer considered specific, the presence of RHS, in association with ancillary CT findings and an appropriate clinical history, can be useful in narrowing the differential diagnosis.

COP is the most common condition described in immunocompetent patients.

Invasive fungal pneumonia should be considered until differently proven in severely immunocompromised host.



Zompatori M (1999) Bronchiolitis obliterans with organizing pneumonia (BOOP), presenting as a ring-shaped opacity at HRCT (the atoll sign): a case report. *Radiol Med (Torino)* 97:308

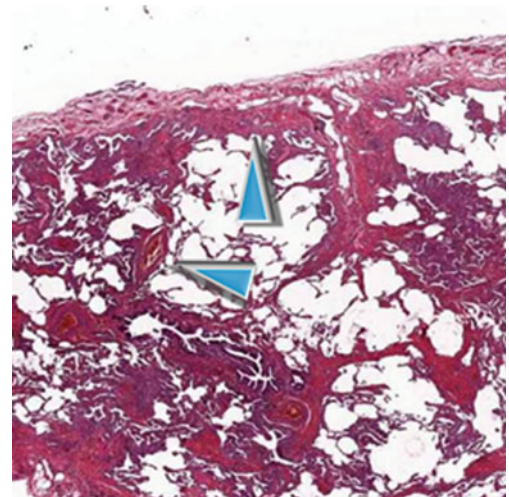
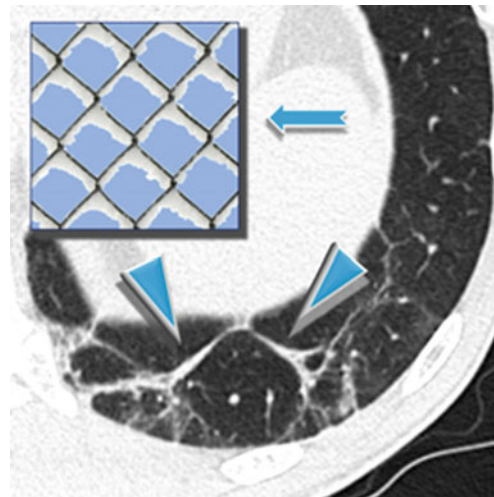
Godoy MC (2012) The reversed halo sign: update and differential diagnosis. *Br J Radiol* 85(1017):1226

## Perilobular Opacities

The term is most frequently used in the context of diseases (e.g., perilobular organizing pneumonia – OP, images below) which are distributed mainly around the inner contour of the secondary pulmonary lobule (▶). This may result in an indistinct thickening of the interlobular septa appearing as curvilinear opacities with an arcade-like or polygonal appearances similar to the snow along a wire net (➡). Perilobular pattern is thicker and, mostly, less sharply defined than those encountered in thickened interlobular septa (please compare to septal thickening visible in chapter “[Septal Pattern](#)”).



Arcade-like, perilobular pattern



The term is most frequently used in the context of diseases (e.g., perilobular organizing pneumonia – OP, see images above) which are distributed mainly around the inner surface of the secondary pulmonary lobule.



Johkoh emphasized that a perilobular distribution of disease may reflect abnormalities of peripheral alveoli but also of the interlobular septa. Differential diagnosis: smooth interlobular septal thickening, nodular interlobular septal thickening, irregular (fibrosing) interlobular septal thickening, and peripheral lobular abnormalities (OP, UIP; please also refer to “[Thinking Through Pathology >Pattern >Peripheral](#)”).

## Lobular/ Sublobular Opacities



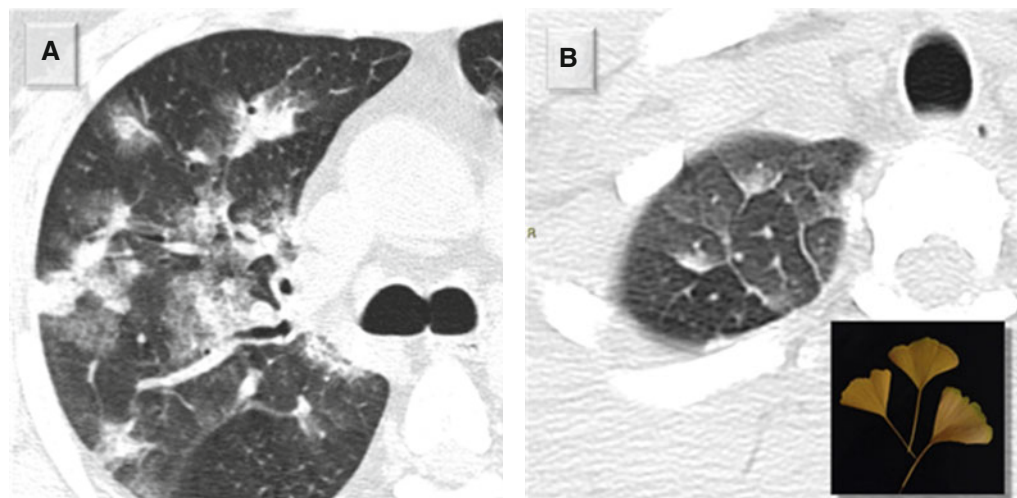
Johkoh T (1999) Perilobular pulmonary opacities: high-resolution CT findings and pathologic correlation. *J Thorac Imaging* 14(3):172

