Approach to Mosaic Attenuation

Stefan Tigges
MD, MSCR, FACR, FAUR
Emory University
School of Medicine
@StefanTigges

Eugene Berkowitz
MD, PhD
Emory University
School of Medicine
Learning Objectives

1. Define the term "mosaic attenuation"
2. Describe an approach to determining whether the hyper- or hypoattenuating lung is abnormal
3. Give a brief differential diagnosis for both hyper- & hypo-attenuating lung abnormalities
The lung is like a balloon, it consists largely of air. On CT lung windows, normal lung is uniformly dark gray.

Mosaic attenuation is defined as heterogenous lung, with hyper- ("white") & hypoattenuated ("black") areas.
Unlike consolidation, in mosaic attenuation, the "white" lung is not dense enough to obscure normal structures like vessels.

Unlike emphysema, in mosaic attenuation, the "black" lung does not represent areas of lung destruction.
The first step in mosaic attenuation cases is deciding whether the white or black lung is abnormal by determining if lung vessels are uniform or non-uniform in size. Normal lungs aren’t completely black in part because of soft tissue density blood flowing through septae that outline air containing alveoli. The smaller the pulmonary vessels, the less blood flow, the darker the lungs.

Normal vessel diameter & perfusion: gray lungs.

Decreased vessel diameter & perfusion: black lungs.
Both diagrams below show mosaic attenuation. If vessels are non-uniform in size (vessels smaller in black lung), then the black lung is abnormal.

If vessels are uniform in size, then the white lung is abnormal. Let's take a closer look: we'll start by considering what can make vessels too small.
The most intuitive etiology for small vessels is pulmonary embolism, acute or chronic. Normal lung with normal-sized vessels, decreased attenuation lung contains abnormally small blood vessels. Coronal mediastinal windows show bilateral chronic circumferential pulmonary embolism.
A common but less intuitive reason for small vessels is air trapping with reflex vasoconstriction. The lung is smart: there's no point in perfusing unventilated lung.

This patient has a congenitally stenotic (atretic) anterior left upper lobe bronchus (arrowhead). Lack of ventilation leads to reflex vasoconstriction resulting in small vessels in the affected lung.
Let's apply what we've learned & figure out why this person has mosaic attenuation.

1) What is our first step?
2) Is the white or black lung abnormal?
3) What are 2 possible causes for the abnormal lung attenuation?
Let's apply what we've learned & figure out why this person has mosaic attenuation.

1) What is our first step? To decide whether the white or black lung is abnormal.

2) Is the white or black lung abnormal? Because vessels are non-uniform, the black lung is abnormal.

3) What are 2 possible causes for the abnormal lung attenuation? Pulmonary embolism & air trapping.
If the black lung is abnormal because of decreased vessel size in the black lung, how do we distinguish between pulmonary embolism & air trapping?

Pulmonary embolism may have findings of acute or chronic PE.

Air trapping may have abnormal airways.

Case: Davina Bates rID: 90694

Chronic peripheral PE.

Bronchiectasis & bronchial impaction.
Inspiratory & expiratory images can help us distinguish between PE & air trapping. In PE, both black & white lung can deflate, resulting in no change in density difference between black & white lung. In air trapping, only white lung can deflate, resulting in increased density difference between black & white lung.
Let's apply what we've learned & figure out if this person has PE or air trapping. What happened to the lung density difference between black & white lung with expiration?
The density difference between black & white lung increased in this lung transplant patient with air trapping due to chronic rejection with obliterative bronchiolitis.
Next, we'll consider the differential diagnosis in patients with mosaic attenuation & uniform vessels. In these cases, we're dealing with ground glass opacity (GGO).
Let's return to our balloon analogy. The lung consists of multiple balloons inside other balloons: lungs contain lobes, lobes contain segments etc., all the way down to the smallest balloons, the alveoli. If alveoli are completely filled with soft tissue density like blood, pus, edema, aspirated fluid or cells, the alveoli turn white on lung windows & obscure adjacent structures. This is called consolidation.

**Diagram:**

- **Normal air filled alveolus:** Black balloon → Dark gray on CT
- **Blood/pus/H2O/aspirate/tumor filled alveolus:** Multiple colored balloons → White on CT
- **Consolidation obscures normal structures:** Alveoli filled with soft tissue density → Lung window changes to white, obscuring normal structures.
If instead of being completely filled, alveoli are incompletely filled with blood, pus, etc., the alveoli turn light gray on lung windows & don't obscure adjacent structures, resulting in ground glass opacity. There are other causes of GGO, like interstitial thickening & pulmonary fibrosis, but if you can remember that GGO is consolidation's little sibling, you'll be in good shape.

Normal air filled alveolus

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<tr>
<th>Blood/pus/H2O/aspirate/tumor partially fills alveolus</th>
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Here are 2 examples of GGO, on the left due to pulmonary hemorrhage & on the right due to lepidic growth of cancer, both with incomplete alveolar filling.
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2) Is the white or black lung abnormal?

3) What are 2 possible causes for the abnormal lung attenuation?
Let's apply what we've learned & figure out why this person has mosaic attenuation.

1) What is our first step? To decide whether the white or black lung is abnormal.

2) Is the white or black lung abnormal? Because vessels are uniform, in size, the white lung is abnormal.

3) What are 2 possible causes for the abnormal lung attenuation? Pulmonary hemorrhage & infection.
Let's do one last case, what do you think of this person's mosaic attenuation? Both average (left) & MinIP (right) images are shown.
This person has mosaic attenuation, but there are 3 different densities: **black air trapping**, **gray normal lung** & **white ground glass opacity**. This combination of normal lung, air trapping & GGO is typical of chronic hypersensitivity pneumonitis. Note **traction bronchiectasis**, an additional finding in HP.
Hey Eugene, that last case was a dirty trick!

I included it to remind people that we kept things as simple as possible in this poster: our approach will work in most cases, but not every case will be straightforward. There’s a brief summary on the next slide!
Take home points: mosaic attenuation algorithm

Is mosaic attenuation present?

No. Go look at another poster!

Yes.

Are vessels non-uniform or uniform?

If vessels are non-uniform, consider air trapping & PE.

If vessels are uniform, consider causes of GGO.
Hey, Papa Bear, isn’t this supposed to be our references slide?

Yes, but we don’t have any! If you want to learn more about mosaic attenuation, your best bet is to look at the article & cases on Radiopaedia.