

When bronchiectasis, reflux and nontuberculous mycobacterial pulmonary disease intersect: An aspiration-focused practice pearl

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In patients with bronchiectasis who also have reflux and nontuberculous mycobacterial (NTM) pulmonary disease, the most useful approach is to treat reflux as a possible aspiration phenotype rather than simply an acid problem. A practical approach is to confirm NTM disease, protect airway clearance from reflux, use objective reflux testing prior to proton pump inhibitor escalation, screen for occult dysphagia when clues are present and reserve surgery or off-label therapies for carefully selected patients with documented reflux.

Keywords

Bronchiectasis, exacerbations, gastroesophageal reflux disease (GERD), non-tuberculous mycobacteria (NTM), proton pump inhibitors (PPI), pulmonary disease

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A 68-year-old woman with nodular bronchiectasis presents with persistent productive cough, intermittent regurgitation and recurrent 'chest infections' despite 12 months of once-daily proton pump inhibitor (PPI) therapy. Her clinician has doubled the PPI dose and is considering long-term azithromycin. Sputum has not been sent for mycobacterial culture in over a year. She has lost 3 kg, at times endorses coughing during meals and prefers to sleep upright. This all-too-common real-world patient scenario illustrates the practical decision points that follow, which include clarifying the microbiology before initiating chronic macrolide therapy, phenotyping reflux rather than escalating acid suppression, looking actively for occult dysphagia and adapting airway clearance so that it does not provoke regurgitation.

Why this phenotype is hard

Bronchiectasis, reflux and nontuberculous mycobacterial (NTM) pulmonary disease frequently coexist, but the challenge is deciding which problem is actually driving symptoms on a given clinical presentation. Reflux may worsen cough, regurgitation and microaspiration; bronchiectasis itself promotes cough and mucus retention; and NTM pulmonary disease adds a prolonged infectious and inflammatory burden that is often hard to distinguish from routine bronchiectasis instability.¹⁻⁵ Clinicians may pursue treatment with repeated acid suppression while under-treating mucus burden, missing occult aspiration or dysphagia or delaying formal NTM evaluation. NTM pulmonary disease is included in this practice pearl, rather than treated as a coincidental comorbidity, for three reasons that are directly relevant to an aspiration-focused approach. First, aspiration-prone airway environments and impaired mucociliary clearance may plausibly facilitate NTM acquisition and persistence through repeated low-level mycobacterial inocula.^{3,5} Second, NTM-related symptoms (chronic cough, sputum change, post-tussive regurgitation and weight loss) are routinely misattributed to refractory reflux, leading to repeated PPI escalation, while the microbiologic driver is missed.^{2,3} Third, the most common therapeutic shortcut for unexplained cough in bronchiectasis, long-term azithromycin, is the intervention that mandates prior NTM exclusion, so any reflux-and-cough algorithm in bronchiectasis cannot responsibly bypass mycobacterial assessment.^{1,2} The clustering of upper-airway and upper-gastrointestinal pathology with bronchiectasis has been recognized for decades: chronic rhinosinusitis is highly prevalent in non-cystic-fibrosis bronchiectasis cohorts,⁶ and upper-gastrointestinal symptoms were documented as a feature of bronchiectasis populations more than two decades ago.⁷ These observations reinforce a whole-airway, aspiration-focused framing rather than an isolated lower-airway one.

The pearl

The most useful practical pearl is to manage this triad as a potential aspiration phenotype rather than as 'gastroesophageal reflux disease (GERD) plus lung disease'. That framing changes several decisions at once. Symptoms alone are poor guides: objective studies show that reflux in bronchiectasis is often clinically silent.⁴ Just as importantly, PPIs reduce acid exposure but do not address non-acid reflux, cough-related regurgitation or aspiration risk, so persistent respiratory symptoms after a PPI trial should not automatically trigger more acid suppression.⁸⁻¹⁰

Table 1: Practical decision points in the bronchiectasis-reflux-nontuberculous mycobacterial triad