Ujita M (2004) Organizing pneumonia: perilobular pattern at thin-section CT. *Radiology* 232(3):757

Some lung diseases result in consolidation or ground-glass opacity (GGO) that involve individual lobules or groups of lobules as a whole, while adjacent lobules appear normal, giving the lung a patchwork appearance; sometimes, there may be a partial involvement of the lobule (sublobular opacities); please see Figure A. At times, lobular/sublobular consolidations and GGO may be associated with smooth septal thickening, reminiscent of a ginkgo biloba leaf. We defined this feature “ginkgo biloba sign” (Figure B). Note that the GGO sublobular areas present a gravitational-dependent distribution inside the lobules.



Lobular/sublobular consolidation/GGO, ginkgo biloba sign



Diseases with lobular/sublobular opacities:

- *Infection pneumonia* (Figure A above): e.g., pneumonia due to infection from *Staphylococci*, *Haemophilus* species, *Pseudomonas*, and *M. pneumoniae*.
- *Pulmonary edema (PE)*: bilateral pleural effusion and smooth septal thickening are often present.
- *Fat embolism syndrome (FES)* (Figure B): in patients with a fracture of a long bone, often young, the presence of lobular/sublobular consolidations, together with GGO and ginkgo biloba sign, is suggestive for FES. These signs result from the toxic and biochemical effects of free fatty acids (FFA) and of other, partly unknown, mediators with consequent edema, vasculitis, or inflammation.



Many other alveolar acute and chronic diseases may present with lobular/sublobular consolidations and GGO but not as the prevalent feature.

In some patients with lobular ground-glass opacity visible on thin-section CT scans, superimposition of a reticular pattern results in the crazy-paving appearance (please see this sign in this chapter but also in “Case-Based Glossary with Tips and Tricks”).



Webb WR (2006) Thin-section CT of the secondary pulmonary lobule: anatomy and the image – the 2004 Fleischner lecture. *Radiology* 239(2):322

Piolanti M, Dalpiaz G (2016) Fat embolism syndrome: lung computed tomography findings in 18 patients. *J Comput Assist Tomogr* 40(3):335

**Tree-in-Bud Sign, Bronchiolar**

The tree-in-bud (TIB) pattern results in a V- or Y-shaped branching pattern together with centrilobular nodularity. This pattern also resembles the small objects used in the childhood game of jacks (see the image below).

