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Non-CF Bronchiectasis: How Radiologists Can Drive Better Outcomes

Announcer:
Welcome to CME on ReachMD. This activity, titled *Non-CF Bronchiectasis: How Radiologists Can Drive Better Outcomes,* is provided by Prova Education and supported by an independent educational grant from Insmed Incorporated.

This replay of a live broadcast discusses how to differentiate bronchiectasis patterns on HRCT to refine differential diagnosis and identify potential underlying etiologies.

Chapter 1 - Case 1: Unmanaged Bronchiectasis: The Alarming Link to Morbidity and Healthcare Costs

Dr. Suh:
We're going to be focusing on non-cystic fibrosis bronchiectasis. How radiologists, all of you, can derive a better outcome.

I'm Rob Suh. I'm a thoracic interventional radiologist from UCLA Health, and to the left of me, we have 2 esteemed speaker panelists, Dr. Lea Azour, who's my thoracic imaging colleague from UCLA Health, and we have a pulmonologist on our panel, Ashwin Basavaraj from NYU.

So here are the learning objectives for this engaging 1 hour. I'll let you read through them. And we encourage all of your participation. And for those of you who are joining us virtually, your lunch is waiting here.

All right, so without further ado, I'd like to introduce Ashwin. He's going to speak about unmanaged bronchiectasis: the alarming link to morbidity and healthcare costs.

Dr. Basavaraj:
Thank you, Dr. Suh. It's a pleasure to be here. I hope everyone's enjoying lunch. So we're going to talk a little bit about bronchiectasis and the impact on morbidity and healthcare costs here. I'm just going to highlight a case to start things off. A very common scenario that we see in the pulmonary world, just to give you guys an idea of how we approach bronchiectasis.

We have a 60-year-old female with a daily chronic cough and sputum production for 2 years, referred to our pulmonary office by her PCP for continued cough. The cough impacts her sleep intermittently, occurs during social events. There's no triggers to her cough. She's given antibiotics 2 to 3 times a year for worsening cough and fatigue, thought to be secondary to pneumonia, and very typical scenario, antibiotics help a little bit and then the cough occurs shortly after stopping antibiotics.

Her past medical history is significant for GERD, and this is one of the common comorbidities that we see in bronchiectasis: GERD, aspiration, and chronic sinusitis. History of hypertension as well in this patient, on hydrochlorothiazide and omeprazole. Remote smoking history and works as a teacher.

On exam, she had some mild bilateral wheezing, and her labs were pretty unremarkable except for a little bit of peripheral eosinophilia that was elevated.

This is a room full of radiologists and I don't have to go through this too much, but her chest X-ray was read as no acute cardiopulmonary abnormality. I'm not going to go too much into detail with this pulmonary function testing, but this is something that we obviously do very commonly in the pulmonary office. What this specific test is showing is a mild obstruction here with the FEV_1/FVC ratio less than 70%.

Because of the wheezing, because of the mild obstruction on PFTs, because of the chronic cough, she was given a diagnosis of asthma. A very common prescription for asthma is inhaled corticosteroids, along with the long-acting beta agonist. But despite the use of inhalers in this patient, it didn't really help her cough. Her cough continued, and because of that, a CAT scan was obtained.

You can see here on the CT that you have a little bit of right middle lobe bronchiectasis, a little bit of bronchiectasis in the lingula, and some bronchiolitis both in the right lower lobes and in the left lower lobes. Really picking up things—the bronchiectasis—that was not originally picked up on the chest X-ray.

Because of the bronchiolitis, there was a suspicion for a chronic infectious process. We checked bacterial cultures, fungal sputum cultures, and AFB sputum cultures, really going after NTM as a possibility for a chronic cough. Two out of three AFB sputums were positive for MAC, so that does meet ATS/IDSA criteria for a diagnosis of MAC. She was referred to our bronchiectasis clinic at NYU for further management.

When we first see patients with bronchiectasis, a very typical treatment regimen is we start with airway clearance even before antibiotics for MAC. A very standard regimen for airway clearance is 7% nebulized hypertonic saline, along with a positive expiratory pressure device to really try to clear their airways and try to improve their cough even before antibiotics.

We obtained bronchiectasis labs to look for potential etiologies. Obviously, if there's an etiology that is discovered, we'll treat that condition to prevent the bronchiectasis from worsening. We oftentimes check for autoimmune causes, immunoglobulin deficiency, alpha-1 antitrypsin, allergic bronchopulmonary aspergillosis, and genetic causes as well, such as cystic fibrosis, which there can be very mild forms of CF that we uncover in the adult population. Primary ciliary dyskinesia and alpha-1 antitrypsin deficiency are also part of our workup for bronchiectasis.

