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Seeing the Invisible: Radiologic Precision to Enable Early Intervention in Bronchiectasis

Announcer:

Welcome to CE on ReachMD. This activity, titled Seeing the Invisible: Radiologic Precision to Enable Early Intervention in Bronchietasis, is provided by Prova Education and supported by an independent educational grant from Insmed.

This replay of a live broadcast discusses improving your ability to differentiate the radiologic patterns of bronchietasis from other lung diseases.

Chapter 1: Introduction

Dr. Suh:
A good, happy lunch hour. Thank you for spending your precious lunch hour with us. I know that this is the noon presentation that you'll be all wanting to attend and looking forward to. There is a QR code to complete a pre-test, as well as polling tests and post-test questions along the way.

So today's presentation is entitled Seeing the Invisible: Radiologic Precision to Enable Early Intervention in Bronchiectasis. I'm Rob Suh out of UCLA Health, and I'm joined by my 2 esteemed colleagues, Mary Salvatore, who is a professor in radiology at the Albert Einstein College of Medicine, as well as the radiology chair at Jacobi Medical Center in the Bronx. Then we have Ashwin Basavaraj, who's an Associate Professor at NYU. He's the section chief of Pulmonary Critical Care and Sleep Medicine at Bellevue in New York, as well as the director of Bronchiectasis and Non-Tuberculous Mycobacteria Program. Did I get that right? Yeah, okay. All right, just see, all right, I'm feeling good. All right, good.

Okay, so these are our conflicts of interest.

And our learning objectives today are to accurately identify subtle imaging signs of bronchiectasis on high-resolution chest CT to facilitate early diagnosis and intervention and to differentiate bronchiectasis patterns on HRCT to refine that differential diagnosis and identify potential underlying causes, and, finally, to describe the impact of bronchiectasis on patient morbidity and healthcare utilization to underscore the need for timely intervention.

All right, without further ado, my colleague Mary will come up and talk about detecting early and subtle bronchiectasis on HRCT.

Chapter 2: Detecting Early & Subtle Bronchiectasis on HRCT

Dr. Salvatore:
Hi. It's very nice to be here with you today. I don't know if you are like me. When I see an abnormality on the CAT scan, the first thing I

want to do is see what it looked like before, right? The earliest appearance of diseases on the CAT scan are most helpful in making the diagnosis. By the time the disease is end stage, everything looks very similar.

Our learning objectives for this talk are to identify the CT scan findings for bronchiectasis, to talk about etiologies for non-cystic fibrosis bronchiectasis, and see how AI might be helpful.

When I think about bronchiectasis, I think about my favorite article on the topic of bronchiectasis. It was written in 1950 by Dr. Lynne Reid, and she was doing dissections on cadavers and looking at the bronchi, and she saw that in the normal patient, from the time the trachea out to the periphery of the lung, the bronchi branched 16 times. That was normal. And she saw with patients that had the mildest form of bronchiectasis, where the bronchus was bigger than the pulmonary artery, she called it cylindrical bronchiectasis, they still had those 16 branches.

She saw that patients with varicoid bronchiectasis, which was the next worst type of bronchiectasis with undulating curves, what was different about them is that instead of having 16 branches, they now had 8 branches, so they were being truncated, the branches. And she called the worst type of bronchiectasis, the one with the most truncation, she called that cystic bronchiectasis, because there were only 4 branches from the time of the trachea to the periphery of the lung. And I thought that's a very helpful way of looking at bronchiectasis.

She only saw 16 branches in 1950. Currently, we see at least 26 branches from the trachea to the periphery of the lung. We have 8 conducting airways that are segmental. We have 8 subsegmental conducting airways that are not involved in oxygen exchange. We have 8 subsegmental respiratory bronchi, which are involved in gas exchange, and then the alveolar sacs and ducts. And just by the shape of them, you could imagine that's where tree and bud opacities are going to fill mucus in that area.

What is bronchiectasis? It has 2 components, bronchiectasis. We say it all the time, the word, but what is it truly? It's irreversible, so if it goes away, it wasn't bronchiectasis, and it's bronchial dilatation bigger than the accompanying pulmonary artery.

The secondary pulmonary lobule is the functional unit of the lung, and the lungs are all composed of these hexagons that measure 17 mm in size. And in the middle of each hexagon is a pulmonary artery and its accompanying bronchus, and usually they are the same size. So bronchiectasis is when that bronchus in the middle of the secondary pulmonary lobule becomes bigger than its pulmonary artery.

But we identify bronchiectasis many times incidentally on a CT scan. I'd like to give you a term called clinically significant bronchiectasis. That's when the patient has both clinical symptoms and bronchiectasis. The radiologist, in order to call clinically significant bronchiectasis, has to see one of the above findings. They have to see the inner airway diameter compared to the pulmonary artery being larger, or the outer airway diameter being larger than the pulmonary artery, or a lack of tapering or pruning, like Lynne Reid described, or if we see airways in the periphery of the lung within 1 cm of the pleura, we can call that bronchiectasis as well.