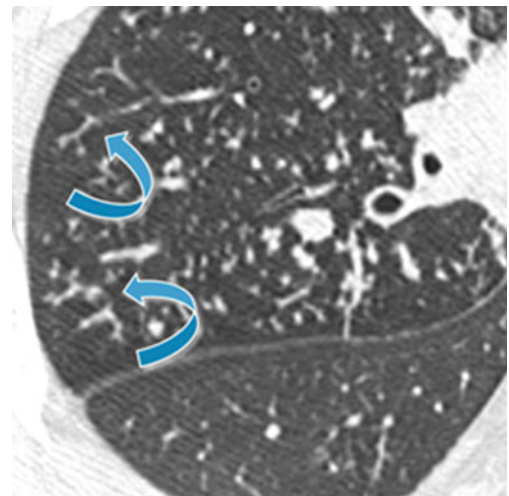
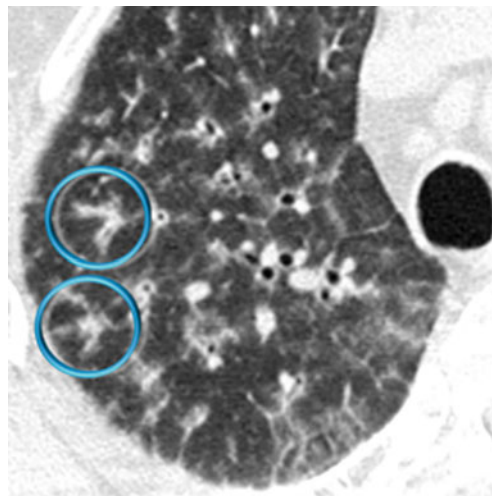
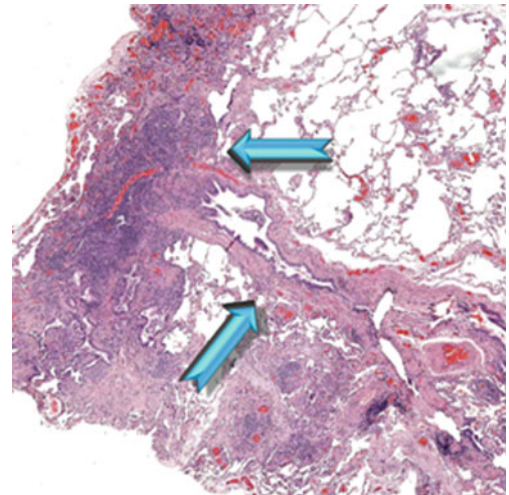
The nodules and connecting branches are peripheral but spare the subpleural lung. In *acute* disease, TIB may present ill-defined margins (○). In chronic diseases TIB presents well-defined margins (↪).

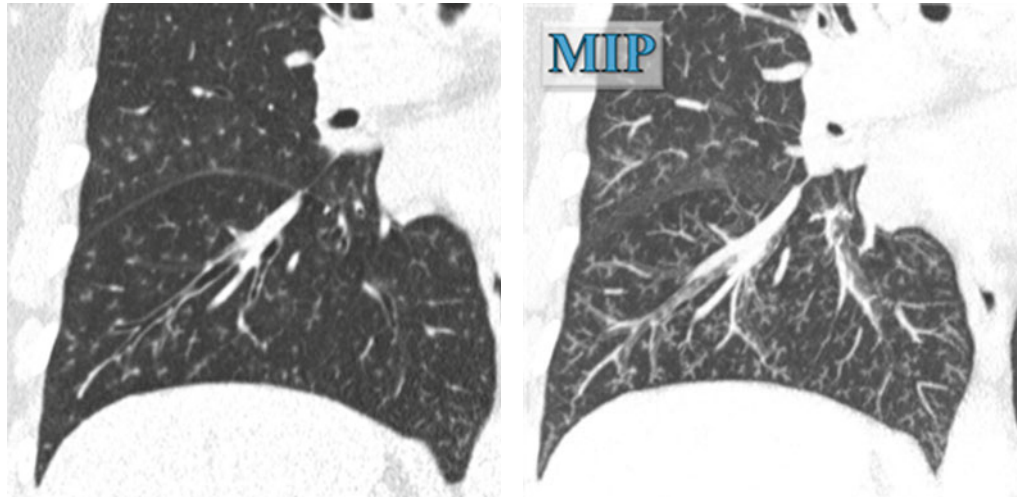
The bronchiolar tree-in-bud sign reflects a spectrum of endo- and peribronchiolar disorders, including mucoid impaction or inflammation of the wall (↪).

Please also refer to tree-in-bud sign, bronchiolar in “Case-Based Glossary with Tips and Tricks”.



TIB, centrilobular branching opacities, budding tree, jacks





Post-processing techniques such as maximum-intensity projections (MIP) facilitate the recognition of the tree-in-bud pattern (please compare the two coronal images above).



*Infection diseases with bronchiolar tree-in-bud:*

- **Bacterial:** in infections caused by *Mycobacterium tuberculosis*, identification of the tree-in-bud sign along with other imaging findings, such as bronchial wall thickening or narrowing, bronchiectases, consolidation, cavitation, and/or necrotic lymphadenopathy, supports the diagnosis. In *nontuberculous mycobacterial pneumonia*, tree-in-bud sign and bronchiectases predominate, usually most severe in the right middle lobe and lingula. The tree-in-bud sign may also be present in other types of acute pneumonia (such as those caused by *Staphylococcus aureus* and *Haemophilus influenzae*), often appearing with ill-defined contours.
- **Fungal and viral:** airway-invasive aspergillosis and *Cytomegalovirus* infection typically occurs in immunocompromised patients.