Despite airway clearance, however, in this patient, the symptoms did continue to progress, and because of that, we started antibiotics for her MAC. We started a very common regimen—the standard guideline-based regimen of azithromycin, rifampin, and ethambutol 3 times a week—and her wheezing improved, her cough improved, and there were no further pneumonias that occurred after we started antibiotics.

A very typical scenario that we see in the bronchiectasis clinic is patients will get diagnosed with asthma, they can get misdiagnosed, and they get treated as such for years until they have a diagnosis of bronchiectasis MAC and we treat the MAC appropriately and their symptoms resolve.

A little bit about bronchiectasis itself. It's increasing in incidence. A couple of reasons: aging population, diagnostic modalities are improving, and the room here knows this, but we're getting a lot more CAT scans. We're picking up bronchiectasis, particularly if patients are coming to the emergency room for chest pain, they're going to get a CT protocol. Cardiac CTs also are picking up bronchiectasis, and CT necks also can incidentally pick up bronchiectasis. So those are some of the reasons why it's increasing.

It's estimated that 500,000 adults have bronchiectasis, but that's likely an underestimate because it's not a reportable condition. So it's a lot more adults that do have bronchiectasis than reported. The incidence is higher in females, increases with age, and bronchiectasis exacerbations significantly increase the economic burden in these patients. These patients who have chronic symptoms and exacerbations have frequent hospitalizations, frequent outpatient visits, and frequent utilization of their healthcare. This condition can really be similar to what we see in terms of the healthcare costs for patients such as COPD and asthma as well.

What is a bronchiectasis exacerbation in the pulmonary world? There's a lot of focus now in trying to understand and identify bronchiectasis exacerbations. The European Respiratory Society defines a bronchiectasis exacerbation as a worsening of 3 of the following symptoms: cough, sputum volume or purulence, shortness of breath, fatigue, or hemoptysis. Basically, in layman's terms, it's a worsening of their underlying bronchiectasis symptoms, and a clinician determines that a change in treatment is required—oftentimes, the need for antibiotics.

Why is it important to identify bronchiectasis exacerbations? It's because bronchiectasis exacerbations can have an impact on clinical outcomes. There can be a decline in lung function, worsening symptoms, worse quality of life, and increased mortality in patients that

have bronchiectasis exacerbations. Having bronchiectasis exacerbations is the leading cause for predicting future exacerbations in this patient population.

It's important to try to identify bronchiectasis exacerbations appropriately and not to have them misdiagnosed as COPD or asthma exacerbations. Oftentimes, if patients are diagnosed with COPD or asthma, they are treated with steroids. For example, if they come into the emergency room or they're admitted as an inpatient, we give them IV steroids to help with their wheezing and their cough. But we're starting to understand that the use of steroids, particularly in patients with bronchiectasis and chronic underlying infections such as NTM, can worsen the underlying infectious process. There's increasing literature supporting that both with the use of inhaled corticosteroids and with the use of systemic steroids, there could be a higher incidence of chronic infections and worsening outcomes in these patients.

The pathophysiology of bronchiectasis is called a vicious cycle or vicious vortex model, where we have bronchiectasis from an underlying etiology that leads to pooling of mucus in the airways, impaired mucociliary clearance, and these chronic infections such as NTM, MRSA, and pseudomonas take advantage of that. They colonize the airways and that leads to further inflammation, and then further bronchiectasis in this cycle pattern. This has now morphed into the idea of the vicious vortex model, where even if we try to treat one of those steps, it may not be enough to prevent further progression of the bronchiectasis, and we have to focus on each of these steps to help our patients with bronchiectasis.

We're starting to understand the importance of the inflammatory process in bronchiectasis, particularly neutrophilic inflammation, which is the predominant inflammatory mode in our patients with bronchiectasis. It's known that neutrophils are abnormal in bronchiectasis. They have impaired phagocytosis, and they can release what's known as neutrophil extracellular traps, or NET formations. What that is, is that it attempts to trap the bacteria in the airways. It does a good job of trapping bacteria, but then that meshwork of DNA and fibers that composes these NETs gets stuck in the airways. When these NETs are trapped in the airways, that in and of itself can lead to further inflammation, lung destruction, and bronchiectasis. A lot of focus and current therapies are really targeting reducing neutrophilic inflammation and reducing the formation of NETs in the airways.