But to be clinically significant, it also clinically has to have at least 2 of the following findings: a cough most days of the week, sputum production, and a history of exacerbations. Then we can call it clinically significant. And the reason that is important is because we can classify the disease as a chronic respiratory disease.

What are the CT imaging findings of bronchiectasis? We see airway dilatation bigger than the pulmonary artery, a so-called signet ring sign, and we'll see pictures of that, a lack of tapering of the bronchus. It just stays dilated along its whole course, it doesn't get smaller as it goes to the periphery, and we call that tram tracking. Bronchi in the periphery of the lung, those are primary imaging findings of bronchiectasis.

But there are additional findings that we might see with bronchiectasis. The bronchial wall becomes thickened and irregular, then we think about chronic bronchitis, mucoid impaction, like I showed you in that first image with tree and bud-like opacities. We're going to think about MAI and mosaic attenuation and air trapping. Mosaic attenuation is a word commonly used. It just means heterogeneous. We say it on an inspiratory CT scan, and air trapping we should reserve for expiratory CT scans.

Things to be worried or thoughtful about. When we measure the internal airway diameter of a bronchus, if there is adherent mucus, it might seem artificially narrowed. And in the same way, when we're measuring the outer diameter of a bronchus, if there's a lot of wall

thickening, it might seem artificially larger in size.

In this picture, there are 6 signet rings. I'd like you to try to find those 6 signet rings. See how this is so beautifully done here, this signet ring. I'm going to give you one of them to begin with. You see that this bronchus is bigger than its accompanying artery. It looks kind of like a fancy ring where you put your finger in this part and you have this gemstone. So the signet ring, that's one of them. Did you find the other 6? Yeah, you did already. Here's a signet ring, there's a signet ring, a signet ring, a signet ring, a signet ring. In order to see a signet ring, the bronchus has to be following perpendicular to the axial image. When they are traveling in parallel and in the plane of imaging, then we're not going to see the signet ring sign.

Here we see examples of signet rings in 2 patients, one where the signet ring over the bronchus is very thin-walled, and the other where the bronchus is thick-walled. And I could imagine that these patients might present with different symptoms. So it's not only important for us to describe that the bronchus is big, bronchiectasis, but also that the bronchus is thick-walled.

I meant to ask Ashwin earlier if they present differently, would patients cough more maybe with bronchial wall thickening? Would it be wetter, the cough?

Lack of tapering, we call tram tracking. So usually, as the bronchus goes out to the periphery of the lung, it gets more and more narrow. When we lose that tapering, that's another sign of bronchiectasis.

Here we see it on the chest X-ray, right? You have to have a high index of suspicion to see this on the chest X-ray, so look carefully for this tram-track sign of cylindrical bronchiectasis.

Here we see another example of tram tracking with thickening of the walls of the bronchi. This is chronic right middle lobe atelectasis.

Bronchi in the periphery of the lung. If you see bronchi at all within 1 cm of the periphery of the lung, it means they're dilated. Usually, we can't see the bronchi in the periphery of the lung, so this is an inherent disease of the bronchi. We're seeing them in the periphery of the lung.

But be careful not to confuse it with pulmonary fibrosis. In pulmonary fibrosis, there is reticulations, right? These linear densities which represent thickening of the interlobular septa. And there's a lack of surfactant with pulmonary fibrosis that causes atelectasis of the alveoli and pulls open the bronchi. So when the bronchi are being pulled open from fibrosis, it's not an intrinsic disease of the bronchus itself like it was in the previous case; this is a problem with the fibrosis causing the bronchiectasis.

When you look at this CT scan, if it came across your desk, what would you describe? Do you see the signet ring? Over here we see a dilated bronchus bigger than its accompanying artery. We see bronchial wall thickening. We see tree and bud opacities. We see atelectasis. We don't see mosaic attenuation, but we see multiple features of bronchiectasis, and we should report those.

Mosaic attenuation can be challenging. Mosaic attenuation, again, on an inspiratory CT scan just means that the patient has heterogeneous attenuation. We don't know what's causing the heterogeneous attenuation. The way that we try to figure out what the cause is, is that we look at the dark area. You see that this area is a little bit darker than the other area, and then we look at the size of the blood vessels in the dark area. If the blood vessels in the dark area are smaller than the blood vessels in the white area, it means that the dark area is the sick area and that there's either small airways disease or there's pulmonary artery hypertension with pruning of the pulmonary artery.