*Congenital diseases with bronchiolar tree-in-bud sign:*

- **Cystic fibrosis:** a combination of upper lung predominant bronchiectases, bronchial wall thickening, mucus plugging, and air trapping or mosaic attenuation is commonly encountered.

*Immunologic diseases with bronchiolar tree-in-bud sign:*

- **Allergic bronchopulmonary aspergillosis (ABPA):** the classic findings include central bronchiectases with an upper lobe predominance and mucoid impaction. Mucoid impaction typically appears as homogeneous, tubular, finger-in-glove opacities (please also refer to finger-in-glove sign in “Case-Based Glossary with Tips and Tricks”).

*Connective tissue diseases with bronchiolar tree-in-bud sign:*

- **Rheumatoid arthritis and Sjögren syndrome:** the most common manifestations include follicular bronchitis, bronchiectases, bronchiolitis, and obliterative bronchiolitis.

*Neoplastic diseases with bronchiolar tree-in-bud sign:*

- **Endobronchial spread of adenocarcinoma:** CT features highly suggestive of aerogenous spread include persistent centrilobular nodules and branching opacities (tree-in-bud), typically with ill-defined margins and GGO attenuation. Well-defined nodules of soft tissue attenuation are less common. Nodules tend to be clustered and invariably show evidence of growth on serial images, in some cases progressing to confluent air-space disease.

Other causes of bronchiolar tree-in-bud sign:

- *Aspiration*: a gravitational and lower lung predominance of the tree-in-bud pattern is often observed. Predisposing factors such as structural abnormalities of the pharynx, esophageal disorders (achalasia, Zenker diverticulum, hiatus hernia and reflux, esophageal carcinoma), neurologic defects, and chronic illnesses are common.
- *Inhalation (toxic fumes and gases)*: acute bronchiolar damage with diffuse resultant ill-defined tree-in-bud.
- *Diffuse pabronchiolitis (DPB)*: DPB is reported almost exclusively in Asians. It is characterized by chronic inflammation of the paranasal sinuses and respiratory bronchioles. A diffuse tree-in-bud appearance predominantly affects the lung bases. Other findings include bronchial wall thickening and bronchiectases.



Rarely, neoplastic conditions such as pulmonary arterial metastases of adenocarcinoma may result in a “vascular” tree-in-bud sign (please also refer to vascular tree-in-bud in “Case-Based Glossary with Tips and Tricks”).



Rossi SE (2005) Tree-in-bud pattern at thin-section CT of the lungs: radiologic-pathologic overview. *Radiographics* 25(3):789

Miller WT (2013) Causes and imaging patterns of tree-in-bud opacities. *Chest* 144(6):1883

**SUBSET ACUTE**

Evaluation of the clinical onset of the symptoms for each patient is the suggested way to duly frame subsets of disorders. In particular, one should know if the onset of respiratory symptoms dates back from days to a few weeks (subset acute) or instead from months to years from the time of diagnosis (subset chronic). The *acute* alveolar pattern usually presents with bilateral and often extensive consolidations and ground glass which may change in appearance, location, and size within hours or days.

Key signs	Distribution	Ancillary signs	Non-parenchymal signs	Acute alveolar disease
GGO with possible patchy consolidations	GGO often bilateral and diffuse, consolidations with multifocal and peripheral basal distribution	Signs of fibrosing lung disease (UIP pattern)	Lymph node enlargement, pulmonary arterial hypertension	<b>Acute Exacerbation of IPF (AE-IPF)</b>
GGO, smooth interlobular septal thickening	Bilateral, patchy without zonal predominance	Possible crazy paving and consolidations	Small pleural effusion	<b>Acute Eosinophilic Pneumonia (AEP)</b>
First week: GGO and/or consolidations	Bilateral extensive GGO, mainly gravity-dependent consolidations	Second week: associated sign of fibrosis	Absent	<b>Acute Interstitial Pneumonia (AIP)</b>
First week: GGO and/or consolidations	Bilateral extensive GGO, mainly gravity-dependent consolidations	Second week: associated sign of fibrosis	Small pleural effusion	<b>Acute Respiratory Distress Syndrome (ARDS)</b>
GGO and/or patchy consolidations	Bilateral extensive GGO, patchy consolidations, possible perihilar (butterfly distribution)	Scattered feeding-vessels low-density (subsolid) nodules, solid and often cavitated macronodules	Hilar or mediastinal adenopathy, tracheal concentric wall thickening	<b>Diffuse Alveolar Hemorrhage in Wegener's granulomatosis (DAH in WG)</b>
DAD pattern: GGO, possible consolidations	Bilateral diffuse GGO, possible gravity-dependent consolidations	Possible crazy paving	Possible pleural effusion	<b>Drug toxicity (amiodarone)</b>
GGO and consolidations, also lobular	GGO and lobular consolidation prevail in the upper zones, while extended consolidations are gravity dependent	Nodules, often subsolid, possible ginkgo biloba sign	Possible pleural effusion	<b>Fat Embolism Syndrome (FES)</b>
GGO	Symmetric bilateral, diffuse, or patchy, mainly the upper lobes and perihilar regions (butterfly sign)	Possible cysts, consolidations in patients with more severe disease	Small hilar or mediastinal lymph nodes	<b>Infection, acute (PJP)</b>
GGO, peribronchial cuffing and vessel enlargement, smooth septal thickening	Bilateral, central, and gravitational, possible perihilar regions (butterfly sign)	Possible crazy paving	Bilateral pleural effusion, cardiomegaly, dilatation of arteries and veins, mediastinal lymph nodes	<b>Pulmonary edema (PE), alveolar</b>