One of the more promising therapies that just recently completed phase 3, and the results were just published in *The New England Journal of Medicine* last week, is what's known as a DPP-1, a dipeptidyl-1, inhibitor called brensocatib, which attempts to hinder the release of neutrophil serine proteases, which are elevated in patients with bronchiectasis. The goal is to try to reduce bronchiectasis exacerbation by administering DPP-1 inhibitors. What this trial showed—it was a randomized, double-blind, placebo-controlled trial involving over 1,700 patients receiving either 10 mg, 25 mg of brensocatib, or placebo.

What the results showed was a decreased rate of annualized pulmonary exacerbations, an increased time to the first exacerbation, about 20% reduction in exacerbations in these patients, and an increased proportion of patients that remained exacerbation-free at both doses. The higher dose—the 25 mg dose of brensocatib—had a disease-modifying effect, where in patients with bronchiectasis exacerbation, that made accelerated lung function decline, but in patients that received the higher dose of brensocatib actually slowed the progression of lung function decline.

This particular medication is currently with the FDA, and the hope is that it gets approved later on this year for our patients with bronchiectasis and exacerbations.

It's not just neutrophilic inflammation, which is the predominant phenotype in our patients with bronchiectasis, but it's also known that patients can present with an eosinophilic phenotype. About 20% of patients with bronchiectasis can have this eosinophilic phenotype, where patients present with an asthma–bronchiectasis overlap and a TH2 phenotype. There's a lot of interest in the potential for therapies that are utilized in asthma—for example, biologic therapies targeting eosinophilic inflammation in bronchiectasis. So more to come in the future in this phenotype.

This is a very exciting time in bronchiectasis. A number of clinical trials have just recently completed or are ongoing. They are studying very standard therapies such as 7% hypertonic saline, which we use a lot clinically, but not a lot of data supporting its use. Also studying some anti-inflammatories that we discussed as well. So more to come in terms of potential therapies in bronchiectasis.

Just highlighting the importance of recognizing bronchiectasis early and recognizing exacerbations and treating them appropriately. So thank you, everyone, for your attention.

Thank you.

Chapter 2 - Case 2: Early Detection, Better Outcomes: Why Early Recognition of Bronchiectasis Matters

Dr. Suh:
Our next speaker is Lea Azour, and she'll be speaking on why early recognition of bronchiectasis matters. We'll take your questions at the end during the panel discussion.

Dr. Azour:
Thank you, Rob, and good morning, everybody. So our objectives today are very radiology focused, and they are threefold. First, to identify the imaging signs of bronchiectasis on CT; second, to review the causes of non–cystic fibrosis bronchiectasis and progression; and then very briefly, to explore the role of AI tools for the diagnosis and surveillance of bronchiectasis.

Let's start with the definition of bronchiectasis. This is quite important for us as we are writing our reports because the diagnosis implies longevity or chronicity or persistence of this imaging finding. It should be irreversible bronchial dilatation to be called bronchiectasis, and that may be questionable if we only have one time point.

That leads us to: what is clinically significant bronchiectasis? When does it matter? Luckily, there was an international expert consensus statement that came out in 2021 in *Lancet Respiratory Medicine*, and it defined this for us. Clinically significant bronchiectasis is a disease that requires 2 components: clinical and radiologic criteria. So I'll briefly review both of these aspects or arms of diagnosis for us all. On the left-hand side, we see the radiology algorithm, and on the right-hand side, the clinical algorithm. When we start out on the left, when we have incidental evidence of bronchiectasis on a chest CT, to satisfy this diagnosis, a patient should have at least 1 of 4 findings. First, an inner airway-to-artery diameter ratio equal to or greater than 1—and we'll show images and examples—an outer airway-to-artery diameter ratio of greater than or equal to 1, a lack of tapering of the airways, or visibility of airways in the lung periphery. Satisfying at least 1 of those satisfies the radiologic diagnostic criteria.

What about the clinical criteria? This is a little more stringent. The patient must have at least 2 of the 3: cough most days of the week, sputum production most days of the week, or a history of exacerbations. So if there is at least 1 of the 4 radiologic signs and at least 2 of the 3 clinical signs, we can then say the patient has clinically significant bronchiectasis.

Let's now turn to the imaging signs of bronchiectasis, beginning with the CT findings. We can see some of the findings on X-ray, but often we confirm these with CT. So we'll look at airway dilatation, lack of tapering, and the presence of bronchi within that lung periphery.

And also as radiologists, we often like to have corroborative or corollary findings, and these, in the setting of non–CF bronchiectasis, would include signs of chronic or indolent infection, which would be bronchial wall thickening or irregularity, mucus stasis, bronchiolitis, and potential mosaic attenuation or air trapping.