Now, how can we differentiate between small airways disease and pulmonary artery hypertension as the cause for this black area? Well, we can measure the pulmonary artery. I use 33 mm for the size of the pulmonary artery that I consider enlarged, but I think more importantly is the pulmonary artery to aorta ratio, and if it's greater than 1, I think I'm dealing with pulmonary artery hypertension. But if this patient doesn't have that, then they don't have pulmonary artery hypertension, and the low-density area is likely due to small airways disease.

On the next CT scan image, I can see the same thing. Again, I see this low-density area, and I can look at the vessels and see how small they are compared to the high-density area. So I believe the disease is the black area, and it's either due to pulmonary artery hypertension or small airways disease. And the pulmonary artery is normal in this patient, so I believe it's small airways disease. And I

could do an expiratory CT scan, and if it gets worse, then it is small airways disease, as it does in this case. If the vessels were the same size in the white part as the black part, then the white part would have ground glass, and that would be the reason for the mosaic attenuation.

There are a lot of etiologies for bronchiectasis, but most of them are going to be chronic disease. We have chronic inflammation from asthma, chronic infection maybe from aspiration, chronic obstructive pulmonary disease, chronic immune deficiency, chronic mucociliary dysfunction. All chronic diseases are going to destroy the bronchi and cause bronchiectasis.

Here we see examples of how bronchiectasis progresses. In 2019, the first CT scan shows these nodules in a tree and bud-type of a pattern, which means there's mucus in the distal airways, the 24th to the 26th branch, first described maybe with tuberculosis, but any infection or aspiration can cause this tree and bud pattern, which over time leads to bronchiectasis.

This patient in 2019 has an equal bronchus-to-pulmonary-artery diameter and develops bronchiectasis in 2025 due to MAI infection. MAI infection we call Lady Windermere's disease. At least, the thinking is that it might be in older women due to a polite cough, you know, “eh, eh, eh,” instead of a rigorous cough. My mother is 88, and when she coughs like this, “eh, eh, eh,” I say, Ma, bring it up. Cough more rigorously so you don't get this disease. And I'll let her know I talked about her during my presentation. So we can see how it can lead to bronchiectasis, typically in the right middle lobe and lingula, but also in the lower lobes.

But be careful, right? This patient on A, I would say for sure they had bronchiectasis and bronchial wall thickening and tree and bud opacities, but after a course of antibiotics, they got better, so this wasn't bronchiectasis. Another thing I would be careful about is things that make the pulmonary artery smaller might make the bronchus look artificially larger.

AI is affecting all of our lives. We're seeing more and more of the role of AI. It's hard. Lynne Reid quantified one bronchus and said that it was cylindrical bronchiectasis or varicoid bronchiectasis or cystic bronchiectasis. But really what's important is how many bronchi are sick, and what AI allows us to do is to quantify the number of bronchi that are big and get a better idea of how sick somebody is with bronchiectasis.

And this eloquent study showed that the more bronchiectasis there was, the more likely patients would have exacerbations, so it helps us to counsel patients about how they're going to do over time.

What AI does is it measures the inner wall of the airway or the thickness of the airway and compares it to the pulmonary artery in a way that it would take us hours to do on a CT scan and allows us to stratify patients.

So to summarize, many patients report cough and dyspnea. In our ED, 10% of the patients come with this report, so we're going to see it all the time, cough and dyspnea. The chest X-ray is not sensitive. So many times with that diagnosis I say it's a normal chest X-ray and wonder why is the patient having this cough and dyspnea.

CT provides us with an opportunity for the early diagnosis of bronchiectasis, so we should definitely say that we see the bronchiectasis, we should say that the wall is thickened, and we should say the ancillary findings, mosaic attenuation and tree and bud opacities, and recommend a pulmonary consultation for early intervention, because that's the best for the patient.

Chapter 3: HRCT Pattern Recognition to Inform Etiology

Dr. Suh:
All right, thank you, Mary. That was a fantastic talk. Please feel free to send questions into the chat. At some point during this hour, we'll answer your questions.

So I'm going to quickly cover HRCT pattern recognition to inform etiology. So in addition to what Dr. Salvatore has excellently covered, bronchiectasis can be classified morphologically. And so if you look at the diagram on your right, at 12 o'clock, you see a normal bronchus that tapers kind of naturally from central to peripheral. Now, as that airway becomes abnormal, we lose that tapering, and so this is what we refer to as cylindrical or tubular bronchiectasis. And as the airway gets more and more damage, we have these outpouching or undulations. In general, these outpouchings are going to be smaller than the transverse diameter of the airway, and this is what's known as varicose or varicoid bronchiectasis. And finally, the most severe form is when these outpouchings exceed that of the

transverse diameter of the airway, and this is what is the saccular or what's referred to as cystic bronchiectasis.

Now, unfortunately, the morphologic classification of airways doesn't necessarily correlate with the etiology, and so that's why a pattern-based approach when you see bronchiectasis is important.