Clinical trigger	Recommended action	Evidence basis
Long-term azithromycin being considered	Exclude active NTM with at least one negative respiratory NTM culture before initiation; avoid macrolide monotherapy where NTM disease is established	Guideline (BTS, ERS 2025 and ATS/ERS/ESCMID/IDSA) ¹⁻³
Single positive NTM sputum culture	Repeat sputum mycobacterial culture; do not initiate NTM therapy on a single isolate alone; reserve treatment for those meeting microbiologic, clinical and radiologic criteria	Guideline (ATS/ERS/ESCMID/IDSA) ³
Persistent respiratory symptoms despite a PPI trial	Phenotype rather than escalate: objective reflux testing (off-PPI pH study or on-PPI impedance-pH; manometry if intervention contemplated); where testing is unavailable, refer to motility services rather than continued empiric escalation	Guideline (AGA and ACG) ^{8,9}
Cough during meals, post-meal 'chest infections', weight loss and unexplained lower-lobe disease	Speech-language pathology referral; consider FEES or videofluoroscopy; treat dysphagia screening as a core pillar of the aspiration evaluation	Prospective data in bronchiectasis ¹¹ ; expert opinion
Airway clearance provokes reflux or regurgitation	Switch to sitting-position technique (PEP, oscillatory and autogenic drainage); schedule clearance ≥2 h after meals and not immediately before lying down; review sequence of inhaled therapies	Partial guideline (BTS and ERS 2025) ^{1,2} + expert/pragmatic
Objectively documented reflux refractory to PPI	Multidisciplinary review; consider alginates, neuromodulators, baclofen (objective documentation required); reserve fundoplication or endoscopic antireflux for selected patients with shared decision-making	Guideline (AGA and ACG) ^{8,9} ; observational lung outcome data ¹⁰

ACG = American College of Gastroenterology; AGA = American Gastroenterological Association; ATS = American Thoracic Society; BTS = British Thoracic Society; ERS = European Respiratory Society; ESCMID = European Society of Clinical Microbiology and Infectious Diseases; FEES = fiberoptic endoscopic evaluation of swallowing; IDSA = Infectious Diseases Society of America; NTM = nontuberculous mycobacterial; PEP = positive expiratory pressure; PPI = proton pump inhibitor.

Thinking in terms of aspiration keeps the clinician focused on airway protection and microbiology, rather than escalating antisecretory therapy. It is important to be explicit about the evidence base. The aspiration-phenotype framework presented here represents a synthesis of recent gastroenterology guidance,^{8,9} bronchiectasis guidelines,^{1,2} NTM guidance³ and observational literature on reflux in chronic lung disease;^{4,5,10} however, high-quality prospective interventional data specific to the bronchiectasis-reflux-NTM triad remain limited. The recommendations below should therefore be read as a pragmatic, guideline-anchored approach informed by expert opinion (Table 1).

Step 1. Confirm the diagnosis you are treating

In bronchiectasis, sputum should be sent for routine and mycobacterial culture, ideally when the patient is stable.² This is especially important if long-term azithromycin is being considered, as the British Thoracic Society recommends ensuring that there is no active NTM infection, with at least one negative respiratory NTM culture, before chronic macrolide therapy is started; the 2025 European Respiratory Society (ERS) bronchiectasis guideline makes the same recommendation.^{1,2} When NTM is isolated, it is important to note that one positive culture is not the same as disease. The ATS/ERS/ESCMID/IDSA guideline recommends more than one positive sputum culture with the same species or subspecies because environmental contamination is common; clinically significant *Mycobacterium avium* complex (MAC) disease is unlikely after a single positive sputum culture but much more likely when two or more cultures are positive.³ Even when diagnostic criteria are met, treatment still requires an individualized discussion. Not every patient needs immediate antibiotics, but watchful waiting should be deliberate.³ Features that tilt toward initiating treatment include cavitory disease, progressive radiographic change (such as new or enlarging nodules, expanding tree-in-bud disease or worsening bronchiectasis on serial imaging), declining lung function, high mycobacterial burden on smear or culture, persistent constitutional symptoms (such as weight loss, fevers