Let's start with looking at the relevance. So why these 4 imaging signs? How did we arrive at these 4, and why are they the most important? This expert multinational consensus panel rated various imaging signs with various levels of stringency for the airway-artery ratio. What they did was correlate which of these imaging signs increased the likelihood of actual true bronchiectasis. The top 4, which were in our algorithm, were the ones that correlated with the highest likelihood to have clinically true significant bronchiectasis. If you look at all of those numbers, the mean score out of 4—2.89 and above—those scores of those top 4 features were greater than the pooled average of all the features.

These included the more stringent cutoff of inner airway-to-artery diameter ratio of greater than or equal to 1.5—same for the outer—the lack of airway tapering, and visibility of airways in the periphery. And of course, these measurements can be under- or overestimated based on whether there's adherent mucus making that inner airway diameter smaller or if there's bronchial wall thickening making that outer airway diameter larger.

On to our pictures. The imaging features. The first is that bronchial dilatation. It's also known as the signet ring sign, as per the Fleischner Society, and the blue arrows are pointing to that dilated bronchus or airway with the adjacent smaller pulmonary artery. This is what we're calling the airway-artery ratio—the AAR. I don't think many of us are putting calipers on this. Qualitatively, we can appreciate when it's about 1 or greater than 1.5, and that's a good gestalt for us to develop.

This is another image to highlight that AAR ratio. And in the algorithm, greater than or equal to 1 did satisfy the radiologic criteria, but we do know now as radiologists that the larger that airway is, of course, the more likely it is that that bronchiectasis will be true bronchiectasis.

The next imaging sign is the absence of airway tapering. This is an early and sensitive sign of bronchiectasis. In this example, there's curved planar reformatted images, we see cylindrical bronchiectasis, and we also note that there is an absence of tapering over 2 cm distal to that airway bifurcation—that the airway diameter is maintained even distally.

The tram-track sign is synonymous with this cylindrical, equidistant, non-tapering airway. We can often note this on radiograph. In this

patient, we actually also see a right middle lobe opacity, so we have an inkling that this patient probably has chronic right middle lobe atelectasis, probably has non-tuberculous mycobacterial infection, and we're probably not hallucinating that bronchiectasis in the right lower lobe.

This is the corresponding CT that goes with that radiograph, where we again see the tram-track sign: perfectly parallel, cylindrically widened, bronchiectatic airways. It is an X-ray or a CT sign.

The final imaging sign that we look for—the 1 of 4 to satisfy those radiologic criteria—would be airways within 1 cm of the lung periphery. In the left-hand image, that is the anterior lung, the right middle lobe, and we see those dilated airways, which are larger than their adjacent pulmonary arteries, within 1 cm of the lung periphery. And that's how we define "periphery," that 1 cm cutoff.

On the right-hand image, we see thickened and impacted peripheral airways. These are not actually vessels. When we trace them back up, they correspond to airways. They can be impacted airways or not impacted at that lung periphery, so trace them back up.

Pitfalls. What we're talking about today is non–CF-related, so non–fibrosis-related airways disease. If we are leaning more toward traction bronchiolectasis or fibrosis being the etiology, we would often have corollary findings of fibrosis. What would these findings be? They would be signs of interstitial lung abnormality—early fibrosis such as subpleural, septal, or reticular opacities.

The next one—having chronic microaspiration and small hiatal hernias—that can lead to interstitial lung abnormalities, but as we know, that can also exacerbate airways disease, so that can go either way. What may also be a helpful corollary finding is looking for the absence of expiratory phase air trapping or mosaicism. For example, we know that IPF- and UIP-related traction bronchiolectasis, mosaic attenuation would not be a consistent feature there.

What are, though, the corollary imaging findings that we look for in non–CF-related airways disease in bronchiectasis? Oftentimes, because the etiology is chronic infection, we are looking for those signs in our parenchyma and in our airways; that can include airway wall thickening or irregularity. Typically, the airway wall shouldn't be more than about 1 mm thick. We could see bronchiolar impaction and bronchiolitis, as we see in this image in this patient with non-tuberculous mycobacterial infection.

Mosaic attenuation is another feature we may see in airways disease. Because bronchiectasis may be an incidental finding, we may not always have expiratory phase imaging. A tip that may help you identify these geographic regions of lower attenuation would be to change your setting to a minIP. That decreases the conspicuity of the vessels and may help you appreciate that geographic lower attenuation, as in this image, which is a minIP.