Now, there are many pattern-based approaches out there, and this is just one relatively simple one by seeing, that we'll kind of go through, and this will help you narrow down a differential diagnosis or radiographic diagnosis as to what that etiology of bronchiectasis may be.

So in this classification, there's certainly focal and diffuse bronchiectasis. Often diffuse bronchiectasis is the result of a systemic or more pervasive issue, and when you have diffuse bronchiectasis, it's important to look and see, is there a lobar predilection, or is a lobe predominantly affected more than other areas? Now, if that's not the case, you may also look at sort of is there a bronchial division pattern? So are we dealing with airways that are more central to the lobe, whether it's the one-third to two-thirds of that lobe or in the outer third of the lobe.

Now, if we look at focal bronchiectasis, it's often caused by a structural issue. And most commonly, if we're dealing with intramural, this is something within the airway, and so you could have a foreign body. This is particularly true in the pediatric population, but you also may have broncholithiasis or some other things that get into the airway. And certainly, we're going to have benign and malignant tumors. And if you look at those images here, you can see that there's a nice image of carcinoid occupying that airway lumen and causing obstruction.

Now, if you're dealing with mural causes, often this is the result of repeated inflammation, infection that causes stenosis or even obliteration of that airway and causes bronchiectasis in the draining pathway. Now, in addition to these inflammatory, infectious causes, you may have congenital issues or causes of why that airway is stenotic or obliterated. And with bronchial atresia that you see in the other images here on the slide, there's a no communication that develops between the more central bronchi and the subtended lobe or the segment. And you can see there's a dilated bronchus, certainly in the left upper lobe. Sometimes this is completely mucus filled or mucocele, other times it might have a little air and mucus, as you see in this image. And certainly, you can have extramural causes, particularly lymphadenopathy or primary lung cancer.

So if we're dealing with a more diffuse pattern of bronchiectasis, the first thing to sort of look for is: are some lobes more involved than others? And so if we have upper lobe predominance or predilection, we think of diseases like cystic fibrosis primarily, but we can also think of allergic bronchopulmonary aspergillosis, as you see in those 2 images on your left.

Now, with granulomatous diseases, there's often repeated inflammation, infection of not only airways but also the air spaces, and this results in bronchiectasis, and this could be accompanied in diseases such as sarcoidosis or post-tuberculosis. And certainly, radiation can affect any part of the lung, but in this case, if it was given in the upper lobe, you might see focal bronchiectasis here, as in this patient that was radiated for a small cell lung cancer.

Now, if you have diffuse bronchiectasis with a middle lobe predilection, as referenced in the first talk, you might think of nontuberculous mycobacteria. But other common diseases, such as asthma, or less common diseases as primary ciliary dyskinesia, may also have a middle lobe predilection. And so if you look at these 3 images, you can see that, yes, there is airway disease throughout in both lower lobes, middle lobe, and lingula, but you can clearly see that the middle lobe is most involved in all 3 of these processes.

So with nontuberculous mycobacteria, if we have what's called the non-classical presentation of nontuberculous, this is known as Lady Windermere syndrome. It's often seen in middle-age to elderly females that are maybe slight in stature or tall. It's thought to have resulted from volitional cough suppression but may also be associated with physical features such as scoliosis, pectus excavatum, and even mitral valve prolapse.

So why are the middle lobe and lingual bronchi more susceptible to this? Well, it's because these airways are narrow, they're angulated, and often are in horizontal position. And it's not uncommon for mucus to pool in these areas, which provides fertile ground for colonization and ultimate infection.

As time goes on, bronchiectasis and bronchiolectasis eventually develop, with, of course, airway thickening and more mucostasis or

mucus impaction. And with coexistent infection or more active infection, you'll see centrilobular nodules, or what we commonly refer to as tree and bud, and even formation of cysts, which are often outpouchings of airways as well.

And so in these 2 examples, you can see that, again, there's bronchiectasis and airway thickening throughout and even centrilobular nodules, but you see that the middle lobe is particularly affected, and you can see nice examples of cystic bronchiectasis.

Now, with lower lung diseases, there are a number of things that could sort of cause this, particularly aspiration, as in this example on your left, where the patient had a hiatal hernia, was repeatedly aspirating from gastroesophageal reflux disease. In the bottom, you have a quadriplegic patient who has chronic ventilator-associated pneumonia with essentially scarred-down lower lobes with bronchiectasis.

Immunodeficiency is also a particular etiology that gives you a basilar predilection for bronchiectasis. So here we've got a patient with hypogammaglobulinemia with chronic pseudomonas infection of the airways, and below that we have common variable immune disorder or deficiency with classic granulomatous lymphocytic interstitial lung disease.

Just as primary ciliary dyskinesia can have a middle lobe predilection, it can also have a lower lung predilection, as in this example here. Certainly, with basilar bronchiectasis, you want to think about diseases such as alpha-1 antitrypsin deficiency. And finally, you may see bronchiectasis in the setting of basilar interstitial lung disease.