and fatigue) and an inability to maintain effective airway clearance; these are weighed against frailty, polypharmacy and drug-interaction risk, anticipated tolerance of multi-drug regimens and the patient's own goals and preferences.³ A documented period of observation with structured symptom, weight, lung-function and imaging review is preferable to either reflexive treatment or open-ended deferral. If treatment is required, avoid the common error of letting reflux management distract from guideline-based NTM care. For macrolide-susceptible MAC pulmonary disease, current guidance favors a three-drug macrolide-based regimen, with intermittent therapy appropriate for noncavitory nodular/bronchiectatic disease and daily therapy preferred for cavitory or advanced bronchiectatic disease.³ Macrolide monotherapy should not be used to cover both problems as it risks resistance and may compromise future NTM options.^{2,3}

Step 2. Phenotype reflux before escalating proton pump inhibitors

In patients with extraesophageal symptoms, there is no single test that proves reflux is the cause of cough or lung injury.^{8,9} The American Gastroenterological Association (AGA) advises considering diagnostic testing before empiric PPI therapy in patients with suspected extraesophageal reflux who do not have typical reflux symptoms and recommends objective testing after a failed PPI trial rather than simply cycling through more PPIs.⁹ The American College of Gastroenterology (ACG) likewise recommends pH monitoring off PPI therapy when GERD has not been objectively established, impedance-pH monitoring on PPIs when established GERD remains symptomatic despite optimized therapy, and manometry when dysmotility is suspected or an intervention is being considered.⁸ We recognize, however, that impedance-pH monitoring and high-resolution manometry are not uniformly available and that referral pathways differ markedly between centers. Where these tests are not readily accessible, a pragmatic stratification is reasonable. In patients with classical heartburn or regurgitation and a corroborating

clinical picture, a time-limited PPI trial with structured pre- and post-trial assessment of respiratory and reflux symptoms can proceed first; if symptoms persist, this should prompt referral rather than continued empiric escalation. In patients with extraesophageal-dominant symptoms (cough, hoarseness and throat clearing), poor PPI response and/or features suggesting non-acid or volume reflux (post-prandial regurgitation and nocturnal aspiration episodes), referral to a center with motility services is preferred over repeated PPI doubling. The point is not that objective testing is mandatory in every case, but that the threshold for further empiric acid suppression should be lower than the threshold for objective phenotyping. Dysphagia screening should sit alongside reflux phenotyping as a core pillar of the aspiration evaluation rather than be treated as an optional add-on. At the bedside, a low threshold for swallow assessment is often more useful than another change in acid suppression. Recurrent 'chest infections' after meals, cough during eating, troublesome throat clearing, weight loss, frailty or unexplained lower-lobe disease should prompt consideration of oropharyngeal as well as esophageal aspiration. In idiopathic bronchiectasis, a recent prospective study found abnormal swallowing parameters in most patients undergoing fiberoptic endoscopic evaluation of swallowing, with aspiration identified in 12.7%; the authors recommended dysphagia screening because it may otherwise be missed.¹¹ In practice, speech-language pathology and gastroenterology input can be as valuable as any other prescription.

Step 3. Adapt airway clearance to protect the airway

Optimal bronchiectasis care remains anchored in airway clearance.^{1,2} The 2025 ERS guideline strongly recommends airway clearance techniques for most patients with bronchiectasis,¹ and the BTS guideline adds a practical point for this phenotype: if gastro-esophageal reflux worsens with modified postural drainage, the patient should be taught an airway-clearance technique in the sitting position.² Many patients benefit from separating airway clearance from meals and bedtime, avoiding head-down positioning and switching to positive expiratory pressure devices, autogenic drainage, or oscillatory techniques when cough and regurgitation are tightly linked.² In our practice, scheduling clearance at least 2 h after meals and not immediately before lying down reduces reflux-provoked coughing during the session and improves tolerability. The interaction with inhaled therapies also matters as where mucoactive nebulizers, hypertonic saline or bronchodilators are part of the regimen, the usual sequence (bronchodilator, mucoactive agent, airway clearance and then inhaled antibiotic) should be reviewed so that the patient is not lying flat shortly after a large fluid or aerosol load. Adherence is the second consideration that is easy to under-emphasize. The most physiologically optimal device is of limited value if the patient cannot or will not perform it daily, so device choice, session length, supervision by a respiratory physiotherapist and re-assessment of technique over time matter as much as the technique itself.^{1,2} The aspiration-prone patient who cannot tolerate prolonged head-down postural drainage is often well served by a positive expiratory pressure or oscillatory device performed sitting, with shorter but more reliably completed sessions. We acknowledge that recommendations around clearance timing, meal separation and modality selection in the reflux/aspiration context are