Let's now turn to the second major topic, which is the etiology of this bronchiectasis. Chronic inflammation, or post-infectious, chronic infectious states, may lead to bronchiectasis. Again, when we're talking about bronchiectasis, we're referring to something that is irreversible. In a meta-analysis of over 50,000 patients, the leading contenders were Pseudomonas, non-tuberculous mycobacterium, Candida, and rhinovirus. This is helpful as we look in our electronic health records, because it can raise our suspicion for when a patient may develop or may be more likely to develop bronchiectasis or progressing bronchiectasis. Of course, there are other etiologies like obstructive lung disease, immune deficiency, and congenital mucociliary dysfunction.

Let's look at progression in the setting of chronic infection or indolent infection. This was the same patient over the course of 6 years. In 2019, we see that they have bronchiolitis. It was multifocal. I'm just showing you one location in the right lower lobe. Even though the infection—the bronchiolitis—is better controlled in 2025, nonetheless, we see the sequelae of a chronic infectious process, chronic non-tuberculous mycobacterium. As we move down further into the lung bases, we see that the patient, over the course of 6 years in the setting of an indolent chronic infection, has developed bronchiectasis. So we see the airways are dilated relative to the adjacent arteries, and they are within 1 cm of that lung periphery, particularly if we look at the paramediastinal right lower lobe.

Another pitfall: presuming progression. We actually saw this quite a bit during COVID. When patients have acute airspace opacities or consolidations, airways may become dilated. However, oftentimes, after the resolution of said infection, they may return to their normal size, and so that is not bronchiectasis. During COVID, many of us had conversations about not reporting out as bronchiectasis based on the one time point. Get the follow-up to resolution of the infection and see what happens to those airways before we diagnose a patient with bronchiectasis.

Now for our final couple of slides. What are the implications? How important is it that you're pointing this out? Does it correlate with what happens to our patients? Yes, it does.

In the COPDGene cohort, they looked at the predictive value of the AAR—that airway-artery ratio—and the percent of airways with an AAR greater than 1, not even using the 1.5, was associated with more exacerbations.

So a sign like this, which I think many times we're not even commenting on the AAR in our reports, it does have a clinical implication for

our patients. Am I encouraging us to put calipers on and measure this? No, I'm not really encouraging that. I'm hoping that we're going to be moving toward automated quantification and the incorporation of tools that help us provide this clinically relevant information to our patients.

This is a diagrammatic representation of that AAR. We see the artery diameter and then the 2 ways of measuring that airway diameter, that inner or outer. There are currently algorithms available to measure these various diameters. Why would that be important? Why is that helpful? Eventually, that can not only support detection but also the monitoring of airways disease. If we have a quantitative parameter that we're following, we can actually quantify in terms of extent and also severity of that airway dilatation. That, then, opens up the possibility to risk-stratify patients and personalize therapy.

So I thank you for your attention.

Chapter 3 - Case 3: Are You Missing These Bronchiectasis Patterns? Essential HRCT Tips for Radiologists

Dr. Suh:
All right, great. Okay, I'm going to do the next talk, and I'll cover essential HRCT tips for radiologists. So in addition to what Dr. Azour has nicely covered, bronchiectasis can be classified by morphology. And although morphology does not necessarily correlate with pathology, it can be a useful distinction in helping you determine the severity or chronicity of the airway disease.

Compared to the normal artery, cylindrical bronchiectasis is a uniform or tubular dilation of that airway. As bronchiectasis progresses, it may become varicose or varicoid, whereby the walls start to expand and you have outpouchings. In general, in this type of bronchiectasis, those outpouchings do not exceed the diameter of the airway. In its most severe form with saccular and cystic bronchiectasis, those outpouchings get worse, and by then certainly do exceed the true diameter of the airway, even when in highly dilated states.

So once bronchiectasis has been identified, a pattern-based approach may be helpful to identify the potential underlying etiology or to help you render a radiographic or radiologic diagnosis. There are several pattern-based approaches out there. This one is published by Singh in 2019, and the reason why I highlight it here is it's fairly straightforward and easy to use. In this algorithm, bronchiectasis can be focal or diffuse. The diffuse can be further subdivided into what we call lobar predilection—meaning what lobe does it predominate in, even though it's diffuse—or a bronchial division pattern. We'll get more into this, but is it central-dominant or peripheral-dominant?

So when you have focal bronchiectasis, most of the time it's going to be due to a structural cause, and this can be divided into intramural, mural, and extramural etiologies. For those intramural etiologies, it means that something is actually in the airway causing a form of obstruction and usually dilation or post-obstructive phenomenon. Commonly, we're looking at foreign bodies, especially in the pediatric population, and you can also sometimes see broncholithiasis. In this situation, you want to check the attenuation of that filling defect within the airway, and if it's calcified, you kind of have your diagnosis. Certainly, tumors of the airway can also arise, whether they're benign or malignant, and on those images on your left you can see a typical carcinoid tumor filling up that airway.