Now, in this situation, it's important to distinguish what we know as traction bronchiectasis associated with fibrosis from an airway intrinsic phenomenon that is really more innate to the airways. And so if you look at the image on your left, you can see that there's a really highly abnormal airway there, marked by the arrowhead, that, again, look very serpentine, corrugated.

Now, clues to why or how you may be dealing with bronchiectasis from traction is that look at the lung tissue immediately outside of the airway, and clearly this is not normal lung parenchyma. And when you see this, you should put 2 and 2 together and think, huh, maybe the reason why this airway is abnormal is because the fibrosis or the abnormal lung is pulling apart that airway. Another clue is for how ugly this airway looks, there's not a proportional or commensurate amount of airway thickening. And so those things are probably your best clues to think about traction. And you can compare and contrast this with the more normal airway in the CT image in your middle, but particularly the airway intrinsic phenomenon that you see way here on the right.

So here you see a lot of peribronchial thickening, dilation, but notice that there's a lot of airway thickening for proportional to how abnormal that airway is, and most importantly, the lung parenchyma immediately outside of the airway looks fairly normal.

So when there's no lobar predilection that you can easily identify, then you might want to switch gears and think about what portions of the airway within the lobe are affected. Is it the middle one-third to two-thirds of the lobe versus the outer one-third? Now, if you're dealing with central bronchiectasis, you might think of ABPA, and you can also, if the trachea and the main stem bronchi are also dilated, then things like Mounier-Kuhn syndrome come to mind. Now, if you're dealing with peripheral bronchiectasis, meaning that these are segmental or smaller airways predominantly affected, then a diagnosis that comes to mind is Williams-Campbell syndrome.

Now, one of the things that you can do very nicely is to use minimum intensity projection images, as you see in these 2 coronal images. And this really highlights the air within the airways, and so when you have diffuse disease, it really makes it easy to identify where the abnormality is most prevalent.

So there are certainly natural complications that go with the bronchiectasis. Certainly, in this case, we have a case of ABPA with this high attenuation characteristic mucus plugging. You may have certainly superimposed infection, as manifested by clustered centrilobular nodules or tree and bud, but you might also have just frank airspace consolidation as in the image here with ABPA. So you have in the right lower lobe the characteristic tree and bud nodules; you have in the left lower lobe airspace consolidation.

In addition to infection in the cases of ABPA, you should think of entities like bronchocentric granulomatosis. And when you have air fluid levels in abnormal airways, you really want to think about, or at least in these situations, there's probably an active airway infection going on.

All right. So air leak complications can also accompany bronchiectasis, pneumothorax and bronchopleural fistula, as you see in this case of cystic fibrosis. You have a right pneumothorax. If you look at the coronal images, you can see that there's an abnormal or

bronchiectasis in the periphery of the upper lobe that communicates with the medial pleura, representing a bronchopleural fistula.

Over time, with inflammation and repeated infection, you can lose the parenchyma in between the airways where basically it's just scarred up, and it looks almost like you have sticks bundled up. And here you can see in the left upper lobe a nice example of this in cystic fibrosis. Because the airways and lung become repeatedly insulted over time, it's not unusual to have obliterated form pulmonary hypertension, as in this case with central dilated pulmonary arteries.

Now, the European Respiratory Society suggests a minimal bundle in the workup of these patients. So this includes a differential blood count, serum immunoglobulins, test for ABPA and aspergillosis, *Aspergillus*, sputum culture for bacteria, AFB, and fungus. And although this is not a part of the bundle, it is often sent, is an alpha-1 antitrypsin phenotype. All of these patients with bronchiectasis should at least get a high-resolution chest CT with inspiratory and expiration, as well as pulmonary function tests. For cystic fibrosis, a CFTR gene analysis as well as sweat chloride tests can be performed. And for ciliary dyskinesia, nasal nitric oxide is a very efficient and cost-effective way to diagnose this, as well as electron and high-speed video microscopy.

So in summary, bronchiectasis results from various etiologies. Early diagnosis is critical to prevent chronic airway and parenchymal complications, as we ran through, and a pattern-based approach facilitates a radiologic or radiographic diagnosis.

Thank you.

Chapter 4: Early Identification, Early Treatment: New and Emerging Therapies for Bronchiectasis

Dr. Suh:
All right, so our next speaker, Ashwin, he'll be speaking on early identification, early treatment, new and emerging therapies for bronchiectasis.

Dr. Basavaraj:
Thank you, Dr. Suh. It's a pleasure to be here today. I'm going to be talking about some of the new therapies that are coming out in bronchiectasis and NTM.

So as we heard earlier from our prior 2 speakers, it's important to investigate for potential etiologies of bronchiectasis. If we try to identify what's causing their bronchiectasis, then we can then treat it appropriately and try to prevent further progression of the bronchiectasis.