pragmatic and extrapolated from physiology and clinical experience rather than direct guideline endorsements. When sputum is tenacious, mucoactive therapy may help; however, recombinant DNase, which clinicians sometimes consider by extrapolation from cystic fibrosis practice when sputum tenacity is the dominant complaint, should not be used routinely in adults with bronchiectasis, where it has been associated with worse exacerbation and lung-function outcomes.^{1,2}

When conventional reflux therapy is not enough

Lifestyle measures matter, but their limitations should be stated. Weight loss in patients who are overweight, avoiding meals within 2–3 h of bedtime, elevating the head of the bed and sleeping on the left side can improve reflux symptoms, particularly at night.⁸ However, the same guideline acknowledges that support for many lifestyle interventions is limited or variable.⁸ That fits clinical experience in bronchiectasis: lifestyle changes are low risk, but they rarely solve chronic cough and suspected aspiration on their own. Similarly, PPIs remain appropriate for typical GERD symptoms and erosive esophagitis, but they are not a comprehensive solution for the patient whose dominant problem is regurgitation, volume reflux or aspiration.^{8–10} Before describing options for refractory reflux, it is worth noting which common recommendations are guideline-based and which are expert/pragmatic in nature. Lifestyle measures, alginate-containing antacids, neuromodulator and baclofen consideration, and the threshold for surgical referral are all directly addressed in the AGA and ACG guidance cited below.^{8,9} Recommendations regarding airway-clearance timing relative to meals and bedtime, sequencing of inhaled therapies in the aspiration-prone patient and modality selection on the basis of reflux provocation are pragmatic suggestions extrapolated from clinical experience, not guideline recommendations, and should be presented to patients as such. When conventional treatment is insufficient, alternative strategies can be useful in selected patients. The AGA notes that alginate-containing antacids, external upper esophageal sphincter compression devices, cognitive-behavioral therapy and neuromodulators may have a role in extraesophageal reflux syndromes.⁹ The ACG notes that baclofen can be considered for patients with objective documentation of persistent symptomatic reflux despite optimal PPI therapy, although dizziness and somnolence often limit use.⁸ These options are most helpful when the team has already clarified whether the dominant problem is acid reflux, non-acid reflux, dysmotility, cough hypersensitivity or aspiration. Antireflux procedures should be reserved for carefully selected patients. Current guidelines recommend surgery or endoscopic antireflux interventions only when reflux has been objectively documented, and they emphasize shared decision-making.^{8,9} Lack of response to PPI therapy predicts a lower chance of response to surgery.⁹ In bronchiectasis, pulmonary outcome data remain limited and largely observational, so fundoplication or other interventions should be framed as part of a multidisciplinary aspiration–reduction strategy rather than as a substitute for airway clearance or NTM treatment.¹⁰

Conclusion

A useful question for every clinic visit is as follows: what is the dominant driver today: mucus, mycobacteria or microaspiration? In patients with bronchiectasis plus reflux and NTM pulmonary disease, the best

outcomes usually come from doing several small things correctly at the same time: document microbiology, avoid inadvertent macrolide monotherapy, phenotype reflux objectively, look for dysphagia, modify

airway clearance so it does not worsen regurgitation and escalate beyond PPIs only when the physiology is clear. 

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