For mural causes, it's often due to either severe or repeated inflammation or infection of the airway, damaging the walls, causing airway stenosis. But sometimes even congenital causes can cause an airway to be abnormal and dilated. On your right, there's a case of bronchial atresia where you see the dilated airway within the central part of the left upper lobe. You see a partial mucocele and the surrounding hyperinflation or air trapping. Further analysis would reveal an atretic communication or a non-existing communication with the main tracheobronchial tree. Finally, if you have extraluminal causes, these are going to be something outside of the airway pushing on the airway, and oftentimes this might be lymphadenopathy or even primary and secondary malignancy, especially in the older population.

So if we look at diffuse bronchiectasis, it's important to try to figure out: is there a preferential lobe involved despite the diffuseness of the bronchiectasis? For upper lobe preference, we think of diagnoses such as cystic fibrosis and allergic bronchopulmonary aspergillosis, but there are situations where there's been damage to both the airway and parenchyma over time resulting in abnormalities of both. So you can see in the middle images, if you've got a fairly diffuse pattern in the upper and mid lungs that's reasonably symmetric, you might think of sarcoidosis. If you had some fibrocalcific residual, on your right, you might think the person has had prior tuberculosis. Whether it's local or multilocal, if you have the appropriate clinical scenario, you certainly should think of post-radiation fibrosis, if appropriate.

When we think of middle lobe preference or predilection—I think it's already been shown, and I think people have been programmed to think of non-tuberculous mycobacteria. But diseases that are diffuse, such as asthma or primary ciliary dyskinesia, can also involve, preferentially, the middle lobe with involvement of the airways diffusely. Here you can see very nice examples of asthma and primary ciliary dyskinesia on your bottom left and on the rightmost images.

Now finally, if you have lower lobe predilection, we're looking at conditions such as aspiration, chronic recurrent aspiration, and congenital causes such as, again, primary ciliary dyskinesia, alpha-1 antitrypsin deficiency, and immunodeficiency in general—specifically hypogammaglobulinemia and common variable immune deficiency. As manifested or as a response, typically, we'll see granulomatous-lymphocytic interstitial lung disease.

Certainly, we see bronchiectasis often as a part of pulmonary fibrosis or interstitial lung disease in the lower lobe. But in this situation, it's really important to distinguish traction bronchiectasis that results from abnormal parenchyma immediately outside of this airway pulling that airway apart, as opposed to intrinsic or airway-intrinsic bronchiectasis, which is the abnormality where the problem resides in the airway itself or in the wall of the airway.

A clue to traction bronchiectasis is if you look at the tissue immediately outside of that abnormal airway. In this case on your left, you see that there's a high degree of textured ground-glass attenuation and reticulation consistent with pulmonary fibrosis, and that's kind of the aha moment. Right? You should think that, well, there's fibrosis there and that's what's making that airway abnormal. This is to compare and contrast with your image on your right, where you see peribronchial thickening and dilation of the airway. But notice that the tissue immediately outside of those airways is fairly normal in appearance and attenuation.

Another clue to traction bronchiectasis, in addition to the abnormality outside of the airways, is that for how abnormal that airway looks, it's not proportionally thickened. And again, that should make you think that maybe the airway is abnormal, but it's not really an airway-driven process.

So in addition to lobar predilection, another way to evaluate or another assessment of diffuse bronchiectasis is to look at what generations or order of bronchi are affected. When we talk about central bronchiectasis, we're really focusing on those airways within the middle two-thirds of the lobe or the lung. In this situation, we think of conditions such as, again, ABPA or, if we're thinking congenital, Mounier-Kuhn syndrome. If we look at peripheral causes of bronchiectasis, meaning affecting the airways within the outer one-third of the lobe or the lung, one diagnosis that should come to mind is Williams-Campbell syndrome.

Here, you can see some nice images of both. Often, you can use a minimum intensity projection to really highlight when you have diffuse airway disease, where the airways are predominantly abnormal. It's pretty easy to see on those coronal images where the majority of airway dilation is in both of these cases.

So again, a pattern-based approach may be useful to help you identify the etiology for the bronchiectasis, as well as to render a radiographic or radiologic diagnosis. Certainly, with bronchiectasis, there are additional or associated findings that we see—often a complication of chronic airway disease. As you all can imagine, with bronchiectasis or abnormal airways, there's going to be some degree of mucus stasis or impaction. Here we have a case of ABPA, and you can see on the corresponding soft tissue windows, again, those characteristic high-attenuation mucus plugs.