So some of the etiologies are genetic, for example, cystic fibrosis. We can diagnose mild forms of CF in adults, and we also can identify CFTR-related disease with one CF mutation and a positive sweat chloride test. We've gotten more aggressive about trying to get modulator therapies for these patients, which can help prevent progression of their bronchiectasis. Immunoglobulin deficiencies like primary immunodeficiency or common variable immunodeficiency, aspiration and esophageal dysfunction, as we heard, ABPA, primary ciliary dyskinesia, and autoimmune causes.

So when we have patients with bronchiectasis that we see in the clinic, we're really aggressively trying to check for these etiologies and try to treat them appropriately. We also, for almost all our patients with bronchiectasis and NTM, put them on airway clearance therapy to help relieve their mucostasis and try to help decrease the inflammation and the infectious burden in their lungs. And a common airway clearance regimen is hypertonic saline, 7% saline, with the positive expiratory pressure device, such as an Aerobika or an Acapella device.

Now, it's not an easy decision to treat NTM if we have it in front of us. A lot of NTM that patients present with can be indolent, they may not have symptoms, and they can have very mild forms of NTM where just airway clearance therapy in and of itself can help control it.

But there are some risk factors that, when present, can help the NTM progress in patients, and we're really looking on whether these risk factors are present to decide whether to treat patients with antibiotics for NTM. And some of these risk factors for progression are weight loss, older age, presence of cavitary disease or smear positivity on their AFB sputum, which tells you there's a high burden of infection and they're likely to progress. So if these factors are present, we then have a low threshold to treat with antibiotics.

Now, there are a number of antibiotic regimens that we can choose from, and it depends on the severity of their bronchiectasis and NTM.

For patients with mild to moderate bronchiectatic disease without the presence of cavities, we can consider a macrolide, a rifamycin, or ethambutol 3 times a week. And for patients that have a more severe form of bronchiectasis or the presence of cavitary disease, we use the same antibiotics every day, and then we consider the addition of an IV aminoglycoside such as IV amikacin if they have the presence of large cavities.

Now, these antibiotics, you know I tell my patients, they're not a magic regimen. They don't get rid of the NTM in 100% of patients. Actually, in patients who are macrolide sensitive, about 50% of patients can achieve culture conversion even after successful therapy for over a year. So there's a lot of newer therapies that are being looked at to try to help improve the outcomes for these patients with NTM.

One of the therapies that was approved by the FDA in 2018 is nebulized, inhaled liposomal amikacin, where the amikacin through a nebulizer is delivered with a liposome that goes into the macrophages in the lungs, where NTM likes to hide out. And what this study showed was that in patients who were with refractory NTM, meaning they tried 3 drugs for about 6 months and they were still culture-positive, those patients had inhaled liposomal amikacin added on to guideline-based therapy, and they had improved culture conversion compared to patients that just received 3 drugs alone. About 29% of patients culture converted after 6 months, compared to 9% of patients who just continued guideline-based regimen alone.

Now, inhaled liposomal amikacin, some of the newer data has shown that even with successful treatment and completion of their therapy, that culture conversion can remain negative even after stopping therapy. About half the patients who received inhaled liposomal amikacin along with guideline-based therapy remained culture-negative at 3 months, and about 46% of patients remain culture-negative after 1 year of therapy.

Now, some new data that was published a few weeks ago looked at inhaled liposomal amikacin at the start of therapy, in addition to a macrolide and ethambutol versus azithromycin, ethambutol, and placebo, and showed a much better culture conversion rate in patients that received inhaled liposomal amikacin-based regimen compared to the placebo group, so potentially a newer regimen in the pipeline where that can improve some of the outcomes in patients with NTM.

Now, with bronchiectasis, you know we think about it as a structural lung disease with airway dilatation and lack of tapering to the periphery, but there's been a paradigm shift recently in bronchiectasis, and is being considered more as a chronic inflammatory disease with neutrophilic inflammation, the predominant driver of inflammation in bronchiectasis.

And with neutrophilic inflammation in bronchiectasis, we get the formation of what's known as neutrophil extracellular traps, or NET, formations, which really are a meshwork of DNA, histones, and protein that trap some of these aggressive organisms such as NTM, pseudomonas, and MRSA, and effectively traps these organisms, but ineffectively gets cleared out of the lungs and gets stuck in the airways. And when these NETs get stuck in the airway, that leads to further inflammation and further bronchiectasis. So a lot of therapies are really trying to focus on reducing neutrophilic inflammation and reducing the formation of these neutrophil extracellular traps.