**SUBSET CHRONIC**

The *chronic* form usually presents with consolidations often localized and patchy which progress slowly, even over weeks or months. GGO often coexists. There may be architectural distortion in long-time disease.

Other signs may be variously associated.

Key signs	Distribution	Ancillary signs	Non-parenchymal signs	Chronic alveolar disease
Patchy areas of consolidation and GGO	Unilateral or bilateral, usually asymmetrical, often peripheral with lower lung predominance	Possible bubble-like lucencies, nodules, crazy paving	Adenopathies, possible pleural effusion	<b>Adenocarcinoma</b>
Patchy areas of consolidation and GGO	Bilateral peripheral (reverse bat wing sign), mainly the upper lobes	Possible crazy paving and low-density (subsolid) nodules	Adenopathies	<b>Chronic Eosinophilic Pneumonia (CEP)</b>
Patchy areas of consolidation and GGO	Bilateral peribronchial and/or subpleural, often with lower lobe predominance	Perilobular and reversed halo sign, possible crazy paving, rarely large nodules or masses	Possible mediastinal lymph node enlargement	<b>Cryptogenic Organizing Pneumonia (COP)</b>
Diffuse GGO	Bilateral symmetrical, often predominant in the peripheral regions	Possible tiny cysts within the areas of GGO, sometimes focal or lobular dark areas	Absent	<b>Desquamative Interstitial Pneumonia (DIP)</b>
OP pattern: patchy consolidations NSIP pattern: GGO and fibrosing reticulation	OP pattern: patchy NSIP pattern: diffuse, often peripheral	Nodules/masses (PET positive), crazy paving, possible hyperdense consolidations	Hyperdense liver and spleen (80 %) and heart (20 %)	<b>Drug toxicity (amiodarone)</b>
Consolidation and nodules, also cavitated, tree-in-bud sign	Patchy unilateral or bilateral, apical and posterior segments of the upper lobes, superior segments of lower lobes	Possible association with diffuse tiny random nodules	Pleural effusion (unilateral), pleural thickening, hilar/mediastinal lymphadenopathy with possible necrosis	<b>Infection, chronic: TB</b>
Patchy consolidations with possible low-density attenuation (negative values), GGO	Often bilateral, middle and lower lobes with posterior predominance	Crazy paving, low-density centrilobular nodules, signs of fibrosis	Lymph node enlargement	<b>Lipoid pneumonia (LP)</b>
Patchy consolidations	Often multiple and bilateral	Macronodules, masses	Lymph node enlargement, pleural effusion (rare)	<b>Mucosa-Associated Lymphatic Tissue (MALToma)</b>

Key signs	Distribution	Ancillary signs	Non-parenchymal signs	Chronic alveolar disease
Patchy GGO and consolidations	Prevalent lower lobe distribution	Low-density centrilobular nodules, possible clusters of tree-in-bud sign, possible beaded septum, and cheerio sign	Lymph node enlargement	<b>Metastases, arogenous</b>
Crazy paving, GGO, possible low-density (sub-solid) nodules	Bilateral diffuse, possible butterfly distribution, predominate in the lower lung	Consolidations	Lymph node enlargement, pleural effusion (rare)	<b>Pulmonary Alveolar Proteinosis (PAP)</b>