Certainly, with abnormal airways, these patients are prone to superimposed infection, which may manifest on CT as airspace consolidation or clustered centrilobular nodularity—or, if you will, tree-in-bud. Another clue is when you have bronchiectasis and, again, in a severe form, if you see air-fluid levels within those airways that are abnormal, that could be signs of an intercurrent or concurrent airway infection.

Patients with severe airway disease for long periods of time can certainly develop airway complications with pneumothorax or even bronchopleural fistula, as in this case of severe cystic fibrosis. Sometimes over a period of time, if those airways are really abnormal, the parenchyma in between gets heavily scarred and damaged and eventually obliterated. And if you look at this case of cystic fibrosis, with this complete cicatricial collapse of the left upper lobe, you can see that those abnormal dilated airways are all bundled together with, essentially, no healthy parenchyma in between.

Certainly, you may have secondary pulmonary arterial hypertension with any chronic airway process.

So in evaluating bronchiectasis, the European Respiratory Society, or ERS, suggests a minimal bundle. This includes a differential blood count, serum immunoglobulins, test for ABPA, and a sputum culture for bacteria, AFB, and fungus. In addition, alpha-1 antitrypsin phenotype testing can be performed. And certainly, we're going to do inspiratory and expiratory high-resolution chest CT and pulmonary function tests. For patients with suspected cystic fibrosis, a CFTR gene panel can be sent in addition to the traditional sweat chloride test. And in those patients that we suspect primary ciliary dyskinesia, nasal nitric oxide is an inexpensive and fairly accurate test. And certainly, electron or high-speed video microscopy can be ordered to evaluate these patients as well.

So in conclusion, bronchiectasis results from various etiologies. Early diagnosis is critical to prevent chronic airway and parenchymal complications. A pattern-based approach facilitates radiographic diagnosis. Hopefully, this is helpful.

Chapter 4 - How Effective Communication Makes Radiologists Key Players in Bronchiectasis Care

Dr. Suh:
Okay. So the remainder of the session, we'll devote to how effective communication makes radiologists key players in bronchiectasis care and for the care of bronchiectasis patients. So feel free to send your questions on any of the talks, and we'll kind of start out with some, I guess, some questions for you.

So, Ashwin, I've got this question. So, in some studies, I think up to 40% have said that if the etiology of bronchiectasis has been identified, this has impacted significantly the care of these patients. So my question to you is—you know, it's a room full of radiologists, right? How much should we be offering in our reports? Is it sufficient enough to say, "Hey, bronchiectasis, not otherwise specified"? Or should we go a little bit more and try to give you a diagnosis? Would you find that helpful at all?

Dr. Basavaraj:
Yeah, it's a great question. I think the way I'm thinking about it is from an academic and community perspective, right? So I'm coming from a specialized bronchiectasis clinic where we have our standard panel of testing, but we see patients referred to us who haven't had that standard testing. These patients can go undiagnosed, for example, with cystic fibrosis for years. And there are effective therapies specifically for that patient population with CF, with the use of modulator therapy.

We also know that these chronic infections, such as NTM, show an association with bronchiectasis, and I think it would be helpful to include that in the report so patients can get an AFB sputum culture earlier and get treated appropriately. So I do think more information for a pulmonologist or for a clinician would be important so they can think about sending some of the diagnostic testing that would be useful for early diagnosis.

Dr. Suh:
Well, thank you. Do you ever get patients where bronchiectasis has been reported on their chest CT report, and they self-refer to you or your team?

Dr. Basavaraj:
Yes. We see the whole gamut. We have patients who have had bronchiectasis for a number of years and they are unhappy with their care or they're interested in clinical trials that we offer at our institution. And we have patients who are referred from community pulmonologists that just may not have the time or the support that's needed for a lot of these patients, for example, for setting up IV therapy for our patients with advanced mycobacterial disease, such as Mycobacterium abscessus. And we have patients whose families are encouraging them to seek better care. So it's really the whole gamut that we do see in our practice.

Chapter 5 - Audience Q&A

Dr. Suh:
Are there any audience questions? I mean, burning questions? Just questions you need to get off your chest for any of the panelists or myself?

All right. Here we go. Yes?

Male Voice:
Are there situations where the AAR can be greater than 1, such as in an older individual—so over 70—where the airways otherwise look normal but they're larger than the arteries? Or is that always pathological?

Dr. Basavaraj:
The question was AAR being—

Dr. Azour:
And the association—if the AAR will increase with age, and if there's a component of it which would be senescent. If I get the question correctly.