And when we talk about bronchiectasis, it’s important to understand what a bronchiectasis exacerbation is, because a lot of the clinical trial endpoints are really focusing on trying to reduce bronchiectasis exacerbations. So what is a bronchiectasis exacerbation? It's really, in a nutshell, a worsening of their bronchiectasis clinically. But the definition, which was published in ERS a number of years ago, suggests that a bronchiectasis exacerbation is deterioration of 3 or more symptoms: a cough, worsening sputum volume, sputum purulence, shortness of breath, fatigue, hemoptysis, and a change in therapy that's required, oftentimes antibiotics that's needed for the bronchiectasis exacerbation.

So with that, there was the recent phase 3 trial that was published in the *New England Journal of Medicine* looking at an anti-inflammatory, brensocatib, which targets neutrophilic inflammation. It's an oral reversible inhibitor of what's known as dipeptidyl peptidase, which is an inhibitor of neutrophil elastase, which is a driver of neutrophilic inflammation. And what this study showed was that it looked at 2 doses of brensocatib, 10 mg, 25 mg, and placebo, and in both doses found a reduction in bronchiectasis exacerbations and increased time to their first exacerbation, and an increased number of patients that remained exacerbation free if they received brensocatib. And in the higher dose, the 25-mg dose, some additional improved outcomes with the slower rate of decline of FEV1 and improved quality of life as well.

So the FDA did approve brensocatib last year for treatment of patients with bronchiectasis, and we oftentimes start with the higher dose, 25 mg, because there's not a lot of differences in terms of side effect profile with the 10-mg dose, and then obviously, if they have difficulty with tolerating the 25-mg dose, we'll back down to 10 mg.

So it's really an exciting time in bronchiectasis. We've had new therapies that are approved and a number of clinical trials that are ongoing right now, a lot focused on reducing neutrophilic inflammation in bronchiectasis, and some studies also looking at monoclonal antibodies, typically that have been used in asthma or COPD are now being thought that they may have benefit in bronchiectasis also. So a number of exciting new therapies in the pipeline.

And there's also new initiatives in bronchiectasis. There was the recent formation of the Clinical Care Networks nationally, which are designated centers of excellence throughout the country through the bronchiectasis and NTM Association. There's about 30 centers and 6 associate centers. There's patient support networks for NTM that have been developed and a number of educational sessions as well that are in the pipeline. And we are hosting the 3rd North American bronchiectasis and NTM symposium in New York. So for those that are interested in learning more about bronchiectasis, this is a great venue to get some more high-depth knowledge there. And also a patient education program as well, which is open for patients nationally, for those that are interested in learning more about their bronchiectasis and NTM.

So in summary, bronchiectasis is a progressive inflammatory condition. Early identification is crucial to prevent worsening disease. There are anti-inflammatory therapies that have been recently approved, and there are more that are in the pipeline. There are new treatment regimens for NTM that are emerging, and several clinical trials and educational initiatives that are ongoing.

Thanks, everyone.

Chapter 5: Panel Discussion and Audience Q&A

Dr. Suh:
Okay, all right, thank you for that. Very enlightening. Well, we'll do a little panel discussion, if you will. But I think we're all in agreement that bronchiectasis needs to be reported, right? And now it becomes more than just an academic exercise, given that there are more and more and effective new therapies potentially coming down the pipe.

So maybe if we can start with Mary, so how do you think we should go about reporting bronchiectasis in our reports in general, whether you're a general radiologist or a chest radiologist?

Dr. Salvatore:
I think it's so important to have it as part of your search to start with. So if you don't look for it, you won't find it. So every time, I think I start with that so I don't forget to look at it. And then in my report template I have a line about addressing whether there is bronchiectasis or not. And I think about bronchiectasis and then I think about bronchial wall thickening separately and make sure I think to include both of them in my report.

Dr. Suh:
Okay, great. Great. And I think you know at least that initial polling question kind of showed that it was a little bit mixed. Some people just maybe recommend follow-up and other people actually mention in the report. So, Ashwin, as a clinician receiving these radiologic reports, good or bad, what would you hope to see that really helps you better steer that patient?

Dr. Basavaraj:
Yeah, so I'm looking at it from a general pulmonologist point of view. When they see bronchiectasis on a report it oftentimes gets ignored and they don't think about some of these chronic infections such as NTM when there's bronchiolitis that's present. So I think it's important to take a next step in the reports and say consider sending an AFB sputum culture. Consider NTM on the differential or considering, as Mary mentioned, a referral to pulmonary. I think that really gives the general pulmonologist some direction in terms of what are the possible differential diagnosis and oftentimes ensures that the bronchiectasis or NTM doesn't get ignored.

Dr. Suh:
So you would appreciate maybe a little bit of an etiology of why we think the airway may be abnormal. And I think that's why the pattern-

based approach is kind of useful because at least it puts you into the ballpark. And so of course, people like Ashwin are going to pick up on the bronchiectasis right away from the report, but we have so many patient-facing physicians in the primary care world that may not be so sensitive to that so a little bit of steering may help facilitate that, right?

Dr. Basavaraj:
Absolutely.

Dr. Suh:
Okay, all right. Well, do you have any other things you want to add about reporting or what do you think some of the barriers are to why we don't report nearly as frequently as we should in some cases?

Dr. Salvatore:
I think sometimes we focus in on what the clinical history is. So like a PE study, it might be the best opportunity. The patient's short of breath because of the bronchiectasis and not a PE. So I think always considering it. Like on that chest X-ray where I showed the tram tracking, I wouldn't have picked that up. But I have to have a high index of suspicion and assume that every patient could have bronchiectasis. It's such a common disease.

Dr. Suh:
All right, very good. Well, in the remaining time that we have, we'll do some of these questions. And so here, these first 2 questions are a little bit similar, or maybe they're close cousins. So if the dilation has to be irreversible to be called bronchiectasis, how can you call early mild bronchiectasis on a single CT without priors? It might be reversible. And the sort of the corollary question is any pointers towards calling dilation of bronchi transient dilation versus bronchiectasis prospectively?

And, Mary, do you have any hints or maybe strategies or wisdom?

Dr. Salvatore:
I think it's analogous to lung cancer screening. When we see a suspicious, maybe, nodule but it could be infectious or inflammatory, following it up in a short time and if it persists then consider it to be something that needs to be worked up for lung cancer. And then maybe with the same way, bronchiectasis, we shouldn't use the word bronchiectasis. So the bronchi is dilated, could be infectious or inflammatory, recommend follow-up in a short period of time. And then if it persists, we could start using the word bronchiectasis. I think it's kind of like with atelectasis and scarring too. I used the word atelectasis in my early reports, and then if it persists, I use the word scarring. So maybe that's a way we could use that term to make sure we're not misusing it.

Dr. Suh:
The next question is many of these entities present in childhood. Which guidelines are the best to bridge that transition between child and adult therapies and long-term follow-up? So that might be something that—

Dr. Basavaraj:
Yeah. No, it's a great question. I think when we think about childhood bronchiectasis and adult bronchiectasis, some of the genetic conditions come into mind such as cystic fibrosis. And when we talk about guidelines, there's not a lot of international guidelines on bronchiectasis that are out there. The main one that's often quoted, as Dr. Suh mentioned, the European Respiratory Society guidelines, which was recently revised in 2025. So that's the main international guideline that's quoted. CHEST is also coming out with bronchiectasis guidelines hopefully published later this year. But ERS is normally kind of the gold standard with bronchiectasis guidelines and really refers to adult management there pretty nicely.

Dr. Suh:
Right. No, that's helpful. And we have this last question here. What does consumption of chlorinated water with mycobacterial infections —I guess they're asking if there's any association with that?

Dr. Basavaraj:
Yeah. I mean, so we know NTM is prevalent ubiquitously in the environment and many of us can kind of fight off NTM. But you know there's certain factors where patients cannot fight off such as immunodeficiencies and such. And NTM likes to hide on water sources; it likes to hide on soil. So it's important to just explain some of those risk factors to patients such as old shower heads, indoor swimming

pools, hot tubs. So just so patients can be cognizant of it and essentially try to help replace some of those risk factors in case they're present.

Dr. Suh:
Yeah, I think, yeah MAI, nontuberculous, it's just prevalent so it's everywhere. So I'm not sure if it's really avoidable or not, per se.

Okay. Are there any questions from the audience? Please feel free to come up and use the mics. Yes?

Audience member:
Do you have any local treatment endoscopic for the bronchiectasis?

Dr. Suh:
I don't know. I couldn't really hear.

Dr. Basavaraj:
Yeah. I think the question is, is there any sort of endoscopic treatment for bronchiectasis. So the short answer is, no, we hardly actually perform bronchoscopies in patients with bronchiectasis. We really try to get sputum induced in the clinic with nebulized saline, and if they're really nonproductive we'll perform a bronchoscopy to get some culture data. But outside of that, it's rare that we need to do a bronchoscopy in these patients.

Audience member:
Like a local antibiotic therapy or something instilling drugs into there?

Dr. Basavaraj:
Yeah, no we're not there yet. It's an interesting thought and question and maybe a future research question can help answer that. But right now, our antibiotics are either oral, IV, or nebulized.

Dr. Suh:
All right, I think if there are no other questions we'll close out the session. I think this is a really exciting time where your reports actually do matter to help direct patients to the right specialists or subspecialists and because there are, more than ever, therapies out there that they benefit from.

Thank you for your attention and joining us during this lunch hour.

Announcer:
You've been listening to a replay of a live broadcast discussing improving your ability to differentiate the radiologic patterns of bronchietasis from other lung diseases This activity was provided by Prova Education and is supported by an independent educational grant from Insmed.

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