So I don't, to my knowledge, think that there – I haven't seen a study that stratifies it by age, that describes it as being – or normalizes it to age, so I think I would still use the AAR irrespective of age. And if there's any equivocation, what helps is the more stringent 1.5.

Dr. Basavaraj:
Yeah, I was going to say that if you want to be a little bit more focused, especially as the patient gets older, you can use that AAR of 1.5

or greater. It might be more useful than if you're calling everybody with just a little over 1 as bronchiectasis—especially if there's no fibrosis or other signs that might result in traction changes of that airway.

Male Voice 2:
Thank you for the talk. I just had a question. Do you guys have recommendations for reporting? Do you guys use like a mild, moderate, severe when reporting a bronchiectasis, or do you just describe the type and say what it is?

Dr. Suh:
Yeah, that was a question that came up, believe it or not, at dinner. And so there really—I mean, there are tools out there and research tools, but I think most of us don't really use that very much in clinical practice. So at least for me—and I think Lea, and I don't want to speak directly for her—but the idea is that we kind of, okay, so you describe maybe how much involvement you have, and I like to report what the morphology is.

So I think if you're saying that it's cystic bronchiectasis, it usually implies that it's fairly severe, right? And if you can identify, again, a lobe—or if it's just one lobe, it might not be as bad as if it's diffuse, right? So I think just sticking to kind of the main branch points of that algorithm would be helpful.

Male Voice:
Thank you.

Dr. Suh:
So we have a question here from the audience. What degree of radiologic progression would support initiating antimycobacterial therapy in normally functioning clinical PFTs in a middle-aged female without history of frequent exacerbations?

Dr. Basavaraj:
Yeah, it's a great question. There's a lot of factors that we take into consideration when we're thinking about antibiotics for NTM. We're not very aggressive about starting antibiotics up front because antibiotics are prolonged. There's multiple antibiotics which could have side effects, and there's a high recurrence and relapse rate. About 50% of patients who complete at least a year of multidrug antibiotics can have recurrence of their NTM. So we really need good reasons to start NTM antibiotic treatment, and that could be progressive weight loss, that could be progressive CT findings despite optimal airway clearance, unintentional weight loss, or hemoptysis or cavitary disease, also, could be another reason. So if patients do progress radiographically but also clinically, such as weight loss or hemoptysis, those could be reasons where we would have a low threshold to start antibiotics for those patients.

Dr. Suh:
Okay, let me ask you this question—not to put you on the spot—but how do you think radiologists could expand their role in sort of helping out this process, right, with better communication? What are the things that we can do right away?

Dr. Basavaraj:
Yeah, I think one thing would be having a differential, right? Just checking the AFB culture early would make a huge difference in these patients. If we can uncover that a patient has underlying NTM or MAC, it just opens up the scope of possibilities and treatment options for these patients. And patients will not get an AFB sputum for years before it's checked, so if you can, in the reports, say consider NTM as a possibility for their underlying bronchiectasis and bronchiolitis, I think that would make a huge difference in terms of an early diagnosis and appropriate treatment for these patients.

Dr. Suh:
Well taken. And Lea?

Dr. Azour:
Yeah, I think there are a few things we could do as radiology departments and as radiologists individually. The first would be to communicate with our pulmonologists and understand what they're looking for and how to make their follow-up easier.

Informatic solutions we could use would be having comments like recommending the clinical workup actually trigger lists in the EHR that would then go to the pulmonologists, though it decreases the burden on them as well. And what I think would also help would be closing the loop to know how effective we are. I mean, how much of our reporting is having a clinical impact? Are we catching it earlier? Does it matter? And that's where the relationship comes in with the pulmonologists. And understanding should we be changing?

And I also do think we need to be able to provide more quantitative measures that are not at our own burden of us measuring more things but incorporating more data into our reports. That doesn't necessarily mean more paragraphs for us to write.

Dr. Suh:

All right. Very good, very good. So, I guess we want improved communication, right? Maybe some sort of, obviously, identification of bronchiectasis on the CT reports and, where appropriate, a differential diagnosis or a radiologic diagnosis. Right? Again, closing the loop and speaking with our referring physicians or the multidisciplinary care team, right, would be all useful to sort of drive this process.

So, again, if you have any further questions, you can grab any one of us after the session ends. Hope you learned a little bit of something about bronchiectasis and the importance and the impact, that diagnosing this condition impacts patient morbidity and, certainly, healthcare costs, identification of bronchiectasis and patterns on CT, and where appropriate communication or good communication is good for good patient care.

Thank you for your attention. Have a good afternoon.

Announcer:
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